

**IN THE HIGH COURT OF SOUTH AFRICA
[EASTERN CAPE DIVISION: BHISHO]**

CASE NO. 25/2018

In the matter between:

S[...] M[...]

Applicant

and

MEC FOR HEALTH, EASTERN CAPE

Respondent

JUDGMENT

JOLWANA J:

Background.

[1] Plaintiff instituted action proceedings claiming delictual damages in her personal capacity and in a representative capacity as mother and natural guardian of A, a girl born on 31 December 2015. The claim is founded on the allegation that A was born with foetal distress, hypoxic ischemic encephalopathy (HIE), and superadded hypoglycaemia giving rise to spastic quadriplegic cerebral palsy and developmental delay. The plaintiff's case is that these birth defects of A were as a result of the negligence of the defendant's employees and further that that negligence caused the above mentioned birth defects. The plaintiff claims that as a result of the aforesaid negligence of the defendant's employees during the delivery process of A, she has suffered damages estimated at about R31 789 970.00. The defendant denies that its employees were negligent in the management of the delivery of A. The defendant further pleads that in any event and to the extent that it may be found that its

employees were negligent, such negligence did not cause the alleged birth defects. Therefore it was not responsible for the birth defects and therefore the damages that the plaintiff has allegedly suffered.

[2] The facts which are substantially undisputed are briefly the following. On or about 31 December 2015 plaintiff attended at Maphuzi Clinic (the clinic) as a result of the onset of labour having started feeling labour pains at about 02:00 am that morning. She was examined at the clinic and referred to Zithulele Hospital (the hospital) where she was admitted at approximately 09h00. She was, shortly thereafter, examined by the nurses. At various times during the day after her admission she was attended to by the nursing staff and medical practitioners in that hospital. Ultimately, she gave birth to A at or about 17:10 on 31 December 2015 through assisted vaginal extraction. Plaintiff alleges that while under treatment, supervision and care of the aforesaid defendant's employees, A was born with the birth defects referred to above.

The pleadings.

[3] Some of the plaintiff's pleaded case on which the damages claim is founded are stated briefly hereunder. The plaintiff has pleaded that the defendant's employees' treatment, care and management of her labour was inadequate in all or some of the following respects. They failed to monitor A's foetal heart rate (FHR) timeously. They failed to monitor the plaintiff's blood pressure timeously. There was no regular monitoring of the plaintiff and her foetus. They failed to diagnose or determine the onset of foetal distress, hypoxia and/or hypoxic ischemic encephalopathy and/or superadded hypoglycaemia. As a result, they failed to provide adequate treatment to the plaintiff and her foetus in order to prevent the development of foetal distress and HIE. Ultimately, during the delivery process or immediately thereafter they failed to diagnose that the foetus had suffered from hypoxia and HIE and superadded hypoglycaemia. As a result, they failed to implement appropriate treatment protocols.

[4] Plaintiff alleges that the defendant's employees failed to provide adequate care with the necessary skill and diligence as could be reasonably expected of medical

practitioners and nursing staff involved. In acting in the manner aforesaid or in failing to act as could be reasonably expected of them, the defendant's employees acted negligently. The defendant is therefore vicariously liable for the negligent conduct of its employees. This is because when plaintiff presented herself at the hospital on 31 December 2015 an oral, alternatively tacit agreement for the adequate rendering of medical services to her came into being. The express or implied terms, alternatively the tacit terms of such agreement were that the defendant would provide all medical services to her at its facilities during the delivery of A. The said services included the provision of all medical, surgical, nursing, monitoring, advisory, supervision, care and midwifery services to her during the delivery process of A.

[5] The defendant's plea is in denial of any aspect of negligence which is alleged by the plaintiff. The defendant further denies that to the extent that some or all of its employees may have acted negligently, that such negligence was causally connected to A's birth defects. Furthermore, the defendant pleads that there is a strong probability that the birth defects of A were as a result of or were caused by an underlying genetic problem in her family rather than negligence on the part of its medical practitioners and nursing staff who attended to her during labour. Negligence is therefore denied in part because, A's condition is atypical to cerebral palsy as it is progressive. Her neonatal encephalopathy is not in keeping with an intrapartum ischemic injury as the onset of her seizures was long after A's first day of life. The plaintiff has a poor pregnancy history. On the basis of inter alia, the above, negligence and causation are denied. Consequently, liability to the plaintiff and her child, A, is denied.

The evidence.

[6] The plaintiff herself was the only factual witness for her case. Her evidence was that she was born on 1[...] A[...] 1999 and she was unemployed. She went to school up to grade 9 at which grade she dropped out. She testified that her second child, A was born on 31 December 2015. Her first child was born on 19 September 2014 when she was 15 years of age. She was at home at the time of the delivery of her first child and an ambulance was called to no avail. She ended up delivering the baby at home in the morning between 8:00 am and 9:00 am. She was assisted in

the delivery process by her grandmother and her neighbours. That baby was a baby boy. He was not alive when he was born.

[7] In 2015 she fell pregnant again and gave birth on 31 December 2015 to a baby girl A, the child concerned in this case. Before A was born, she attended the antenatal clinic and she was examined. She visited the antenatal clinic four times before the labour date. On the 31 December 2015 she started feeling labour pains at about 2:00 am in the early hours of the morning. Her aunts called an ambulance which did not arrive. She eventually took a taxi to the clinic. At the clinic she was examined vaginally but not much was done and she was told that she was being transferred to hospital. Indeed, she was taken to hospital where she was attended to at about 09:00 in the morning.

[8] At the hospital she was again examined vaginally. An object that looked like a funnel was also pressed against her to examine the heart beat of the unborn baby. The nurses would listen to this instrument. She was thereafter told to walk around the passage. At about 11:00 am or 12:00 am, water erupted and she was taken to a bed. After she was put on a bed she was tied with a certain belt around her stomach and was examined. She was not told anything. At some point the belt was removed and a finger was inserted in her vagina and then she was told to push. She pushed but at some point she lost her strength. She was assisted to deliver the baby which she delivered at about 17:00. She did not see the baby which was not screaming or crying. The baby was taken away and she was told that she was being taken to theatre to be sutured. She only saw the child at about six or seven that same day when the child was brought to her.

[9] She was told to breastfeed the baby. She tried but the baby could not suck. She was advised to press her breast to get the milk into a mug. The baby was not able to latch. She expressed the milk into a mug and cup fed the baby. The cup feeding must have taken about a week. The baby started having seizures about two or three days after birth. The child remained with her in hospital until she was discharged on 11 January 2016. At the time she was discharged the baby was able to breastfeed. She was never told that there was anything wrong with the baby.

[10] When the baby was four months old she noticed that her eyes were squinting. When the baby was ten months old she could not lean or crawl. She had been taking the child regularly to the clinic for immunisation. She testified that up to the time she gave evidence, the child was developing well. However, she could not talk or eat properly on her own. She never had epileptic fits after she was discharged from hospital. On 29 July 2018 she had another baby. On this occasion she went to hospital at 08:00 in the morning and the baby was born at 12:00 midday. This baby was a stillborn.

[11] The plaintiff testified under cross-examination. Her evidence was that she had fallen pregnant three times and that the child, A was the second born of the three pregnancies. The child who was born on 29 July 2018 was a baby boy and was a still born. The first child was also a boy and was born at home on 19 September 2014. She attended antenatal clinic before that child was born. She used a clinic card in which the antenatal visits were recorded. She could not recall how many times she visited the clinic for the monitoring of her pregnancy but she only went to the clinic when she was eight months pregnant in respect of the third child. With respect to A she went to the clinic three times before the delivery date. She confirmed that she went to the clinic on 17 August 2015, on 17 September 2015 and on 24 November 2015. It was put to her that according to the hospital notes, A was sucking well on the 01 January 2016. She disputed that. With regard to the seizures which she had testified that they had happened on the second or third day, she conceded that she may have been wrong when she was referred to hospital notes which indicated that seizures only occurred for the first time on the fourth day.

[12] When it was pointed out with reference to the hospital notes that she had difficulty breastfeeding the baby and that on the 01 January 2016 at about 18:00, A was sucking well, she insisted that the baby could not feed on her own as a result of which she had to express milk into a cup. She however, confirmed being trained by the hospital nurses on how to breastfeed the child. She explained that at the time she was given instructions on breastfeeding, the baby would be with her but she could not suck. She could only put her mouth on the breast nipples. After the plaintiff's evidence, no other factual witness was called for the plaintiff. All the other

witnesses were expert witnesses specialising in various areas as will become apparent from their evidence.

The plaintiff's expert witnesses.

[13] The first expert witness called by the plaintiff was professor van Toorn. He testified that he is a paediatric neurologist at Stellenbosch University and Tygerberg Hospital. He explained that his area of expertise is anything that has to do with the brain, the foetal brain, the neonatal brain, and the infant brain. He has been practising as a child neurologist for no less than 15 years. He treats epilepsy, cerebral palsy and any condition that affects the brain. He explained that hypoxia ischemia is simply lack of oxygen, lack of blood to the brain. He was called in this case to opine on the cause and timing of the child's brain injury. He testified that based on the consideration of all the evidence that was at his disposal, his opinion was that A suffered a hypoxic injury of a mixed acute profound and prolonged partial variety occurring in the brain of term maturity. He timed the global insult to have occurred during the labour process.

[14] He also did a neurological examination of the child. This examination revealed that A has dyskinetic cerebral palsy. This means that the child has a movement disorder with reasonable mobility. The injury in this child was confined to the middle part of the brain that is responsible for movement. He testified that A was born in a compromised state which indicates that there were problems in the womb and that is indicative of foetal distress. He further testified that the hospital records reflecting that the baby was sucking after birth cannot be a correct reflection of what happened for the simple reason that absent a suck reflex there can be no sucking. This baby was born flat but recovered after a bag mask ventilation and stimulation, there was lethargic behaviour which is a subnormal level of consciousness and excessive sleeping. All of that was not normal. There was a weak grasp reflex. The suck reflex was not poor, it was absent.

[15] Dr Murray was the plaintiff's obstetrician and gynaecologist expert witness. She testified that the plaintiff was on early active labour on arrival at hospital and she was 5cm dilated. She testified that according to the Guidelines for Maternity Care in

South Africa, 2007 (the guidelines), when a mother is in active labour the mother's blood pressure and her heart are monitored hourly, her temperature, four hourly and her urine is measured two hourly. The foetal heart rate (FHR) should be measured every 30 minutes during and after contraction. The liquor should be observed every two hours. Contraction should be monitored hourly and caput moulding must be assessed two hourly. All of this type of monitoring is for low risk pregnancies. The outcomes of the monitoring should be plotted on a partogram from the time a patient presents in labour to the end so that the labour in its entirety is correctly documented.

[16] Dr Murray testified about the entries in the hospital records as follows. The first entry in the hospital records is at 09:00. She then said that based on this, no entries were made in the partogram until some two hours later when she arrived at the hospital as she had been at the clinic at 06:50. At that time her cervix was 5cm dilated. The FHR was recorded as being 135 beats per minute (bpm) and no decelerations were noted. She testified that the plaintiff had been at 5cm of dilation for some two hours by 09:00 with no change. She had arrived at the [clinic] at 6:50 which means it was some two hours by 09:00 with no progress.

[17] The next assessment was at 10:00 with no change and at 10:30 the (FHR) was recorded as normal with mild and moderate contractions. Dr Murray testified that had the plotting been done correctly, it would have been realized that there was poor labour progress which required reasonable management including assessing the mother to check if she was well, preferably by a doctor. This would have been to check if she was not exhausted or dehydrated and that there was no obstruction or disproportion to ensure that the foetus was not too big for birth. The foetal well-being needed to be assessed or monitored by CTG to ensure that the foetus was well and stable for the labour to continue and assessing the contractions to see if they were enough. Depending on what was found, a plan would be made including breaking the mother's waters, assist labour progress, doing a caesarean section, giving oxytocin or doing what she called, watchful waiting which is cautiously allowing more time. She explained that according to the guidelines labour progress which has crossed action line is indicative of a need for CTG monitoring. Because no capturing

in the first plot was done, the slow progress was never recognised. Therefore, at 09:00 already there was a problem which was not picked up or noticed.

[18] The next assessment was at 12:00 at which time the FHR was recorded as being 160 bpm before contraction and 140 bpm after contraction. There were no decelerations or no slowing of the heart rate. The membranes were intact, the water not having broken. The cervix was 7cm dilated with the head being three fifths above the brim and contractions were moderate. Therefore, at 12:00 plaintiff had made some improvement in the labour progress but it was still unsatisfactory progress. The partogram indicated that the heart rate before contraction was 160 bpm which was borderline high as the normal heart rate is between 110 and 160 bpm. So there was a marked increase in the heart rate. After contraction the heart rate fell to 140. When a heart rate falls after contraction, that is, by definition a deceleration which is indicative of foetal hypoxia or a foetus in distress. So this was suggestive of an abnormal FHR at 12:00. Therefore, the foetus should have been carefully monitored by CTG so that if there were signs of distress, resuscitation would be done or even a caesarean section if things did not improve. However, the abnormality was not recognised.

[19] The next review was at 14:00. However, the FHR was not assessed as there was no recording of the FHR at this time for some two hours since 12:00. The cervix was noted to be 5cm dilated with strong contractions. There appears to have been a regression in dilation. The best that she could posit was not that there had been a regression as that does not really happen, but that there was no progress in labour from the earlier 5cm and therefore progress was abnormally slow. Therefore, a caesarian section should have been performed at 14:00. But because there was no cognisance of the slow progress, no plans were made to remedy the situation. There was no documented heart rate for two hours despite the abnormality shown by the previous plotting. This is important because even if the patient was low risk, there should have been assessments of the foetal condition every 30 minutes which did not happen. This was therefore a substandard care of a very severe degree.

[20] At 15:00 the cervix was 7cm dilated. This was not much better but again the FHR was not recorded and there were strong contractions. There was also an

indication of a prescription for syntocinon. Syntocinon stimulates the uterus thus increasing the strength and frequency of contractions. This drug could not be used if there were signs of foetal distress or obstructed labour as it can cause foetal distress and uterine hyperstimulation by causing too many contractions. The most common complication of syntocinon is uterine hyperstimulation or tachysystole. Hyperstimulation is by definition having more than five contractions in ten minutes. This means there is not enough rest between contractions for the foetus to maintain oxygen levels. Therefore, continuous foetal monitoring by CTG is imperative. At 15:00 there was no record of the foetal condition since 14:00. The possibility of syntocinon infusion with no CTG monitoring would constitute substandard care of a severe degree. When there is CTG monitoring, its readings should be recorded in the notes showing heart rates, accelerations, decelerations, variability and all the things that one would read from the CTG.

[21] This ensures that a record of everything that happened is kept because CTGs do fade or even get lost or are misplaced. In this case, the partogram was not plotted in four hours which means there was no foetal monitoring and when it is plotted it shows a baseline of 170/140 bpm. This shows that the FHR was definitely higher and that the foetus had tachycardia. At this point the cervix was fully dilated at about 16:00 with the head being right into the cervix and there were no signs of obstruction. At full dilation she was now in the second stage of labour. At this stage strong contractions are frequent making it a potentially dangerous stage of labour. The guidelines say that the FHR should be oscillated every five minutes whereas in the first stage of labour it is done every 30 minutes. Therefore, monitoring must be increased to pick up any foetal distress timeously and act on it. In this case the baseline heart rate was 170 and there were decelerations according to the partogram. It seems that foetal distress was going on between 12:00 and 16:00 as the baseline had gone up from 160 at 12:00 to 170 at 16:00. However, the FHR is written as 150 to 155 in the assessments which is contrary to what appears in the partogram.

[22] Dr Murray explained that there was substantial improvement in the progress of labour in the last hour of labour which she attributed to syntocinon infusion. With that kind of progress, the plan to let her bear down was correct. However, in light of

a long labour and foetal distress, there should have been very careful monitoring during the second stage to make sure that there was no foetal distress and to quickly intervene if necessary. The next note was at 16:30 under assessment no. 5 in which it was noted that the patient had been pushing for 30 minutes with a plan to inform the medical officer. The FHR was 150-155 bpm and the medical officer was informed for a second opinion as the patient had been pushing for 35 minutes.

[23] Dr Murray further testified that in 2019 while attending this trial she received the original CTG tracings that were not readable because they had faded with some areas being readable. This was the last hour of the CTG tracings that were slightly clearer and she could read the FHR and the contraction pattern. She took some pictures so as to expand the CTG tracings on her cellphone to enable her to see them more clearly. However, the trial was postponed and she understood that the postponement was to enable her and Dr Koll to examine the CTGs together and do another joint minute. Dr Koll felt that the quality of the CTGs was too poor and he was therefore not prepared to comment on them. Following the availability of those CTGs she compiled an addendum to her initial report with the latter having been compiled also without some two pages of the records that were missing.

[24] These included assessment no.1 which was completed at 09:00. It reflected that the labour progress was good with maternal vitals being normal. The FHR was 135 – 140 bpm on a CTG and the plan was to monitor the labour progress and the patient was to be seen by a medical officer. Dr Murray testified that as a matter of fact the patient was not progressing well. The nurses had failed to take note of the fact that at 06:50 at the clinic she had been at 5cm of dilation already. Assessment no.2 was done at 12:00 which indicated that the progress of labour was good and the FHR was 136-151 bpm. Vaginal examination was done and the recording is that the cervix was 7cm dilated with no show on the glove. At 14:00 the assessment recorded poor labour progress with the cervix still at 7cm dilated. The heart rate was 140 – 150 bpm and the plan was to inform the doctor about the poor progress who ordered syntocinon noting that it should be started if the CTG was reactive.

[25] She summarised the labour progress as follows based on the completed hospital notes. At 06:50 the patient was at the clinic and she was 5cm dilated. At

09:00 she was at hospital where the first assessment records the cervix at 5cm dilated with a FHR of 135 – 140 with a plan to do a CTG monitoring and for her to be seen by a doctor. There is no note of a doctor seeing her. At 10:00 the partogram reflects a cervix that was 5cm dilated and the heart rate was reflected as 135 bpm with no decelerations. At 10:30 the heart rate was 130 bpm with no decelerations. At 12:00 the partogram shows a 7cm dilation and a FHR of 160 before a contraction and 140 after contraction with no decelerations. She pointed out that this was a contradiction. Assessment no.2 at 12:00 reflects a FHR of 136 to 151 bpm. Therefore, the two entries both at 12:00 are different. The partogram suggests decelerations but assessment no.2 says something else and the heart rates are different. At 14:00 the partogram shows 5cm dilation with no regard to the heart rate. However, according to assessment 3, the dilation was 7cm. Therefore, there was a discrepancy in the state of the cervix. There was no evidence of a reactive CTG at 14:00. The CTG of that time is unreadable.

[26] At 15:00 the partogram shows 7cm dilation but the heart rate is not recorded. The doctor's note shows a dilation of 7cm at 15:00 but no mention is made of the heart rate. The doctor ordered syntocinon to be administered if the CTG was reactive. Unfortunately, at 15:00 the CTG is unreadable and no recording of its reading at 15:00. At 16:00 the partogram says the cervix was fully dilated with two strong contractions in 10 minutes. For the most part it stays under the 110 beats per minute. She explained that at about 16:20 it goes up to 140 but immediately falls down to 85. It then goes up to 145 or 150 and then falls down to another deceleration of 90. So from two to three minutes past four the FHR fell into a sustained bradycardia, falling as low as 60 bpm for at least 5 minutes after which there is a pattern of recurrence probably late decelerations which failed to stabilise at a normal baseline. This means there was very little recovery.

[27] At about 16:06 there was a prolonged deceleration of about four to five minutes and ongoing recurrent decelerations. For 20 minutes the FHR was severely abnormal with a pathological CTG tracing. This was indicative of severe foetal hypoxia and probable acidosis at this time. The second abnormality on the CTG was in respect of the contractions at 16:05 and 16:15. There were eleven contractions in 10 minutes. So there were as little as 15 seconds between contractions at times

meaning there was no rest. There was one contraction immediately after another contraction. This was severe tachysystole which means too many contractions which caused a pattern of severe distress. In those circumstances assessment no.5 ought to have reflected a foetal condition of severe distress or some indication that the foetus was in trouble. However, it appears that it was not noted as it is not recorded anywhere. It appears that save for the notes on the partogram, nobody realized the abnormality in the heart rate.

[28] Then at 17:00 there is a delivery with vacuum extraction sheet indicating that the doctor did vacuum extraction at 17:00. She pointed out that it was correct for the vacuum extraction to be done which in any event needed to be done much earlier. The doctor noted that at 17:00 the heart rate was initially good but then there was tachycardia. The indication that the heart rate was initially good was misleading because for an hour prior to the vacuum extraction there was no indication of the heart rate being good. Perhaps the heart recovered to a tachycardia but still the severe decelerations were not recognised. Another discrepancy is that the time of birth was reflected as 17:10 in some places and 17:34 in others. Whichever the correct delivery time was, what becomes clear is that for at least an hour prior to delivery there was a pattern of severe distress which was not acknowledged anywhere in the hospital notes.

[29] Had the partogram been plotted correctly including while the patient was still at the clinic at 06:50 am the progress of labour would have crossed the action line by 13:00. This would have necessitated an assessment by a doctor and some intervention, be it allowing more time, rupturing membranes, oxytocin infusion or performing a caesarean section. Despite the progress of labour crossing the action line at 13:00 intervention in the form of oxytocin was only taken at 15:00. Oxytocin intervention depended on a good foetal condition. However, at 12:00 the partogram and the records do not correlate. The partogram was suggestive of decelerations whereas the records were indicating a normal heart rate. Unfortunately, the CTG is unreadable to ascertain what the situation was at 12:00. Even at 16:00 there is a discrepancy between the partogram and the notes. The partogram indicates foetal distress in the form of tachycardia and late decelerations and the records indicate a normal baseline heart rate. The CTG tracings from 16:00 are readable and they

confirm severe foetal distress. Clearly the indication of a normal heart rate was misleading. This means that at that time what was recorded in the partogram was correct. This suggests that the partogram at 12:00 might be correct in which case there were decelerations from as early as 12:00. She then testified that there was a complete failure to recognise the tachysystole and the foetal distress. This resulted in the foetus being left in utero for a further hour and probably it was during this time that the hypoxic injury was sustained. This is illustrative of a severally substandard and dangerous obstetric care in a high risk labour.

[30] It was put to her under cross-examination that according to Dr Koll and his understanding of the notes there never was a time when there was an indication for a caesarean section as there was adequate progress. She testified that she disagreed with that because labour progress was inadequate for the following reasons. The patient arrived at 06:50 being 5cm dilated [at the clinic] and five hours later at 12:00 she had made only 2cm of progress. She then referred to the guidelines where it is stated that “poor progress in the active phase of labour: There is poor progress and the cervix dilates at a rate of less than 1cm per hour in the active phase”. Therefore, if she was 5cm at 06:50 and was 7cm at 12:00 she had only made 2cm of progress in five hours. Therefore, according to the guidelines there was poor progress. There were also multiple discrepancies in the records between what was recorded in the partogram and what was written in the assessments which would lead to the inevitable confusion about the foetal condition.

[31] The next defendant's witness was Dr Gericke. He testified that he is a registered specialist paediatrician and a specialist medical geneticist. He had an interview with the plaintiff and did a clinical genetic examination of the child, A. His interview with the plaintiff did not reveal any information that raised any issue of concern. He testified that on 19 November 2019 he saw the child who had a global neurodevelopmental delay with dyskinetic cerebral palsy. There were no external features present which would indicate the possible existence of an underlying clinical genetic disorder or chromosome disorder. He saw the imaging report of professor Andronikou which was done on 19 June 2018. The scan essentially indicated two main things. Firstly, it was a mixed type injury, a profound type of brain injury. There was also the possibility of other conditions. He explained that genetic conditions are

inherited conditions. They can include all kinds of genetic syndromes or structural brain disorders. On the other hand genetic metabolic diseases are biochemical processes gone wrong in the body which are inheritable and which can mimic cerebral palsy. He then discussed his addendum report which he compiled after he had received the results of blood tests that were done to exclude certain conditions. His conclusion was that there was no genetic predisposition which had been clinically indicated. Before Dr Gericke could be cross-examined it transpired that there was some engagement between the parties' genetic experts for purposes of concluding a joint minute. This necessitated the cross-examination of Dr Gericke to be put in abeyance.

[32] In the meantime professor van Toorn was called. Plaintiff's counsel brought to the court's attention that professor van Toorn, the plaintiff's paediatric expert witness had filed a supplementary report which dealt with a report filed by professor Rothberg, the defendant's counter-part. In dealing with professor Rothberg's report he touched on an number of issues like macrocephaly and the significance of apgar scores both of which he said on their own they were not decisive of anything. He testified that the foetus was exposed to a lack of oxygen for prolonged periods, possibly hours. This, he said was with reference to professor Adronikou's report which talked about prolonged partial brain injury to the middle part of the brain. So there was a pattern of prolonged partial lack of oxygen for an hour or even longer which was severe and sustained which culminated in the injury where the central structures of the brain were compromised. As a result, there were many signs of severe neurological compromise like the baby being born flat, needing resuscitation, lethargy, not sucking, difficulty to arise and neonatal seizures. He felt that the apgar scores as they appear on the records were assisted because of the bag mask ventilation that was performed. There were signs of significant compromise with the baby staying in hospital for 11 days whereas healthy normal babies get discharged within the next day or so after a vacuum delivery. He was of the opinion that the use of bag mask ventilation was indicative of secondary and not primary apnoea. The importance of secondary apnoea which led to bag mask ventilation was that the foetus stopped breathing while inside the mother, in other words the baby was deprived of oxygen during the labour process.

[33] He then went on to discuss the lethargy, grasp reflex and lack of sucking. He explained that encephalopathy implies a child with a depressed brain. In this case there was no evidence of serial neurological examination on days two and three which made it difficult to assess the severity of the encephalopathy. Absence of the suck reflex was a sign of a moderate neonatal encephalopathy so was the lethargy. On day four the speech therapist could not wake up the child. He was of the opinion that all these taken together were signs of a mild evolving to a moderate degree of encephalopathy. He agreed with professor Rothberg's view that hypoxic ischemic encephalopathy usually results in seizures within the first three days of life. However, there were no notes or very little notes on days two and three in the life of this baby. He then postulated that diagnosing seizures in the context of hypoxic ischemic encephalopathy can be challenging and are often underdiagnosed.

[34] He then referred to Volpe¹ who says that even medical staff may have difficulty in diagnosing epileptic seizures at an early age because of the subtle nature of the seizures in young babies which manifest in things like blinking, lip smacking, tonic seizures where the baby goes stiff or subtle jerks to myotonic seizures. It may well have been tonic seizures when the baby was arching backwards or having episodes of intermittent extension. These may have been manifestations of a compromised brain or neonatal seizures. There was also no blood sugar documentation in the critical first three days of life. He explained that the brain injury pattern of a prolonged partial nature as professor Andronikou reported was consistent with low blood sugar levels resulting in injury in the thalamus area of the brain. He explained that intra-cranial haemorrhage as a cause of neonatal seizures could be discarded as there was no evidence of any inter-cranial bleeds. Similarly, hyperbilirubinemia which is brain injury that occurs when a baby is very jaundiced shortly after birth could also be discarded as a cause of this child's brain injury.

[35] The next witness called by the plaintiff was professor Smith. He testified that he is the head of the neonatal intensive care and neonatal care at Tygerberg Children's Hospital. He testified that in his view this baby was born with in secondary apnoea because she was born flat with no efforts of breathing which led to manual breathing

¹ Volpe J.J.: Neurology of the Newborn fifth edition page 402.

support. This would be in keeping with the bradycardia that the baby probably had and the low apgar scores underscore this point although appear to have been changed from 1 to 2. He testified that in terms of the guidelines, the baby's care, following a successful resuscitation should have been escalated to a higher level of care. This did not happen. With reference to the hospital records where at 07:30 on 01 January 2016 the note read that the baby was crying, poor latching, was hungry. He understood this to mean that the doctor regarded the inability to latch and crying as being hungry. He considered the crying to be attributable to irritability. There was cerebral irritation. There is no record showing vital observations performed between the 31 December 2015 and the 4 January 2016 other than recording body temperatures and pulse rate. Blood sugar level which should have been checked and maintained was not checked. Blood sugar level was checked for the first time late on the 4 January 2016. There should have been frequent assessment of fluid balance which was not done. Renal failure exclusion was never done. The serum phosphate and calcium level was only done on the 4 January 2016.

[36] The baby required good observation in the first hours after the delivery. It is that observation that may lead to the infant case being escalated. These are also necessary to prevent secondary insults to the foetal brain. This is also done by checking blood sugar level as the most basic bedside investigation to diagnose hypoglycaemia. If you detect them you institute treatment to prevent another drop and maintain stable levels of blood sugar. It was also not sufficient to simply say the baby is sucking well, they should have recorded how long did the sucking take place. He testified that the speech therapist saw the baby at 15:30 on the 4 January 2016. Her report indicates a clear level of depressed consciousness. She reported on an abnormal oral mouth and tongue movements. The baby was difficult to wake and recommended a syringe feeding. The speech therapist reported on many other problems she observed at that stage. He testified that the baby going on occasional extensions were in his view, typical tonic posturing or tonic seizures. Therefore, seizures were there but not recognised as such. The report of the speech therapist indicated that she also noticed that the baby was jaundiced.

[37] He testified that the baby had a mild encephalopathy which is ascribed to intra-partum asphyxia which therefore is hypoxic ischemic encephalopathy. He and

professor Rothberg agreed that it was initially stage 1 encephalopathy which evolved to stage 2. The baby must therefore have deteriorated at some point before she was seen by the speech therapist on the 4 January 2016 at 15:30. The difficulties in waking up the baby indicated deterioration in the level of consciousness which the attending staff did not pick up until the neurological challenges with the baby were highlighted by the speech therapist. It was from this point that care was escalated. They could not have picked up the deterioration in the neurological status of the baby as they did not perform proper assessments. He then referred to the doctor's note referring to brain swelling which he said was in keeping with intrapartum sustained asphyxia because brain swelling developed three to five days after the insult which is what happened in this case. The baby exhibited the typical case of an early onset of neonatal encephalopathy of a moderate degree probably due to intrapartum sustained hypoxic ischemic injury. He and professor Rothberg agreed that there was no infection that contributed as a cause to the outcome of the baby and the development of neuro disability. He explained that there was no way of explaining the compromised baby at birth and the subsequent neurological compromise besides intrapartum asphyxia.

[38] Professor Smith further testified that he and professor Rothberg, the neonatologist of the defendant entered into an amended joint minute in which there was agreement on the following issues. They agreed that the baby developed and presented with mild hypoxic ischemic encephalopathy between birth and around 16:29 on 3 January 2016. The baby would not have qualified for therapeutic hypothermia or cooling during the first six hours of life. The foetus suffered intrapartum hypoxic ischemia. The baby's neurological condition, the encephalopathy worsened around the 4 January 2016. The MRI report of professor Andronikou described features consistent with combined partial prolonged and acute profound hypoxic ischemic injury. However, the report also indicated that the MRI pattern may be seen with toxic, metabolic and post infectious causes. The infection as a probable causal factor in the aetiology of the baby's encephalopathy has reasonably been excluded.

[39] There were also disagreements between himself and professor Rothberg. They disagreed on the interpretation of the baby's head circumference at birth and its

implications to this matter. Whether or not there was a macrocephaly, that is a large head or megaloccephalic head. He was of the view that there are no independent head circumference values for the new borns in South Africa. Some ethnic groups have large head circumference than others. He testified that the ratio of the head circumference over the weight of the baby at birth was 0.01 which was normal and therefore proportional. His conclusion was that it was unlikely or improbable that the baby suffers from megaloccephaly or macrocephally. In any event subsequent metabolic and genetic investigations found no associated causal aetiological factors. He went on to say that professor Andronikou made no finding of congenital anomalies of the brain or anatomical abnormalities.

[40] He disagreed that after the evening on day 1 the neonate was well except for the problem with latching. His view was that the recommended systematic observations were not done, there was no neurological assessment performed at 22:34, there was poor latching and the baby being hungry. At 07:30 the next morning the records showed that the baby was crying, there was poor latching and she was hungry. He was of the view that this condition of the baby was attributed to hunger, mistakenly. There was cerebral irritation and the baby was unable to latch properly and suck due to the encephalopathy. Therefore, the nurse's note at 17:10 on 01 January 2016 was not in keeping with a baby who was sucking well as there were repeated references by the doctor at 22:34 the previous evening to the baby's inability to suck hence there was cup feeding reference at 07:30 on the 01 January 2016 in the morning, cup feeding on 16:33 on 02 January 2016 and at 03:35 on 03 January 2016. This taken with the evidence of the mother it was likely that there was inability to latch and suck. He was of the view that there were very poor observations and assessments.

[41] He explained that progressive HIE signs were there but were only picked up for the first time on the 04 January 2016. They both agreed that there was mild hypoxic ischemic encephalopathy but in his view on the 4 January 2016 it was of moderate degree. He referred to the plan to perform hemugluco test (HGI) blood sugar level which was not done and he described that as being substandard. There was no record that blood sugar level was checked before the 4 January 2016. New borns who suffer intrapartum asphyxia are prone to develop early neonatal hypoglycaemia

and hypoglycaemia is an independent brain injury factor. There is a significant overlap between brain injury patterns of hypoxic ischemic nature and patterns related to neonatal hypoglycaemia. Therefore, a contributory role for hypoglycaemia cannot be excluded.

[42] Professor Smith further testified that looking at a photocopy of the CTG trace, a pathological foetal condition was present around 16:00, a grossly pathologic trace was in keeping with foetal hypoxia and acidosis. There were excessive uterine contractions per time interval. The nurse's note that before birth there had been no foetal distress was in contradiction with the recording of the doctor who performed the vacuum extraction around 17:05 to 17:10 who recorded that the FHR was initially good then tachycardia. There were also entries in the partogram showing foetal tachycardia before contractions at 16h00. The baby was diagnosed with hypoxic ischemic encephalopathy as the reason for her neonatal encephalopathy. HIE is usually preceded by foetal distress during labour manifesting with detectable abnormal heart rate as it occurred in this case. He and professor Rothberg disagreed on whether expedited delivery or intrapartum obstetric management would have avoided the neurodisability. He was of the view that substandard intrapartum obstetric care directly contributed to the child's neurological disability and that timeous expedited delivery would have avoided the outcome. Furthermore, had proper neonatal care been done the possibility of aggravating factors such as hypoglycaemia contributing to the outcome would have been avoided. Professor Rothberg agreed that in the absence of the family history in this case there would likely have been little to argue other than the presence and/or severity of foetal distress.

[43] Dr Gericke, the plaintiff's geneticist was called to continue with his evidence on the basis that a joint minute had since been entered into between himself and the defendant's geneticist, professor Christianson since his previous evidence. Dr Gericke testified that when considering a need for genome wide testing or other exome sequencing, according to a paper published in 2019 on genetic mimics of

cerebral palsy², there are eleven indications that must be present for genetic and metabolic conditions in a patient presenting with symptoms of cerebral palsy. He said that because none of these indications were present in this child, further whole exome sequencing or genome wide sequencing was not indicated.

[44] Dr Genicke testified under cross-examination. He testified that there are five levels of motor disability according to the GMFCS classification and he considered the child in this case to have a level 4. He explained that stage 4 would mean the child can move with some assistance but would probably require to be carried or moved around with some form of support like a wheelchair. He explained that the classification system uses a scale from one which is the least disabled to five which is the most disabled. He said that GMFCS stands for Gross Motor Function Classification System. This child was at GMFCS 4 cerebral palsy which indicates a severe level of motor muscle disability. He examined the child on 19 November 2019 and he based his opinion on the examination that he conducted on that day and his assessment was that the child was at GMFCS 4 level.

[45] He was then referred to various reports by other experts. He was referred to a report by professor van Toorn dated 26 November 2018 in which he concluded that the child was GMFCS1. In his report professor van Toorn explains that at GMFCS 1 the child can floor sit with both hands free to manipulate objects, movements in and out of floor sitting and standing up performed without adult assistance. These children walk as a preferred method of mobility without the need for any assistive mobility device. He agreed that the above description was not of a patient with a cerebral palsy that is at GMFCS 4. He explained that he would not differ with professor van Toorn. He therefore, did not know why on that particular day, the child appeared to be in the condition which led him to classify her as level 4. Dr Gericke was then referred to the report of Dr Freda van Rensburg which was dated in

² Pearson T.S et al. Genetic Mimics of Cerebral Palsy: Movement Disorders, Vol. 34, No.5, 2019 page 625 at page 628 where the following are listed:

Absent history perinatal risk factor for brain injury, family history of sibling with similar neurological symptoms, motor symptoms onset after an initial period of normal development, developmental regression, progressive neurological symptoms, paroxysmal motor symptoms or marked fluctuation of motor symptoms, clinical exacerbation in the setting of a catabolic state (e.g, febrile illness), isolated generalized hypotonia prominent ataxia, signs of peripheral neuromuscular disease (reduced or absent reflexes, sensory loss), eye movement abnormalities (e.g., oculogyria, oculomotor apraxia, or paroxysmal saccadic eye-head movements).

November 2019 the same month as his report in which Dr Freda van Rensburg concluded that the child was totally mobile without any help. He conceded that there was a huge difference in these two assessments. He said that he could not explain why on that day that he saw the child he got the impression that she was at level 4. He then said that he deferred to paediatric neurologists and was prepared to accept that the child was actually at level 1. He then said that the court could disregard his conclusions with regard to the level of disability of the child.

[46] Dr Gericke was then referred to his report where he said that the child has a history of early convulsions occurring shortly after birth or in the first week neonatally. He testified that he also concluded that the child had a standard 3 neonatal encephalopathy but said that was based on his assessment of the child being GMFCS level 4. He now accepted that the child was GMFCS level 1. He explained that neonatal encephalopathy is graded 1, 2 and 3 with 3 being the most severe form of neonatal encephalopathy usually characterised by the presence of convulsions, disturbed consciousness and the presence of neurological features. It was put to Dr Gericke that some of the information in his report like the baby having been hospitalized for three weeks or longer did not refer to the baby in this matter. He then said that he was accepting that some of the information from two separate patients may have been included in the bundle he was working on. He explained that information from two different patients was entered into the historical findings in his report in this matter. He, however, maintained that the clinical genetic findings and conclusions were correct and were applicable to the child in this matter.

[47] He testified with reference to the report of professor Andronikou which says that the pattern of injury in this patient can also be seen with toxic, metabolic and post infectious causes. He confirmed that because of the non-progressive evolutionary cause of neurological feature in this child, if the toxic, metabolic and post infectious causes were present, there would have been a loss of previously acquired skill. This is because cerebral palsy is static. He therefore concluded that there was no variation in the features of this child and therefore genetic testing was not indicated. The question is, is there a recurring genetic problem or is there a possibility of a recurring mechanical problem during labour. Therefore, before consideration is given to genetic causes, one must consider a possibility of prolonged second stage

of labour associated with obstructed labour and a relatively large baby in a smaller sized mother. There are specific indications which he mentioned before, for genome wide sequencing or whole exome sequencing. None of those eleven indicators are relevant to this child. His first concern even before getting into a genetic disorder was the obviously very short mother with likely insufficient pelvis known as cephalopelvic disproportion or CPD for a normal delivery. The second consideration in the indication for a genetic test is the absence of an incriminating birth history which the obstetricians have indicated was a prolonged second stage and foetal distress before delivery. Lastly, the baby was delivered with a neonatal encephalopathy. He found nothing in the examination of the child that would indicate a need for further testing. The results from Centogene³ were negative.

[48] Dr Gericke was cross-examined on the defendant's case about genetic testing. He started by explaining that a rather extensive genetic testing has already been done and it excluded a large amount of candidate genetic disorders. The next genetic testing would be genome wide sequencing (GWS). The problem with it is that its clinical utility could be for about five percent of individuals with cerebral palsy. The information that is then generated from GWS creates a large amount of variants of unknown significance which may have no bearing on the problem at hand. If a variant of unknown significance is found it requires protein structural analysis to elucidate its relevance. Therefore, the fact that Centogene says further investigations are necessary or many be helpful, is a comment that laboratories make routinely if an answer has not been found. But in a clinical setting one would not go for GWS after a large number of specific biochemical and genetic structural disorder which could be relevant to the patient's problem have been exhaustively excluded.

[49] It was put to him that the plaintiff has had four pregnancies. Two of those were boys and they died, the first one reportedly after birth and the second one a stillborn. There are two girls, the child A and the last girl child. Both suffered seizures days after birth. He confirmed that of the eleven indications for genetic testing at least one of them must be there. With regard to the family history of the four children, there is

³ Centogene is a laboratory based in Germany which conducted the metabolic testing in this matter which are referenced in the evidence

no clinical information about the problems the other children suffered from. In order to establish that there is a causal contribution of a genetic factor to a cerebral palsy outcome, there must be a direct pathway that can be inferred from the genetic abnormality to a cerebral palsy. In this case there were obstetric problems with the delivery which were an excluding factor for genetic testing.

[50] The other witness for the plaintiff was professor John Anthony. He testified that he is a qualified gynaecologist, a registered subspecialist in maternal and foetal medicine and the head of maternal foetal medicine unit at Groote Schuur Hospital. He was a nominated South African representative in a multinational panel of experts which produced a paper on intrapartum FHR monitoring on behalf of the International Federation of Obstetricians and Gynaecologist which was published in 2015. He testified that the occurrence of labour for the foetus represents possibly the single most dangerous time in pregnancy. The baby on average needs about 2 to 3 minutes between contractions to allow the choriodecidual space to fill with fresh blood. If anything is done to increase the frequency of the uterine contractions by giving the mother oxytocin or prostaglandins, the time between contractions during which the body can extract more oxygen as the uterus relaxes is reduced. This may increase the risk of the baby becoming hypoxic and acidotic during the course of the labour. In addition, there are other things that happen physiologically like when the mother goes into the second stage of labour. This is when the cervix is fully dilated and the birth process actually begins as the baby begins to descend through the birth canal. In those situations, the mother has a desire to push or bear down. This pushing increases the pressure inside the uterus. The injunction that comes from the international and national guidelines is that during the second stage of labour, the foetal well being needs to be assessed, not every half an hour as it is during active labour but every five minutes or after every second contraction. This is because during this period the foetus is vulnerable.

[51] He then testified that he and Dr Koll agreed on the facts recorded in the clinical records. Some of those facts were that her last menstrual period had been on 13 April 2015 and therefore her due date would have been on 18 January 2016. However, she went into labour on 31 December 2015. She therefore delivered just over 37 weeks gestation which is term. Her examination was normal. At 151cm tall

her height would not arouse any particular obstetric concern. It is at less than 150cm where she could be regarded as particularly short. All her vital signs were normal. Her body mass index was normal. As a result the only issue of concern was the child who died. Routine investigations were performed and were all normal and the screening test for HIV was negative. There was no problem during the antenatal period. The plaintiff then presented at the clinic just before 07:00 in the morning on 31 December 2015. She complained of abdominal pain. On vaginal examination her cervix was 5cm dilated. The FHR which they listened to at the clinic was 124-126 bpm and contractions were palpable. The plan was to refer to hospital.

[52] The plaintiff then arrived at hospital at 09:00 in the morning. It was noted that she had been referred from the clinic and that she had been experiencing pain for seven hours which was from 2 o'clock in the morning. Her cervix was still 5cm dilated. The FHR was recorded as 135-140 bpm. It was indicated that the heart rate was derived from a CTG. It was evident that they did not have any concern about the foetal well-being at 9:00 and they were happy with the CTG.

[53] The next assessment was at 12:00 midday. At that time the cervix was 7cm dilated. The attending staff felt that the progress of labour was good. They wrote a FHR of 136 to 151 bpm. At 12:50 the notes indicated that her membranes ruptured spontaneously and that the liquor was clear. The FHR was recorded as 136 to 150. At 14:00 the cervix and the vaginal examination were done and she was still 7cm dilated. The nurses noted that the progress of labour was poor and the FHR at that stage was 145-150 bpm. These findings were discussed with the doctor on call who ordered the administration of syntocinon and said that if the CTG was reactive it could be started. In terms of the guidelines the administration of syntocinon requires an assessment of the foetal well-being before it is infused. This is because it can cause an increase in the frequency of uterine contractions and can lead directly to foetal distress. At 15:00 another examination was done and the cervix was found to be still 7cm dilated. Now over a period of three hours from 12:00 the cervix was still 7cm dilated. An assessment was made that the plaintiff had crossed the action line and the oxytocin was prescribed and a catheter was to be inserted and the management was to proceed provided the CTG was reactive.

[54] A patient can spend up to 8 eight hours in the latent phase of labour without much sign of cervical change and no rapid cervical dilation without there being any problem. Once the patient is in the active phase of labour it is expected to progress at a recognisably rapid rate. So there are two lines drawn at an angle on the partogram. The first line is the alert line. This line exemplifies a cervical dilation taking place at a rate of 1cm per hour. The second line which is drawn parallel is called the action line. If a woman in labour gets into the active phase of labour and starts out in alert line but then falters during labour and the rate of cervical dilation does not take place at 1cm per hour and it crosses that action line, that indicates that there may be a problem requiring further assessment. So the people who assessed her at 15:00 decided that she had crossed the action line and decided to augment the contractions by prescribing oxytocin. At 16:00 she was having very strong contractions. When she was examined she was now fully dilated and she had an urge to bear down. The notes say that the FHR was 150 to 155 and the CTG was reactive.

[55] A reactive CTG is a term used to imply that the CTG tracing is normal. The plan of the nursing staff was to get ready for delivery and to let the mother push. At 16:30 they called the medical officer and said the plaintiff had been pushing for 35 minutes and the baby had not yet been delivered. They wrote down that the FHR was 150 to 158. Then at about 17:00 the medical officer had arrived and proceeded to deliver the baby using vacuum extraction. A woman in her second pregnancy delivering vaginally for the second time, from the time she starts bearing down should deliver within 30 minutes. Therefore, under those circumstances it would have been correct to say the second stage was prolonged and therefore consideration of an instrumental delivery was correct. Also if foetal distress had been diagnosed, it too would have been valid indication for an instrumental delivery. The vacuum extraction seemed to go without any complications and it took two pulls and it lasted five minutes and the baby was delivered vaginally.

[56] The partogram was incorrectly plotted in the latent phase and began at 09:00. The 06:50 examination at clinic was not entered where it should have been which was in active labour part of the partogram. The first examination at the clinic when the patient was 5cm dilated ought to have been considered as active labour and

considered as being the first plot on the active phase labour part of the partogram. The first indication that the patient had crossed the action line would have been at 12 o'clock. However, on the partogram that diagnosis was not made until 15H00, some three hours later. Any patient who is not progressing in labour needs to be assessed carefully and the reason for the slow progress needs to be found. In first phase of labour where slow progress is found, a deliberate examination must be done to make sure that there are no signs of cephalopelvic disproportion (CPD). A parous woman with a slow progress should lead to immediate, very critical assessment of the labour to look for signs of disproportion and in such circumstances oxytocin should only be prescribed after such examination. This is also stated in the guidelines.

[57] The heart rates that were charted in the active phase of labour reflect 'o's and 't's. These refer to the heart rate before and after contractions. The FHR on the partogram was only recorded at 12:00 and then again at 16:00. The requirement is that during active labour the heart rate must be observed before and after contractions every half an hour. If the partogram began at 09:00 there should have been observations at 09:00, 09:30, 10:00, 10:30, 11:00, 11:30, 12:00 when it took place, 12:30, 13:00, 13:30, 14:00, 14:30, 15:00, 15:30 and 16:00 when it took place. So the number of observations was clearly not according to the prescription that exists for patients who are in spontaneous and even completely normal labour.

[58] Professor Anthony explained that CTG tracings are divided into three categories. One is normal, the second category is called suspicious and the third category is called pathological. If the tracing falls into a pathological category, there is a high probability that the baby is hypoxic and acidotic. In this case the CTG was pathological. There was a very prolonged FHR deceleration beginning shortly after 16:00 which lasted for more than three to five minutes. That on its own was so abnormal that the tracing was pathological with a high probability that the foetus was hypoxic or acidotic. There are three main situations that may arise leading to a hypoxic environment. It is the uterine contractions. If this is followed by the mother pushing, there is even less blood flowing into the choriodecidual space and the baby is in further trouble in terms of oxygen supply. Then a further problem is when oxytocin is administered thus making uterine contraction more frequent than it

should. What should then happen in that situation is that the foetus should be taken out of the hypoxic environment through instrumental delivery. Oxytocin should be stopped and the mother should be stopped from pushing.

[59] At the time of the pathological tracing there should have been an immediate action to correct reversible causes of intrauterine hypoxia followed by expedited delivery. The contractions are stopped by means of tocolytic drugs that relax the uterus. The CTG tracings show many contractions which may have been aggravated by the mother being encouraged to push when she was in the second stage of labour. Both the use of oxytocin and the maternal bearing down efforts should have been stopped pending further assessment and to allow restoration of foetal oxygenation by means of intrauterine resuscitation prior to expedited delivery. There are tocolytic drugs, putting the mother on her side to optimise perfusion of the uterus and also to give her face mask oxygen. Those are the components of intrauterine resuscitation. He testified that seemingly the pathological tracing was not recognised as a result of which the necessary intervention did not take place. This was substandard care which was directly linked to an increased likelihood of an adverse outcome.

[60] Tachysystole, is too many contractions or more than five contractions per 10 minutes. The decision to allow the mother to push in the face of the pathological tracing was substandard care which would have increased the risk of hypoxia. The requirement is also not just to auscultate the FHR before and after contractions and to plot on the partogram. There has to be the process of interpreting what is found. It is not good enough to do a CTG if it is not interpreted. The fact that the CTG is running is in and of itself insufficient for monitoring foetal wellbeing. There was no adequate interpretation and the mother was allowed to push. He said that to describe a FHR that varied between 170 and 140 bpm at 16:00 as being normal or reactive was beyond belief. The uterine tracings showed tachysystole, severe recurrent decelerations and a pathological tracing. The nurses called the doctor after 35 minutes of bearing down without any recognition of abnormality, and no attempt was made to curtail uterine contractions and no intrauterine resuscitation was done.

[61] Professor Anthony talked about inherent difficulties associated with monitoring the condition of the foetus during the second stage of labour and the danger of interpreting badly faded tracings, a postulation of Dr Koll. He said that decelerations happen during the second stage of labour but of importance is that they should not persist after the contraction. If they persist after the contraction they are more consistent with a diagnosis of hypoxia than compression as the head moulds through the birth canal. The nursing staff were required to listen to the heart rate after contraction. If they hear a slow heart rate after the contraction they will notice if there are decelerations. He agreed with Dr Koll about the danger of interpreting badly faded tracings. However, the fact that the tracing is faded does not mean it is always going to be impossible to discern anything from it. He said if one looked carefully at pages 39 and 40 of bundle 1G using a good light you will be able to make out the FHR pattern and the contraction pattern.

[62] On the issue of what is called loss of contact, Dr Koll said that in the second stage of labour the patient is often moving about in pain, causing loss of contact between the probe and the foetal heart which may affect the tracing. While that is true during labour the mother hyperventilates which may cause loss of focus on the FHR as she pushes which may disrupt the tracing to some extent, that does not validate what one can see and it does not make the tracings completely uninterpretable. He then dealt with Dr Koll's statement that during the second stage of labour the foetus will often experience periods of hypoxia resulting in abnormal FHR patterns that would be quite alarming if found during the first stage. Professor Anthony said if there is evidence of hypoxia no matter how it occurs, there is an obligation to act by resuscitating the baby and expediting delivery. On variability he said this is where the baseline wiggles about before there is acceleration or deceleration in the heart rate. When it does not wiggle about as it should, when it is a flat line, that is usually consistent with a diagnosis of hypoxia. The diagnosis of hypoxia is based a several issues like, what is the baseline, variability, are there decelerations and what is their nature, are there accelerations present. All of those are components of an assessment of the trace and variability on its own does not define a pathological tracing. He therefore disagreed with Dr Koll that because the tracing is faded it is very difficult to make out variability in the first two minutes and therefore you cannot tell if the baby is in trouble or not. What is clear on this tracing

is that there is a very prolonged deceleration that begins shortly after 16:00 and that, more than anything else, is an indication of a pathological tracing.

[63] About the partogram, professor Anthony explained that the partogram is divided into two sections. The first 3rd of the graph is the latent phase of labour which is when the cervix is less than 3cm dilated. The active phase of labour on the right hand side of the graph where there are two parallel lines drawn running from 3cm to 10cm dilation at a given rate of 1cm per hour. At the active phase, labour is expected to progress at a predictable rate. If the deviation goes beyond the second line which is called the action line, there must be some investigation to ascertain why the labour is progressing slowly. The partogram was incorrectly annotated for two reasons. First the examination at about 07:00 in the morning where she was 5cm dilated which was evidence of second stage of labour should be the starting point on the graph or on the alert line and that should have been plotted. The starting point determines the plots that follow to the hourly time intervals. At 09:00 the plaintiff was still at 5cm dilated which would have been a plot on the action line. The next examination at 12:00 showed evidence of progress from 5cm to 7cm which was a change of 2cm over three hours.

[64] This was slow progress and it ought to have been evident at 12:00. The error was not to start with the first finding at the clinic. Second, the hospital examination at 09:00 was incorrectly plotted in the latent phase part of the graph whereas it should have been plotted on the alert line as the beginning point. All of this led to the delayed progress of labour being identified later than it should have been. By 12:00 it should have been evident that the action line had been crossed warranting further investigating. The requirement in the guideline is that there should be a critical evaluation of slow progress in labour to exclude CPD and foetal distress. Either of those events would require an emergency delivery by caesarean section. If neither of those clinical diagnosis is evident, the labour can be allowed to continue. Other interventions can be considered like oxytocin. There was an indication for an intervention by means of caesarean section delivery as early as 12:00. However, the baby was delivered some five hours later after 17:00. The ongoing slow progress together with the evidence of foetal compromise strengthened the intervention indications. Intervention indication was also evident at 15:00.

[65] He testified that according to the guidelines a woman who shows slow progress in active labour, must have foetal distress excluded. In this case the doctor initially did not evaluate the plaintiff at 14:00 before issuing instructions for the use of oxytocin. She was unaware that they needed to exclude foetal distress as is clearly indicated by her indication that CTG should be done first. The fact that the doctor examined the patient at 15:00, indicated that oxytocin should be administered and the putting up of the Ringer's Lactate at that particular time would have been done in order to administer oxytocin intravenously. Oxytocin is known to be associated with the risk of uterine tachysystole or too many contractions. That is why it should be used with great caution in multiparas after excluding CPD and there must be no evidence of foetal distress and CTG monitoring should be used where possible. In this case oxytocin was prescribed to a parous patient which is only done in exceptional cases. As a result, tachysystole became evident. Professor Anthony testified that he disagreed with Dr Koll on his view that oxytocin was ordered with due precautions in this case. He said due precautions are those prescribed by the guidelines which is the exclusion of CPD and the exclusion of foetal distress prior to commencing with the drug and then the monitoring that was required after the drug was started.

[66] Dr Koll said there is no evidence that oxytocin was administered. Professor Anthony dealt with Dr Koll's argument by saying this ignores the circumstantial evidence of a slow labour progress, the doctor's assessment at 15:00 and the instruction to administer oxytocin, the establishment of the intravenous fluid entry through Ringer's Lactate and finally the more than five contractions per 10 minutes interval which is the evidence of tachysystole which is associated with the use of oxytocin. In addition, the mother was encouraged to push which made things worse for the baby which was also substandard care. He said that there was evidence of abnormal FHR patterns from 12:00 onwards. The labour was augmented with oxytocin without proper assessment. The second stage of labour was prolonged with maternal bearing down efforts despite evidence of hypoxia. The baby was then delivered in need of resuscitation and developed seizures during the neonatal period which then led to a diagnosis of hypoxic ischemic injury. Finally, the neuroradiological images were found to be consistent with the possibility of

intrapartum hypoxic ischemic injury. His conclusion was that the adverse outcome was therefore consistent with intrapartum hypoxia which was avoidable with proper standard of intrapartum care. The delivery took place at 17:10 whereas there were several times where intervention was indicated well before the actual delivery.

[67] Professor Anthony was cross-examined at length. It was pointed out to him that when the plaintiff gave birth on 31 December 2015 she was 16 years old and that in 2014 she gave birth to a child that died soon after birth. He accepted both of these facts. He also accepted plaintiff's evidence that suggested that after the rupture of her membranes which was at 11:00 she was put on a CTG which was subsequently removed before she was told to push which would have been at 16:00 when she was fully dilated. He also pointed out that the CTG was started with the result that the tracings started at 16:02 which coincides with the time at which she was pushing. He testified that even if there was a CTG running before 16:00 his issue was that the FHR before and after contractions should have been appropriately evaluated with a provision for electronic FHR monitoring as well as the correct interpretation of the tracings.

[68] He explained that there is a difference between performing an observation and incorrectly interpreting it. There seems to have been a problem of recording a FHR before and after contractions from about 12:00. Secondly, at the onset of the second stage of labour at 16:00, the nursing staff wrote in their notes that the tracing was reactive when it was probably pathological which also points to misinterpretation. For instance, at 16:00 there were strong contractions and the plaintiff was fully dilated with an urge to bear down. The FHR was said to have been 150 to 155 and the CTG reactive. The plan was to get ready for delivery and to let the mother bear down. If all of that is considered together with the CTG that was running at that time which was probably pathological, it shows that the nursing staff did not understand the foetal circumstances because they misinterpreted the FHR condition. He said that if one goes back to 12:00 the partogram shows O at 160 and X sitting at 140 which is a deceleration. This means the FHR was slower after the contraction. By definition, a deceleration is a reduction of FHR of 15 bpm lasting for at least 15 seconds. If auscultation monitoring is used, the slowing of FHR after a contraction merits further investigation to ascertain that the baby is well.

[69] He agreed that it is in the nature of CTGs to fade after some time and he agreed that in this case the available CTG tracings have faded. He testified that he inked the faded CTG tracings so that they could become more visible. He said that he used a good light and a magnifying glass to discern the components of the tracing to determine the overall pattern and used a rollerball pen to mark the tracing to get an overall picture. There was quite clearly decelerations from 16:00 onwards which were discernible. He then referred to the guidelines where they deal with foetal monitoring. There the guidelines say after a CTG interpretation, write a note about the findings in the woman's notes so that a record of the CTG is still available even if the CTG tracing is lost. He bemoaned the fact that in this case there are faded tracings and no comment from the clinical staff on the tracings.

[70] It was put to him that his enhanced CTG showed a very dire situation. He explained that the correct term was a pathological tracing which is associated with a high risk or high probability of foetal hypoxia and acidosis. If the tracing is pathological, it is likely that the baby is hypoxic and acidotic. However, insult precedes injury and it is the severity and the duration of the insult that will determine the likelihood of the injury. He explained that one cannot look at a pathological tracing and predict the onset of injury. Where the baby is inadequately oxygenated there is a need to intervene to reverse any underlying hypoxia and to expedite delivery as soon as possible. He testified that from the beginning of the pathological tracing there are decelerations with no discernible recovery. There was evidence of a high probability of hypoxia towards the end of the labour which is evident from the tracings. The child was born flat and required resuscitation. All of this was consistent with a diagnosis of intrapartum hypoxia in the delivery room.

[71] From 12:00 onwards there was demonstrably slow progress of labour and beyond the action line. At that point there should have already been a consideration of the fact that the labour progress was too slow for a parous labour. He concluded his evidence by saying that it beggars belief that any trained person in any form of midwifery obstetric care could have looked at those tracings, faint as they are, and thought that they were reactive. They were highly abnormal tracings and ought to have been recognised as such. Proceeding during the second stage of labour with

an oxytocin infusion running with the mother bearing down for half an hour without any consideration for the foetal wellbeing was substandard care.

The defendant's factual witnesses.

[72] The defendant's first witness was sister Mbada who was called to testify as a witness of fact. She testified that on 31 December 2015 she was on duty at the labour ward at the hospital. She was taken through the hospital records starting with an entry made at the clinic at 06:50 before the plaintiff was transferred to hospital. She testified that the plaintiff arrived at the clinic at 06:50. On examination she was found to be 5cm dilated. The first entry at the hospital was at 09:00 which was not done by her. She testified that her first assessment was assessment no.3 which was at 12:50 by which time the plaintiff had a spontaneous rupture of the membrane with clear liquor being observed and the FHR ranged between 136 to 150 bpm. She used a CTG machine to determine the FHR. She noted that the progress of labour was slow at 14:00. This was because the cervical dilation was 7cm whereas it should have been 9cm. Therefore, she remained 7cm dilated for two hours as she was 7cm even at 12:00. Her plan was to advise the doctor about the poor progress of labour and she did that telephonically.

[73] At 15:00 the doctor made a note. The patient was still at 7cm dilated and the liquor was clear. The head was 3 to 4 fifth above the brim. The plan was to administer syntocinon if the CTG was reactive. Once the doctor has ordered syntocinon she also completes a form indicating how syntocinon administration is to be done. That form is then clipped to the front of the patient's file. When it is actually being administered she would tick on that form indicating that she has started the infusion according to the amounts and intervals pre-determined. But if the patient delivers, further infusion is discontinued and there would be a note saying infusion stopped. In this case the form was not ticked at all. This means that syntocinon infusion was never started. With reference to the partogram she explained that at 16:00 on the row meant for decelerations there is a C symbol. That symbol stands for clear liquor. Putting that C symbol there was an error. That symbol should have been written below I even though I should not be there anymore. That I stands for intact membranes. Where there is an I there should be

C instead indicating membranes draining clear liquor as the membranes had ruptured. The O at 12:00 stands for FHR before contraction and X is for the FHR after contraction. The O at 160 and X at 140 mean that the FHR before contraction was 160 and after contraction it was 140. The early deceleration goes down in a V shape after contraction and is expected to go back to the base line. The heart is not beating in the same way it was before contraction as the contractions have exerted pressure on it and the oxygen supply is curtailed. The X would depict an early deceleration. The X and O at 16:00 would mean the same thing.

[74] At 14:00 the progress of labour was poor. At 10:00 until 10:30 the contractions as depicted on the partogram were lighter at less than 20 seconds per 10 minutes. Then the contractions improved to 20 to 40 seconds per 10 minutes and finally the contractions became serious at 40 seconds per 10 minutes. All that is reflected in the manner in which the partogram is shaded. The middle segment of the partogram is between 11:00 and 14:00. The contractions were improving slightly. They were now above 20 to 40 seconds per 10 minutes. Then at 14:00 contractions became stronger. At 15:15 contractions were strong and therefore the likelihood is that she did not start administering syntocinon as that is what syntocinon would have been intended to achieve. If it had been administered, on the relevant block in the partogram where there is medication IV fluid, syntocinon would have been written and the time it was administered. At 16:00 the patient was fully dilated and therefore, syntocinon would not have been administered to a fully dilated patient. The baby was delivered, according to the partogram notes, at 17:34 by vacuum extraction. That entry relating to time was when the entry was made. The entry on comments and complications where it refers to prolonged second stage means that 20 to 30 minutes passed before the baby was delivered. At 16:00 she was fully dilated at 10cm which means she was on the second stage. The baby was born flat with an apgar score of 6/7 and 7/10 at 1 minute after birth and then at 5 minutes after birth. She explained that bag mask ventilation was used to give the baby oxygen as resuscitation as a result of which the baby recovered. There would have been stimulation and then bag mask ventilation. There was a haematoma caused by vacuum extraction. The child was lethargic and the grasp reflex was absent.

[75] She then testified under cross-examination. She confirmed that the guidelines require that there be a clinical record of everything that is done and to ensure that all clinical records are complete and accurate as regards the information they contain. She confirmed that she was at all times aware of her record keeping responsibilities as provided for in the guidelines. She testified that she was trained on how to complete a partogram and how to interpret it during her training. She confirmed that there is poor labour progress if the cervix dilates at a rate of less than 1cm per hour in the active phase and crosses the partogram alert line. She further confirmed that the second stage of labour is prolonged if delivery has not occurred after 30 minutes of pushing in a multipara and that the plaintiff was a multipara. She confirmed that if the patient is already in hospital and crosses the action line, action is mandatory and therefore there must be syntocinon infusion or a caesarean section. She confirmed that she had no independent recollection of what happened to the plaintiff and relied on what was written in the maternity case records for her evidence.

[76] She testified that the plaintiff would have been in the active phase at 06:50 that morning because at that time she was 5cm dilated. She confirmed that in this case the partogram was only started at 10:00 with the dilation being 5cm at the time and therefore it was not correctly completed. She confirmed that she did not pay attention to the fact that the partogram was started incorrectly when she took over at 12:00. She testified that the notes or entries on the partogram are all hers save for the ones at 15:00 which were made by Dr Cilliers. At 14:00 she recorded that the dilation was 5cm according to her partogram entry but that was incorrect because in her clinical notes she had noted it to be 7cm which was a mistake on her part as the patient was actually 7cm dilated at 14:00. She confirmed that from 12:00 when she took over monitoring the plaintiff until she gave birth at about just after 17:00, five hours had passed. She testified that it was not possible that from 12:00 she checked the plaintiff once as the partogram seemed to suggest. She might have forgotten to record on the partogram everything she did.

[77] Her first assessment was at 14:00 according to the assessment notes. However, she did make an assessment at 12:50 and made entries not on assessment no.3 but on the clinical notes. The necessity to write on the clinical

notes was caused by the change with water breaking which she regarded as a remark as against an assessment even though she did assess the patient at 12:50. She also did not record that in the partogram because there was no space and she had already indicated in the partogram that the membranes were intact. She accepted that she ought to have recorded the FHR on the partogram and she made a mistake in not doing that. She also accepted that the time at 12:50 was overwritten as well as the date. She testified that she does her assessments two hourly in the same way she does with the partogram. She denied that the entry she made at 12:50 in the clinical notes was made later than that time. She wrote it as a remark on the clinical notes and not as assessment no.3 and the partogram because two hours had not yet elapsed.

[78] She confirmed that according to the assessment at 12:00 it was done by her colleague, sister Bingwa but according to the partogram entry the 12:00 assessment was done by herself. Her explanation for this discrepancy was that sister Bingwa was a newly appointed nurse for whom it was not easy to remember to make entries both in the assessment and the partogram. She then had to fill the gap. She testified that all she recorded at 14:00 was that the FHR was 145 to 150 bpm. There was no indication of contractions that would have been observed on the CTG. There were no decelerations recorded and there was no indication whether the CTG was normal or suspicious. There were therefore no notes of what the CTG showed. She insisted that there was a CTG that was running as the contractions were not normal and the labour progress was also not normal but she could not remember whether she was in fact on a running CTG at 14:00 but procedurally that should have been the case. She confirmed that according to the guidelines the FHR in the active phase should be assessed half hourly. She said she was not sure if the FHR was assessed every half an hour as it should have been the case.

[79] She confirmed that from what is recorded on the partogram, from about 11:00 up to the end, there were only two contractions per 10 minutes. She confirmed that at 16:00 the heart rate was 170 and after the contraction it was 140. There was a non-reassuring FHR at 12:00 and at 16:00 and nothing was recorded in between. She accepted that under decelerations in the partogram she recorded C which was completely wrong as C should refer to clear liquor. Under decelerations she put an I

which stands for intact membranes. She explained that this was because she panicked as she tends to when dealing with a young woman. She confirmed that there should have been entries of membranes rupturing and clear liquor draining from 12:50 and that they were not there until 16:00 when a completely wrong entry was made.

[80] She confirmed that at 06:50 the plaintiff was 5cm dilated and at 09:00 she was still 5cm dilated and therefore for two hours there was no further dilation as normally there should be 1cm dilation per hour. Therefore, at 09:00 already there was poor progress. The next assessment was at 12:00 by which time she should be 8cm dilated as normally over three hours as from 09:00 to 12:00 there should be 3cm dilation. At 12:00 she was 7cm and at 12:50 when membranes ruptured she should have been at 8cm. She accepted that she did not check the cervix for dilation at 12:50. She explained that this was because two hours had not yet lapsed. At 14:00 when she did the next assessment the plaintiff was still 7cm and therefore for two hours there was no further dilation which called for action. She informed the doctor who prescribed syntocinon but to be administered only if CTG was reactive. She accepted that she should have made a note of what the CTG showed as instructed by the doctor at 14:00. She generally did what the doctor ordered. She insisted that it was not possible that she did not start the CTG but could not remember the events of that day but she did not do the syntocinon.

[81] She accepted that the only reason for not starting the syntocinon administration would have been if the CTG was not reactive. She later changed to say that the condition of the patient could have changed which would result in her not administering syntocinon even if the CTG was reactive. It was put to her that the instructions from the doctor was at 14:00. It would take her 30 minutes for her to observe the CTG. This means by 14:30 there should have been a note saying the CTG was reactive or not reactive. If it was reactive syntocinon would start according to the doctor's orders. She accepted these postulations. She testified that she would not have ignored doctor's orders. The reason she did not start the syntocinon was not because the CTG was not reactive. The possibility was that contractions would have changed from that time to 16:00 and therefore there would be no need to administer syntocinon when what it was sought to achieve was happening.

[82] She confirmed that the frequency of contractions did not increase from 10:00 to 14:00 when she got the instructions from the doctors. However, she testified that there was a great change because the contractions were stronger. At 16:00 the dilation changed which is why she did not carry out the doctor's orders. The frequency of contractions was not improving but the contractions were intense. From 14:30 to 15:00 the doctor arrived and made a note at 15:00 and recorded her findings. Her notes indicate that the dilation was still 7cm, which is what it was at 14:00 and at 12:00. It was then put to her that if the CTG was running the doctor would not have told her to check if CTG was reactive before syntocinon infusion and catheter insertion. Sister Mbada testified that she was unable to confirm that the CTG was not running.

[83] Sister Mbada was then referred to the guidelines where they deal with partogram alert and action lines. The mandatory action is the transfer to hospital, the oxytocin infusion at the hospital or a caesarean section. The doctor indicated that the plaintiff had crossed the action line and decided that syntocinon infusion must be done. The syntocinon infusion would be done through a drip in which it is injected in a fluid called Ringer's Lactate. Sister Mbada confirmed all of these propositions put to her. It was further put to her that at 15:00 when the doctor made entries on the partogram she did not indicate what the FHR was at that time. Therefore, if the CTG was running continuously the doctor would have written the heart rate at 15:00 and would have indicated if there were any decelerations. She maintained that she did not administer the syntocinon. In that regard she relied on the fact that if she had administered it she would have ticked the syntocinon infusion document indicating the infusion rates as well as the times. Secondly she would not have done the infusion because of the increased intensity of the contractions.

[84] She confirmed that the Ringers Lactate was the way in which the syntocinon infusion was to be done. It was put to her that she did administer syntocinon as instructed. Her evidence in this regard was that according to her recollection she did not do it if her memory was correct. However, she confirmed that she had no memory of this particular case and relied on the maternity case records. She also confirmed that she would not have ignored the doctor's orders. However, as an

independent nurse she can ignore the doctor's instructions if the situation changed. The intensity of the contractions improved and the patient was experiencing some birth pains. On this latter issue she relied on the shading of the partogram on the contractions portion from 14:30. It was pointed out to her that the doctor examined the patient at 15:00 by which time she would have been aware of the contractions before 15:00. She agreed that the frequency of the contractions did not change. It was put to her that even the intensity of the contractions was the same at 15:00 as it was at 14:00. Her evidence in this regard was that the patient was experiencing strong contractions even though their frequency had not improved. She was therefore satisfied as a midwife with an independent function. She therefore exercised her discretion and overruled the doctor.

[85] She confirmed that on 31 December 2015 she was still a registered nurse and not a midwife and that she did her midwifery qualification in 2018. She was trained in interpreting CTGs and how to complete a partogram. It was put to her that she recorded a heart rate of 170 before contractions and 140 after contraction which is different to what she recorded in her assessment no.4 of the hospital records where she indicated that the CTG was reactive. Her explanation for this discrepancy was that when the patient pushes the heart rate picture changes. She confirmed that at that stage she was not pushing yet, she merely had an urge to bear down. She then said that the urge to bear down has got a way of causing the heart rate to change. She confirmed that the heart rate in assessment number 4 which was 150 to 155 bpm was different to what she wrote on the partogram. Her explanation for the discrepancy was that when she wrote an assessment based on a running CTG, by the time she wrote on the partogram there would be a change. It was then put to her that if that was the case she would have seen on the CTG that the heart rate was non-reactive as it would have changed to 170 before contraction which is tachycardia and abnormal as it is higher than 160 and then after contraction it was 140.

[86] At this stage of the cross-examination sister Mbada went on a tangent about her experience of delivering babies which she said in some cases she would think that the child would not be alive because the patient had become uncontrollable with the baby also pushing out on its own especially when the baby is in the perineum. But

such babies would come out perfect and crying vigorously. Therefore, at this stage of birth, the second stage especially when the baby is in the perineum the CTG may be grossly abnormal but the baby comes out alive and crying. She agreed that her partogram entry at 16:00 showed a heart rate that was not normal. She accepted that what she recorded in the partogram indicated a late deceleration. However, what she wrote was a C not an L. The C related to clear liquor. If she had seen a late deceleration she would have recorded it with an L. She insisted that her entry was not an L but a C even though the heart rate was 170 and 140 before and after contraction. She agreed that the block on which she wrote C is meant for deceleration which is either E/V/L/N and there is no C that is applicable. However, her intention was to write C not an L. She later accepted that she should have written an L for late deceleration. She accepted that she therefore could not have written that the CTG was reassuring. It was put to her that at 16:00 there were two contractions per 10 minutes. She accepted that it was an abnormal CTG. It was put to her that the CTG showed at least 8 contractions per 10 minutes. She accepted that such a CTG would lead to a suspicion of foetal distress which is when the FHR is abnormally high or abnormally low.

[87] The next witness for the defendant was Dr Linde. Her evidence was that on 31 December 2015 she was stationed in the maternity ward as the medical officer in charge. The patient had been seen at the clinic at 06:50 and was referred to hospital where she was attended to from 09:00. She had been in labour since 02:00 am and she was 5cm dilated. The plaintiff's second assessment was at 12:00 and her 3rd assessment was at 14:00. During all these three assessment she was not involved. An entry was made at 12:50 that there was a spontaneous rupture of membranes. The third assessment at 14:00 which was done by sister Mbada indicated that there was no progress since the last assessment at 12:00. The 3rd assessment which was at 14:00 was done on time in that a patient has to be examined every two hours. She was 7cm dilated with the cervix still thick or the presenting part or head was still high up or above the pelvic brim. All of these were indicative of poor labour progress.

[88] The plan was to inform the doctor about the poor progress. At 15:00 she recorded her own assessment which she had done. According to the partogram the

patient had crossed the action line which was also indicative of poor progress. The CTG would have been reactive because the plan was based on its remaining reactive which was the syntocinon infusion to augment the labour. She would also be put on a catheter in order to empty the bladder to remove any obstruction. In the absence of risk on allowing labour to continue it would be allowed to continue. In that case it would not be necessary to immediately perform a caesarean section. Finally, part of the plan was that the doctor would be called if there were any problems. She confirmed that she did not make a note that in fact the CTG was reactive at the time. However, if there was any evidence of a non-reactive CTG or any decelerations of the heart rate she would have been concerned about the foetal distress. She plotted her dilation finding of 7cm.

[89] She personally put up a drip to give fluid to support the patient in poor progress to prevent dehydration so she connected her to the drip which was Ringers Lactate. The syntocinon administration infusion is done by first ensuring that the CTG tracing does not reflect a worrying FHR to exclude any sign of foetal distress. If there were signs of foetal distress, the syntocinon administration is contraindicated. It would only be started based on a reactive CTG. Every woman in labour has their own oxytocin but sometimes to augment labour an artificial oxytocin called syntocinon is used if the contractions are either too weak or too uncoordinated. At Zithulele Hospital they had a specific prescription sheet that was used which indicated the rate it was to be given as well as the time. Where the patient is a grand multi-gravida, meaning she had had many pregnancies and labours before, it would be risky to augment her labour. The second thing would be to check that the progress of labour had been plotted at least 2 hourly on the partogram. Thirdly the foetal heart rate is monitored through a CTG while syntocinon is being infused and if continuous CTG monitoring is not done or possible, syntocinon infusion cannot be started. If the CTG shows too many contractions or any foetal distress, syntocinon should be stopped immediately as it can be stopped at any point if it is unsafe to continue with it. This would be the case if there is an overstimulated uterus which is five or more contractions per ten minutes which would reflect on the CTG. Also if FHR is becoming too fast or showing signs of decelerations or any signs of foetal distress syntocinon should be stopped immediately.

[90] She testified that the prescription itself does not confirm administration. There does not seem to be confirmation that it was in fact given. Medication that has been given is documented. When the Ringer's Lactate was put up the syntocinon administration did not start. If it had been administered there would have been a tick on the infusion chart. The patient was fully dilated at 16:00 and depending on the situation one could keep it running even if she was fully dilated. She testified that it was unclear if syntocinon was actually administered or not. However, it was unlikely that it was given as there was nothing to document its administration. There was no note of the syntocinon in the partogram or in any of the following assessments. There was no time at which she was concerned about the welfare of the foetus. A consideration to augmenting the contractions through syntocinon would be given if there are no other risk factors and an emergency caesarean section is not needed. The bladder is most commonly the biggest reason for the slow progress and if membranes are not ruptured one would need to rupture it which normally causes labour to happen quickly. On CPD or cephalopelvic disproportion her evidence was that this would mean that the head is not able to pass through the pelvis.

[91] Under cross-examination Dr Linde testified that by the end of 2015 she would have had about six to eight months experience in the maternity ward. She appreciated the importance of making a note of everything she did as provided for in the maternity guidelines. It is provided that a note should be written about the findings of a CTG monitoring process so that a record of the CTG is still available even if the CTG tracing is lost. She confirmed that she found the plaintiff to have crossed the action line when she assessed her at 15:00 indicating poor labour progress. A normal labour should progress at 1cm dilation per hour and in this case it was much slower. In that case where action line is crossed, if there is no foetal distress that action could be syntocinon infusion but if there is foetal distress the action is a caesarean section. But things like emptying the bladder or even rupturing the membranes are some of the actions that could be performed when the action line is crossed. In this case membranes had already ruptured at 12:50 and while the bladder was still full it was unlikely to be the reason for the poor progress but she had the catheter put in to empty it so as to rule it out.

[92] When she saw the partogram and noticed the poor labour progress she determined that there was a need for syntocinon infusion to make contractions frequent and to make the labour progress quickly. She noted that the patient was 7cm dilated with clear liquor and the head was 3 to 4cm above the brim which was still very high up. She agreed that no FHR was noted and that FHR should be monitored every 30 minutes by auscultation or CTG monitoring. She confirmed that she did not make a note of what the FHR was on the partogram. The plan was to start syntocinon and to put a catheter and to allow labour to progress and to call the doctor if there were problems. CTG was to be checked before syntocinon infusion and the catheter insertion. She admitted that it was possible that if she had seen the CTG she would have written a note thereof.

[93] She admitted that she had a duty to make notes of the conditions she found from the examination. Any mistake with syntocinon infusion may cause foetal distress with bad outcomes for the baby. The guidelines do provide that there must be no evidence of foetal distress before syntocinon infusion and that it must be used with great caution in multiparas and after the CPD would have been excluded. It was then put to her that if there had been a CTG running she would have written what the FHR was and what the contractions were. She testified that while a CTG finding was a central finding practically it would be almost impossible to make the note that she did without having seen a CTG. She understood her note in that regard to mean that if the CTG remained reactive the syntocinon and catheter could be done. She agreed that the latest heart rate recorded on the partogram closer to 15:00 was at 14:00 which was an hour before. She therefore, agreed that from 14:00 there was no record of a reactive CTG but she denied that there was no CTG monitoring. As for putting up Ringer's Lactate her evidence was that it could have been in preparation for syntocinon. But it is also used very often in prolonged labour where the patient would not have been eating or drinking. It was common to run fluids for such patients and therefore denied that the sole purpose of putting up Ringer's Lactate was to administer oxytocin. If after putting the catheter on and the CTG remained reactive and the progress was still poor the intention was for the nurse to inject the syntocinon into the Ringer's Lactate fluid.

[94] She confirmed that nurses and midwives would not start a medication without prescription from the doctors. But they could increase or discontinue it or lower it depending on the contractions. They could not refuse to administer it if it was prescribed. But if the patient's labour progressed before the infusion started they may not administer it. For instance, when a catheter is put in sometimes things change unexpectedly. In that situation a midwife could decide what to do if syntocinon was no longer needed.

[95] She agreed that if the progress of labour crosses the two-hour action line it is required that syntocinon infusion should be started if there is no CPD and no evidence of foetal distress. She was satisfied that there was no foetal distress and there was no indication of CPD. She was therefore required to start syntocinon infusion and preparations for it were done. However, it was very difficult for her to say it was actually infused as there were no notes to support that conclusion. Her intention after her assessment at the time was that the patient could benefit from syntocinon augmentation. She agreed that a properly drawn partogram should look like exhibit 1G drawn by professor Anthony and therefore the partogram for this patient was incorrectly drawn. Therefore, the labour in this matter was severely delayed and the contractions were inadequate. Inadequate contractions call for syntocinon augmentation. There was no indication or entry of syntocinon infusion or that it should be stopped. It was put to her that at 16:00 there were two contractions per 10 minutes and she was 10 cm dilated and if syntocinon was infused it had to be continued until birth. She agreed that there was no note indicating a change that would have made syntocinon infusion unnecessary but there were a few missing links to syntocinon administration because of the absence of ticks for it. But there was missing information both for and against the possibility of syntocinon administration.

[96] If there was tachysystole from 16:00 until the CTG was discontinued about half an hour later, which indicates going from very poor contractions to tachysystole, that would point strongly that syntocinon had been administered. She testified that tachysystole would be when the contractions were happening too frequently and too quickly which causes a problem for the oxygen supply to the baby. But that could happen during labour even without syntocinon. The effect of syntocinon would be to

increase the frequency and strength of contractions. But contractions can increase both in frequency and strength on their own without syntocinon administration.

[97] Dr Cilliers testified that she was on duty at Zithulele Hospital on 31 December 2015 where she had been a grade 2 clinical medical officer since 2013. She was asked to come and assist with the vacuum extraction delivery. The process started at 17:05 and was completed at 17:10. She had been asked to come and assist by the first on call, Dr Phillips saying there was a delayed second stage labour. The CTG was reassuring, the assessment of the pelvis was normal, the patient was a 16 year old grav-2 para zero with a second stage delayed labour. Dr Phillips was a junior doctor doing his community service having been at the hospital for six months. So it was her responsibility to assess the situation as a senior doctor and confirm if vacuum extraction was the correct procedure. She did all the examinations and she was satisfied that the case was a good case and a learning opportunity for Dr Phillips to be skilled. When she arrived in the ward the patient was on a running CTG and there was Dr Phillips who would have been called by the midwife. In that setting there were two patients, the mother and the baby. She needed to determine the wellbeing of both patients. One of the pre-requirements for a vacuum extraction is to look at the foetal wellbeing. This is necessary to assess if the baby is well enough to go through the process of vacuum delivery. This is done through a running CTG where the assessment is whether the CTG is normal, suspicious or pathological. The baby has to have enough reserve to go through a vacuum normal vaginal delivery from a reading of CTG tracings. The notes she made on the day indicated that initially the CTG was good which means it was reassuring and then tachycardia. Foetal tachycardia meant that there was a stressful situation but it was not a pathological one. A pathological one would be bradycardia, a very low FHR which is the concerning one and if that was the case she would have made a different assessment.

[98] Her assessment was that the foetal well-being was good and she excluded foetal distress so that she could carry on. She checked the cervical dilation and the mother was fully dilated. She had with her the maternal records including the assessment notes which had been recorded at various times. She was then referred to the maternal notes which indicated that the baby was born a bit flat but recovered

after bag mask ventilation and had a heamatoma caused by the vacuum cup. She testified that in the first minute after birth while doing apgar scores and assessing the foetal wellbeing, if there is a need to help the baby breathe more, to supply more oxygen you first start with stimulation and then move towards bag mask ventilation. That led to a good recovery and the five minutes' assessment showed the baby to be no longer blue but pink which means that the baby was well oxygenated and breathing better. She did the bag mask ventilation herself. She was on duty during that whole weekend but at some point on 31 December 2015 she handed over to Dr Mans who saw the baby at about 22:00. She only saw the baby again on 3 January 2016 which was the last time she saw the baby. She made a note that the baby was breast feeding. She noted the need for counselling with the grandmother to provide breast feeding support. There was also a note for a speech therapist and dietician intervention to support the mother with the baby's breast feeding. The baby was still cup feeding, with no active breastfeeding. The plan was to help the mother to do breastfeeding before she was discharged by establishing good breastfeeding practices. The note on speech therapy and dietician intervention reflected that the baby was only cup feeding and needed assessment by a speech therapist. This was a multidisciplinary approach to deal with the cup feeding situation so that dietician and speech therapist would come and help in establishing breastfeeding.

[99] Under cross-examination she confirmed that for respiration she gave the baby an apgar score of 1. She confirmed that the record did not reflect that the baby was crying. She put the baby on nasal prong oxygen and after she was happy with her breathing she went to the theatre to deal with the 3rd degree tear the mother had sustained. At 18:30 the bay was still on nasal prong oxygen. It was put to her that an hour and a half after birth the baby was not yet normal. She testified that if she was concerned about the baby's neonatal outcome she would have admitted her to the neonatal high care unit and put up an IV line and not to initiate breastfeeding. She would have written good notes for monitoring for an extensive plan. Her assessment was that the baby was well enough. The apgar score of 7 at 5 minutes was a good apgar score according to the guidelines and was not concerning. She testified that every baby that is born is resuscitated to stimulate it and this is done within 30 seconds of birth and therefore resuscitation is not indicative of a bad

outcome. She however, agreed that an apgar score taken on a resuscitated baby is an assisted score.

[100] If she had been worried, she would have admitted the baby to the high care unit for a higher level of monitoring. If she had time she could sit next to the baby, take off the nasal prongs and make sure that the baby was 100% off oxygen and do a non-assisted apgar score. On the baby being born flat and lethargic an hour and a half later, the baby still receiving oxygen according to the nurse her evidence was that that changes everything significantly but according to her notes she was convinced that the baby was okay which was why she did not admit her to high care. When it was put to her if she accepted that the baby was not okay, she testified that she would accept that. She was referred to grasp reflex which was weak and another fundamental finding that the suck reflex which was absent meaning the baby could not latch or feed from the breast. Even at 22:34 a note was made by the doctor about poor latching which would be a consequence of an absent reflex. She testified that there were two components to poor latching. It also involves not just the baby but the mother as well in getting the baby to breastfeed but she could not comment much as she was not there at that stage.

[101] Dr Cilliers testified that when she first arrived to attend to the patient there was foetal tachycardia which meant that the baby still had reserves. The CTG that she saw that was running was normal and there was a good response from the baby's heart rate during that time. All of that told her that the baby was well. When the baby was born, the apgar score was 6 and 7 and 10 later which was a very good outcome for a delivery. A diagnosis of a poor foetal outcome is made if the apgar score is less than 3 according to the guidelines. On that basis she could not have picked up that there was ischemia. The Standard Treatment Guidelines of Paediatrics of 2017 say if the one minute apgar score is less than three and the five minute apgar score is six or less, that is a poor outcome which is more suggestive of a diagnosis of HIE. When she assessed the baby, none of that criteria was met. She testified that according to her notes when she arrived, the CTG was initially good and then tachycardia. She testified that while she did not have a recollection of what the CTG showed, she did write in her own handwriting that it was initially good but then tachycardia which was when she made the note on the vacuum delivery.

She therefore presumed that the CTG was running at the time because that is what usually happens. She would not have made such a comment or note if she was not reading from a CTG. The notes are made retrospectively. If there was a pathological CTG she would not have proceeded without assessing other emergency measures to take to get the baby out as soon as possible.

[102] The tachycardia was at about 17:00 although she could not tell exactly when it started because they work on the presumption that the CTG trace would be available. Some of the indications for a safe vacuum delivery are that the head of the baby must be in the perineum which means the head must be down at the base of the pelvis. The mother must be 10cm dilated and if the baby's delivery is delayed then the vacuum or ventouse delivery can be done when all those clinical signs are indicated. Consideration is given to the fastest way of getting the baby out. She testified that tachycardia is a heart rate of about 160 or higher. But if it is more than 160 for an extended period of time, that is more concerning. When it is above 160 it is non-reassuring. If it is non-reassuring you must consider it in detail to see what other features are there on the CTG. Those would be things like variability and the contraction pattern. These help determine if the CTG is in fact reactive or nonreactive. She admitted that she did not do any of those further investigations. She was satisfied that the condition of the baby and the mother was such that a ventouse delivery could be done relatively safely. She admitted not writing her other observations but said if there was a problem she would have written it down along the lines that the CTG is pathological with poor beat to beat variability. So when there was foetal tachycardia, that was indicative of a suspicious CTG trace as against a pathological one.

[102] A suspicious trace has a low probability of hypoxia. Her noting foetal tachycardia means there was sufficient reserve, a suspicious CTG but the baby could be delivered normally. If there was poor beat to beat variability or if she was worried about prolonged decelerations she would not have allowed a junior doctor who has never done vacuum extraction by himself to do it on a pathological CTG. She would have gotten the baby out herself as the most experienced clinician there. She agreed that to assess whether a CTG is suspicious or pathological it has to run for a certain period of time. At one time a CTG may be suspicious at other times it

may be normal and yet at others it may be pathological. There was no obstruction for the baby to come out. She did not do an episiotomy because they did not think that it was an obstructed labour. They do it if they think that the perineum is too tight and will not stretch and they rarely do it as they do it only in extreme cases. She denied that the third degree tear was caused by the head of the baby being big relative to the vaginal opening. She testified that she did not think that that was a problem at all as there was no moulding, no caput and no oedema. The vaginal examination done before the baby was born was not indicative of an oedema. The cause of the third degree tear in her view was because Dr Phillips was not skilled enough to do a vacuum extraction without causing a tear.

[103] The vacuum extraction was very easy in that there were only two pulls. It started at 17:05 and by 17:10 it was completed which was 5 minutes later. She confirmed that the first stage was delayed. She testified that if there is a delayed first stage of labour you intervene but that does not mean the baby is hypoxic. If there was foetal distress the syntocinon augmentation would not have been considered. A caesarean section would instead have been considered. This was not an abnormal labour at all. She agreed that according to the guidelines there was poor progress in the active stage of labour. She testified that from 16:00 to 17:00 which was an hour she progressed from 7 to 10 cm dilation. Two hours for delayed second stage of labour is allowed according to the guidelines. When she arrived and saw poor progress she could see that there was intervention and it worked and there was a good CTG. She wanted to get the baby out as soon as possible as she did not want to wait for the two permissible hours. She remembered a vigorously crying baby following a very good vacuum extraction performed by one of her junior colleagues whose outcome was good because the baby was crying.

[104] It was put to her that taking everything together from poor progress going into the second stage, the poor respiration on which she acted correctly, she should have had a high suspicion that there could have been foetal distress. She testified that excluding the apgar score of 10 out of 10 at 10 minutes given by sister Mbada, still on apgar score of 6 and 7 out of 10 and a crying baby was very good. An apgar score of 7 out of 10 at 5 minutes was good but if it was less there would be a worry of an acute incident of hypoxia in which case an IV Line is put up and the baby is

kept at nil per 02 and it is admitted to high care unit. She testified that without taking anything away from Dr Mans, if she came back from theatre and there was a problem and concern she would have picked it up at 18:30 and would have made extensive notes at 18:30 saying the baby is not doing well, maybe she missed a foetal distress. Instead the note she made was that the baby needed to start breastfeeding and was hungry. At 19:00 the baby was with the mother in the labour ward which is a low risk ward so the baby was not in a high care unit. She did not know why there was poor latching which Dr Mans noted. The cardiovascular system was good, good respiration, the spine was normal and neurologically the baby was fine.

[105] It was put to her that when she saw the baby at 18:30 it was not well and that she missed clear signs like absent suck reflex which leads to poor latching. There were clear signs of encephalopathy which she missed as a result of which she should have referred the baby to a higher facility or ICU and not return it to her mother. She disagreed with all of that proposition.

[106] The last witness of fact for the defendant was Dr Mans. He testified that on 31 December 2015 at night he was the doctor on duty. His shift started at 19:00 and went through the night until 08:00 the following morning. During that period, he was the only doctor on duty. Emergency cases are highlighted during the handover. On the 31 December 2015 no specific problem was mentioned that he could recall. He saw the baby on 31 December 2015 as part of his normal duties. This baby was not a patient that was highlighted during the handover. The post-neonates are generally well and low risk and are therefore easy to attend to. The doctor would see one patient, rush to emergency and back to the ward to do the next patient. With respect to this patient his note was done at 22:34. This means that he would have been busy with other duties before doing the post-neonates round. Before he made the note at 22:34 he would first have spoken to the patients. He noted that the baby was hungry and there was poor latching and also wrote, cardio-vascular system to look at the heart and pulses. He did the respiratory examination and he was not worried. He checked the spine and also did a neurological examination.

[107] He saw both the baby and the mother at the same time and noted that the mother had no clue on how to breastfeed. He would have asked the mother to breastfeed while standing next to her and could see that she was not able to position the baby comfortably to latch on to the nipple. The plan was to review the baby on discharge. This meant that at that point there was nothing of concern to him about the baby who was essentially lodging until the mother was counselled and her perineum wound was dealt with. His reference to cardio-vascular system review was because the baby was crying. He has done informal training in breastfeeding and was aware that there were many people who struggle with connecting the baby well to the breast. His assessment based on the notes was that the baby looked ready and willing and wanting to feed but was not being positioned optimally for breastfeeding. The mother needed to be taught on how to do it. His expectation was that his colleagues who would come after him would see the notes and do the necessary instead of processing the patient routinely. The next morning at 07:30 on 01 January 2016 he noted that the baby was crying and there was poor latching and was hungry. By that he meant that the baby wanted to feed but the connection between the baby and the mother's breast which is called latching was not good. The plan was to cup feed and then breast feed. They had a situation of a feeding difficulty in a baby that appeared to be able to feed and a feeding connection or latching that was not good.

[108] He saw the baby again on the 04 January 2016 at 10:19. He noted that the baby was not breastfeeding properly after looking at the feeding which he presumed to be a contributing factor to the baby being slightly yellow meaning the serum bilirubin levels was slightly high. The plan was for a dietician and speech therapist interventions which is a multi-disciplinary approach. Breastfeeding counselling was normally done by the dietician and the speech therapist at Zithulele Hospital. The plan was also for the total serum bilirubin levels to be confirmed through the necessary test. He saw the baby again on 04 January 2016 at 16:22 after being called by a nurse reporting that the baby was fitting. At that point the baby appeared yellow and his note therefore referred to jaundice. It seemed to him that there had been a deterioration because the baby was fine for the first three days and then had fits all of a sudden. He then questioned or queried HIE as he did not know what could have led to the insult in the brain leading to seizures. He then made a plan to

look into the situation and manage it in line with their set protocol and operational management book. He made further notes at 18:21 as he spent time with this baby trying to understand the situation. The next note was on 05 January 2016 at 07:39 and it indicated that there were no further seizures since the previous day at 21:26. His issue was that there seemed to have been a shift from the earlier picture from birth as depicted in the notes. He made further notes on 05 January 2016 at 14:45 when he did a ward round. He made a note referring the baby to the occupational therapist. As he was the doctor in the labour ward that week he saw the child again on 06 January 2016 and the whole patient management process continued and his notes ended.

[109] His evidence under cross-examination was that when he starts his round he would speak to the nurses to hear about problems. He would then go to the labour ward and speak to all the mothers after which he would speak to each mother individually. He speaks to the mother while examining the baby. Then he would go to the file and look at the delivery time and apgar scores. He saw Dr Cilliers' notes including the fact that the baby was on nasal prongs for oxygen. He testified that when he saw the baby it was not on nasal prongs. He agreed that there is reason to believe that there may be a risk of hypoglycaemia as the glucose level may be depleted after a baby has suffered hypoxic ischemic injury. He added that there was no recorded risks of hypoglycaemia. He testified that they have high regard for abnormal findings that are reported and the procedure is that if a nurse makes an abnormal finding the doctor must be informed immediately. It was put to him that because of time limitations he did not consider the condition of the baby after birth properly. He testified that he examined the baby and he felt that the baby could latch. He had examined the baby twice and there was no scalp trauma. The nurse's note on a haematoma must have been a thumb suck.

[110] If there was an issue of a suck reflex that was absent, he would have been called immediately. He was referred to the notes of a speech therapist regarding an absent rooting reflex and suckling difficulties the baby had and its sleeplessness made on 04 January 2016. He insisted that any suggestion that his initial assessment was incorrect was denied by him. He said that the baby was clearly crying and active and moving all the time during the first two days and therefore his

initial assessment was not incorrect. He testified that his recorded notes showed that he watched the baby breastfeeding. When he made reference to poor latching it meant that he saw the baby moving to the breast on 31 January 2015 at 22:34. On 01 January 2016 at 07:30 there was another note on latching and the baby being hungry. That was indicative of an active baby wanting to latch. In the morning on 04 January 2016 the baby was not breastfeeding nicely which meant that he saw that the baby's breastfeeding was not happening nicely. There was no comment on 4 January 2016 on his notes about the level of consciousness of the baby. He agreed that feeding difficulties are common in cases of HIE and they are typical features of hypoxic ischemic injury. He then said that while poor feeding and lethargy are associated with HIE, his starting point was the mother's inability to breastfeed the baby. Once that is addressed they then look at other features. Where the baby is crying, moving well and active, an association is not normally made with HIE. He agreed that this baby had HIE and did end up having sucking problems. But at the point of his examination of the baby there was no HIE concern as the baby did not have the classic features.

The defendant's expert witnesses.

[111] The first expert witness called by the defendant was Dr Koll. He is a semi retired specialist obstetrician and gynaecologist. He compiled a report and also did joint minutes with Dr Murray and with professor Anthony. Based on the information that they had at the time he and Dr Murray concluded a joint minute accepting that syntocinon had been prescribed and administered on the plaintiff. He testified that antenatal assessment of a foetus can only indicate that the baby is alive and measuring fundal height only indicates that the baby is growing. There is no way of making a neurological assessment of the baby. Therefore, an injury or abnormality, be it congenital or genetical that was present in the womb prior to the onset of labour, routine antenatal care would not have picked it up. Radiologists can time it to the peripartum area, in other words late antenatal to some time after birth. That is why he and professor Anthony agreed in a joint minute that there was nothing detectable antenatally that could have affected the outcome.

[112] In other words the antenatal course progressed fairly uneventfully. He was referred to Dr Murray's comment in their joint minute in which she said that the foetus had several significant labour related risk factors for hypoxic brain injury. Those were the prolonged labour, the oxytocin infusion, the substandard foetal monitoring especially during oxytocin administration and the fact that the second stage of labour was complicated, needing instrumental delivery. The minute indicated that he did not disagree with those postulations. He explained that there is a difference between risk and cause. There were discrepancies in the assessment of the cervical dilation. The doctor assessed the patient when she perceived that the patient had crossed the action line. In the absence of disproportion, she decided to augment the labour. The labour progressed rapidly after augmentation thus justifying the decision of the doctor. There was CTG monitoring as evidenced by the recording of FHR ranging between different figures. That can only be done on a CTG. One can also see a range on a handheld doppler. At 16:00 the CTG is reflected as reactive and at 16:00 the FHR was 150-158. His view was that for a large portion of the labour process the patient was on a continuous CTG although the detail when she was on it and when she was off it is unknown.

[113] He testified that there are things that should have been filled in on the partogram which were not and therefore it does not document the full picture. The definition of tachysystole is more than five contractions in a ten minute period. That indicates that the uterus is being overstimulated which, as professor Anthony illustrated, may lead to progressive hypoxia threat as labour progresses as a result of relative lack of oxygen. Therefore, tachysystole needs to be managed on a fairly urgent basis. The CTG monitors only two things, the heart rate by giving a tracing of a heart rate of the foetus. That has four elements; variability, the beat to beat variability which suggest a healthy baby. Then there are decelerations where you get early decelerations, variable decelerations and late decelerations. Early decelerations are a mirror image of a contraction. Variable decelerations are not related to contractions and late decelerations occur regularly after a contraction. The one looks for the presence of accelerations. You look for baseline variability, presence of decelerations and accelerations. If accelerations are present, that is a very good sign of foetal well-being. That is the cardio part of the CTG. The tocograph simply measures the surface tension on the mother's skin. While the CTG

gives a very good representation of contractions, it is not so good during the second stage because of the mother pushing and changing positions.

[114] While writing notes in a busy labour ward is very important, however, he was of the opinion that the primary responsibility of a clinician is to care for patients as one cannot sacrifice care of a patient in order to write comprehensive notes. He agreed with professor Anthony that a random measurement of the foetal heart does not tell anyone anything. The assessment at 12:00 indicated foetal movement and the plaintiff was 7cm dilated. So from 10:00 until 12:00 the progress was adequate. The next assessment was due to 14:00 at which time the cervix remained 7cm dilated. The FHR was 145-150 bpm. The head was high and the plan was to inform the doctor about poor progress. The doctor ordered syntocinon with an instruction that if CTG was reactive it could be started.

[115] At 15:00 Dr Linde made a note which also noted that the patient had crossed the action line and ordered syntocinon, a catheter and that CTG was to be checked if it was reactive before those things were done. Syntocinon was to be administered in half an hour if the CTG was reactive. At 16:00 the patient was fully dilated. He and professor Anthony were in total agreement that oxytocin is a dangerous drug which is to be used with extreme caution especially in a multipara. So if sister Mbada felt that something had changed and decided not to administer it, that was a commendable decision if something had changed. In that case it would not have been wrong for sister Mbada not to follow the doctor's orders and not administer it when it would have been inappropriate to administer it. At 16:00 when the second stage started there was no caput and no moulding so the pelvic assessment was adequate and therefore there was no evidence of CPD. Up until 16:00 he could not find fault with the actual management of the labour. What can be faulted was note keeping.

[116] In assessing poor progress of labour CPD must be excluded as a first step and foetal distress. If foetal distress or CPD are present, then caesarean section must be done. Any other intervention is not acceptable. The main determinants of CPD are caput and moulding. Prior to doing vacuum delivery, pelvic assessment must be done and if the pelvis is contracted then a caesarean section might be a better

option. In this case both moulding and caput were excluded. Assessment no.4 at 16:00 in respect of the FHR was 150 which was indicative of a reactive CTG. 30 minutes thereafter there was an assessment by the sister and the patient had been bearing down for 30 minutes and the doctor was informed at 16:30. The delivery was completed by vacuum extraction at 17:10.

[117] The guidelines do provide that if the patient has been pushing for 30 minutes, the diagnosis of a prolonged second stage labour can be made. He testified that he and professor Anthony are in agreement that hypoxic stress on the baby gets progressively worse. So if this baby was under progressive hypoxic stress and the CTG was reactive at 16:00 then it would be highly unlikely that there was evidence of foetal distress prior to 16:00. In light of the fact that in labour hypoxia is a progressively increasing risk, the risk at 16:00 would have been more than the risk at 15:30. He had huge reservations in commenting on a ghost of CTG that he could not see as he said in the joint minutes. He was therefore not prepared to say that there was evidence of foetal distress or that there was no evidence of foetal distress as he was unable to read the faded CTGs.

[118] His evidence when he was cross-examined was that he could find no fault with the management of the labour based on the maternity case records. However, he would agree that the record keeping was faulty as there were some discrepancies. His view was that the fact that hypoxic stress on a baby gets worse until it is delivered and the recorded normal FHR shortly before delivery would indicate that there was no evidence of foetal distress and therefore no indication that intervention should have been sooner. He saw the CTGs that were put on the screen by Dr Murray and heard her evidence. He also heard the evidence of professor Anthony who used the pictures Dr Murray had photographed, of the same CTGs to draw a clear line of what the CTG's showed. He would not comment on those CTGs for the reason that they both hold the opinion they do based on what they could see. He was of the view that what you cannot see is much more important than what you can see.

[119] Where there are two tracings intermingling, one cannot see clearly where one comes down and where another one goes up. Therefore, he could not comment on

foetal distress. His view was that the tracings were unreadable. He testified that the tracings that professor Anthony drew were suggestive of a severely distressed foetus. The tracings he drew were in the early part of the second stage of labour almost an hour before the baby was born. If the baby was subjected to that degree of hypoxic stress for an hour the baby would have been severely compromised at birth. His field of expertise ends at the apgar scores and with the apgar scores for this baby he would not have been concerned as an obstetrician. His understanding of an assisted apgar score is if a baby is on a ventilator and the ventilator is breathing for the baby or the baby is on continuous bag mask ventilation at five minutes. But if the baby needed a little bit of oxygen and by five minutes the baby is breathing spontaneously on nothing more than a nasal prong oxygen that did not represent an assisted apgar score but he would defer to the neonatologist.

[120] With regard to his minute with professor Anthony, Dr Koll testified that they agreed that the active phase of labour should have been diagnosed at the clinic at 06:50. They agreed that the prescribed frequency of maternal and foetal observations were not recorded. With regard to the intrapartum foetal monitoring and the decelerations on the partogram, abnormal variability is over 25 bpm or less than 5, so normal variability is 5 to 25 bpm. Therefore, a range of 136 to 151 would indicate the normal range of variability. He testified that the evidence of the plaintiff was that she was on CTG monitor from the time of ruptured membranes. He agreed that there should have been re-evaluation of the tracing at 12:30 and there is no record that it was done. That is substandard record keeping. In terms of the guidelines there should have been re-evaluation of the condition of the foetus every half an hour even for a normal labour, never mind a patient who is on a CTG. He, however, said that if the court accepted that professor Anthony has given a correct depiction of that tracing then it was a very pathological tracing and immediate action was required.

[121] He added that the nurses continued with the labour. They had a running CTG tracing that they could see and they were experienced and were not concerned. He emphasised that only if the court accepted the hand drawn tracing of Prof Anthony, then that would represent a pathological tracing and therefore immediate action was required. He testified that his experience indicated that looking at a CTG in the

second stage of labour is extremely difficult. The only way is by actually putting the hand in the abdomen, feeling the contractions and listening to the foetal heart with a transducer. When one looks at the tracing for that time afterwards you cannot make head or tail of it. However, the tracing would be almost unreadable on a large number of occasions. Therefore, he disagreed with the proposition that a CTG is accurate in the second stage of labour and easier to interpret than listening and feeling.

[122] On the original CTG the tocograph was clearer than the cardio portion. The cardio portion was completely unreadable. The original tocograph was faint but he would not dispute its enhancement but disagreed very strongly with the enhancement of the cardio. As far as the contraction pattern is concerned, it could be uterine contraction or it could be anterior abdominal wall. It is unusual to get that number of contractions in that sort of pattern lasting for that short period of time in a tachysystole. A tachysystole is more commonly longer contractions. Such patterns are normally caused by patients pushing uncontrollably. The ideal is to try and coach the mothers and encourage them to push with contractions to create an expulsive force with all the available powers, the uterus, the abdominal wall muscles and if you are doing a forceps or vacuum delivery, all these forces must act together to get the expulsive force. However, sometimes mothers just lose it, they lose control in the second stage of labour and start pushing uncontrollably, unrelated to the contractions. He therefore felt that the picture in this case looked like a case of a mother pushing uncontrollably.

[123] He confirmed that the second stage of labour was delayed and when there is a delayed second stage action should be taken. He was of the view that there was no evidence of substandard care but there was undoubtedly substandard care in the record keeping of observations. He was of the view that at the time that syntocinon was prescribed, it was indicated. That time was at 15:00 when it was prescribed but there is no evidence that it was administered. If there was a change the nurse could decide not to administer it. It was put to him that there were strong contractions, in other words, contractions that were longer than 40 seconds in duration and there were two of those every 10 minutes. He confirmed that that started at 14:00 and it was recorded until 17:00. There was nothing in the partogram to indicate a change

that would allow the nurse not to follow the doctor's orders. He confirmed that on the records there was nothing that indicated any change. It was put to him that according to the guidelines the second stage is prolonged if the foetal head has not descended onto the pelvic floor after two hours of full dilatation or if delivery has not occurred after 30 minutes of pushing in a multipara woman. Using this criteria, the patient was fully dilated at 16:00 and therefore a prolonged second stage should have been diagnosed at 16:30. He agreed with this observation in his minute with professor Anthony.

[124] It was put to him that if one looks at the tocograph and accept that it is correct, what is depicted there is what the nurse would have seen. The nurse would not have been able to distinguish between deviations caused by contractions or by the mother pushing. Seeing the pattern of deviations, she should call the doctor immediately if the mother did not stop bearing down. He testified that the first step would be to try and talk the mother down and if that did not work and she was worried about foetal distress as excessive pushing is not a problem unless it is associated with foetal distress. Therefore, if she was happy with the foetus and indeed there is an indication that at 16:00 and at 16:30 there was no concern about the well-being of the foetus. Furthermore, the tracing that the nurses were seeing at 16:00 and 16:30 was not the tracing drawn by professor Anthony, it was a tracing that was just from the machine at that moment. They were seeing a tracing that himself, professor Anthony and Dr Murray were in agreement were capable of interpreting. Seeing that tracing they were not concerned about foetal distress. When it was put to Dr Koll that the nurses, seeing abnormal deviation pattern on the tocograph should have been concerned and that the partogram indicated that at 16:00 the FHR was 170. His response was that the CTG drawn by professor Anthony showed a single spike lasting just a few seconds up to 170 which would not constitute tachycardia.

[125] He went on to explain that on the baby being hypoxic, hypoxia is a normal event during the second stage of labour. The determinant was whether that hypoxia was severe enough to cause injury especially because hypoxic stress gets worse and worse. If the baby was born and the brain injury had occurred at the time of birth, then it would have been severally hypoxic at the time of birth and this baby was

clearly not. He went on to say that even with normal deliveries babies are often born with mild hypoxia that responds very rapidly to resuscitation. This happened even at an elective caesarean section where a small resuscitation is sometimes required. This would be because it is not just intrapartum hypoxia that is at play but also the transition of the baby from intra uterine to the extra uterine life which is a difficult transition for which often the babies need a little help. As an obstetrician if one accepted the tracing drawn by professor Anthony, it would be very suggestive of intrapartum hypoxia. As hypoxic stress gets progressively worse, the said tracing was done at least 40 minutes before the baby was actually born. Therefore, if hypoxic stress had continued for that period of time he would have expected the baby to be born in a severely hypoxic state and would have required significant resuscitation.

[126] Dr Janse van Rensburg who compiled a joint minute with professor van Toorn first testified about her initial report. She testified that she had consultation with the plaintiff who told her that in 2018 her third baby was delivered following a normal pregnancy that was for a full term of nine months. During the first pregnancy, when she went into labour she stopped feeling foetal movements and the same thing happened during her third pregnancy in the process of giving birth while in hospital. Her first baby had already died at birth and the same applied to the third baby. It transpired at some point that both the first and third babies who both died were males. With regard to A she testified that she was fully mobile without any help and she therefore classified her as being GMFCS 1 in her report. She and professor van Toorn also agreed with each other on this in their joint minute. They also agreed that she had a dyskinetic type of cerebral palsy functioning at level 1 with severe global developmental delay. She was asked about Volpe's criteria for neonatal encephalopathy that would implicate the intrapartum period.

[127] She testified that according to Volpe⁴ the neurological syndrome that accompanies serious peripartum hypoxic ischemic injury is the prototype for neonatal HIE. In considering the nature and timing of hypoxia-eschemia as the etiology of neonatal HIE, three features are considered to be important: 1. The

⁴ Volpe page 483.

evidence of foetal distress and of foetal risk for hypoxia-ischemia, for instance foetal heartrate abnormalities, sentinel event, foetal acidemia; 2 the need for resuscitation and or low apgar scores; 3, an overt neonatal neurological syndrome in the first hours or day of life. She testified that neonatal encephalopathy is divided according to mild, moderate or severe degrees. A had mild neonatal encephalopathy after her birth until the 4 January 2016. It is only when she suddenly deteriorated on 4 January 2016 that the question of a change to moderate neonatal encephalopathy came into discussion. She testified that if one takes these criteria and apply them to A, the people who did the delivery did not think there was foetal distress and/or foetal risk for hypoxia-ischemia. There was a need for resuscitation. Therefore, criteria number 2 was met and criteria number 3, she did have a mild neonatal encephalopathy but definitely there was no moderate to severe neonatal encephalopathy.

[128] When A deteriorated on 4 January 2016, her HIE score was still 6 out of 22. That means her score of 6 out of 22 would fall in the mild category. But if there were seizures that may have turned into the moderate neonatal encephalopathy. Contrary to what is usually seen in severe hypoxic-ischemic encephalopathy that leads to brain injury, A's level of consciousness was normal despite not feeding and then suddenly deteriorated on 04 January 2016. The evolution according to Volpe, of the overt, neurological syndrome, hypoxic ischemic encephalopathy is the first 12 hours, 12 to 24 hours, and then from between 24 and 72 hours and then beyond 72 hours. So contrary to what is usually seen, this case is not a typical case of neonatal encephalopathy.

[129] Professor Andronikou who compiled the MRI report described the injury that he had seen on the MRI as one that could possibly fit in with hypoxic-ischemic brain injury but that other causes such as metabolic, infective and toxic causes would need to be excluded. Metabolic causes also include genetic causes. He did not mention supper added hypoglycaemia. She testified that as neurorologists, they start with the history. In the case of A, there appears to have been no concerns during the pregnancy. The factual witnesses did not detect foetal distress. She was born flat and needed resuscitation but her level of consciousness improved immediately. She did have a problem with feeding and then there is a debate about how much of it

could be attributed to her and how much to her mother. At most she would have scored a mild neonatal encephalopathy. Although children with HIE can improve and then deteriorate, she did not show that progressive deterioration over the next hours and days until the 4 January 2016 when she started having seizures. At that point her HIE score was done and it was 6 but she was clearly ill. She had difficulty to control epileptic seizures which had to be treated with two drugs. After that her development has been slow. Her biggest problem when she saw her was that she had cognitive impairment and not her motor function which was level 1.

[130] A had atypical history in the neonatal, perinatal period and peripartum period. This is a very unusual neonatal encephalopathy. Her situation taken as a whole does not sound like the usual post hypoxic-ischemic cerebral palsy that is normally seen. The features in the MRI scan are atypical and the reporting radiologists mentioned that other conditions would need to be excluded. Professor van Toorn and herself then agreed that metabolic and genetic testing needed to be done. Indeed, shallow testing was done but further testing was unfortunately prohibited. When one looks at the family history as a whole, there are four children and the mother said she had the same partner. He was not open to being examined. There were two boys who then died and the history indicates that they may have died during the labour process. A has a neurological condition. Then the youngest daughter had a normal birth or at least her apgar scores were normal. Out of the blue she had a seizure which was investigated and no cause was found. She did not have any other signs of neonatal encephalopathy. Not only was the examination of the fourth child prohibited but also a request to do further genetic testing on the blood of A that had already been sent overseas was prohibited and that blood was ordered to be destroyed.

[131] With all these uncertainties it is not known what further examination and chromosome studies would have revealed but it looks like there is a genetic disorder in this family. It looks like the two male children were exceptionally vulnerable and they died during the process of being born. Then there is A with her condition and then there is the fourth child. Both girls did not have the same clinical picture but A had a neonatal encephalopathy level 1 from her birth on 31 December 2015 until the 4 January 2016. The fourth baby was normal until two days after delivery. Both of

them developed epilepsy. There is therefore a need to exclude epileptic encephalopathy and X-linked genetic disorders and there is a need to exclude genetic conditions that would interfere with delivery of energy. It appears that during the normal asphyxia process of birth, the children who were normally grown up with no evidence of intrauterine growth restriction, but the moment they suffered asphyxia and the stress of the asphyxia, they just decompensate it and the males died. They were stillborn and A was born in a flat state. There was therefore a need to look at the genes that would generate energy and the genes that code for membrane stability which would be the genes that are affected in the epileptic encephalopathies.

[132] During cross-examination Dr van Rensburg was referred to an article by Bhorat⁵ in which the writers say that clinical features that should prompt evaluation for genetic metabolic conditions in a patient presenting with symptoms of cerebral palsy are an absent history of any perinatal risk factors for brain injury. It was then put to her that genetic testing or metabolic testing should be considered in cases where you do not have a history for brain injury and that abnormal or a pathological FHR would be a perinatal risk factor for brain injury. Her evidence was that one cannot take the absence of one single factor and conclude that there should be no genetic testing. It cannot be said that because there is an abnormal CTG there should be no genetic testing. She was asked if the court were to accept the evidence of Dr Murray and professor Anthony regarding the features of the CTG, her opinion would change and she would therefore accept that A had an intrapartum hypoxic ischemic injury. She testified that as a neurologist she still had difficulties with the doctors being there and not seeing any evidence of foetal distress. Furthermore, there was the retrospective finding based on a very poor CTG in circumstances in which the CTG may be affected by other things that are not distinguishable from how it would read in respect of foetal distress.

[133] It was put to Dr van Rensburg that seizure activity might have taken place but missed because for the first three days in hospital there was skeleton staff with relatively fewer observations of the child and the child was lodging with its mother

⁵ Bhorat I et al: Cerebral palsy and criteria implicating intrapartum hypoxia in neonatal encephalopathy – an obstetric perspective for the South African setting: (2021) 111 SA MJ 3 pages 280-288.

and not at nurse. Her evidence in that regard was that while it was possible that some clinical seizures could have been missed, most of the seizures occur within 6 to 12 hours of delivery if it was hypoxic ischemic in nature. During that period Dr Mans was still on duty where he popped in again to see the mother and the child. If there were any severe or prolonged seizures, there would have been a change in the level of consciousness of the baby and there was no evidence of that. It was further put to Dr van Ransburg that clinical features that should prompt the evaluation for genetic and metabolic conditions were not present in A. She disagreed with that postulation saying that clinical means history and examination. Any parent who has lost two children before birth should in any case be genetically investigated. Then there is a child with a disability and another one who had seizures in the neonatal period. A family history is clinical evidence which is prominent in this case. With regard to the absence of the history of any perinatal risk factors for brain injury which would be absence of a risk factor like intrapartum asphyxia, as the question was put to her, she testified that it cannot be said that there were no perinatal risk factors in light of the family history. The terrible obstetric history was a huge perinatal risk factor which professor van Toorn agreed should be regarded as a distal factor.

[134] The defendant's next witness was professor Rothberg who together with professor Smith did a joint minute as neonatologists. He testified that the clinical information in this case was that there were four pregnancies. Two males who died and two females who had seizures and one progressed to cerebral palsy. He explained that a phenotype is what a person, the individual looks like and the genotype is what the genetic pattern would look like when one investigates it. In this case there is a picture of babies that appear to have grown well during pregnancy and then have different outcomes with the boys dying, either immediately postnatally or before birth. The females survive with variable expression. He said that this is a genetic picture that needed to be further investigated. He referred to ACOG, the American College of Obstetric Gynaecology and the AAP, the American Academy of Paediatrics task group on the study of encephalopathy and cerebral palsy⁶. Therein the writers say:

⁶ The American Academy of Pediatrics: Neonatal Ancephalopathy and Neurological Outcome, Second Edition: Pediatrics Volume 133, Number 5, May 2014: <http://pediatrics.aappublications.org/.page> e 1483.

“To determine the likelihood that an acute hypoxic-ischemia event that occurred within close temporal proximity to labour and delivery contributed to a neonatal encephalopathy, it is recommended that a comprehensive multidimensional assessment be performed of neonatal status and all potential contributing factors, including maternal medical history, obstetric antecedents, intrapartum factors (including heart rate monitoring results and issues relating to delivery) and placental pathology.”

[135] In this case a multidimensional assessment would obviously include the family history and the obstetric antecedents. He testified that essentially the plaintiff's case was that the proximal risk factors led to the neonatal encephalopathy and cerebral palsy. He said that there are proximal risk factors and distal risk factors. The proximal risk factor which is close to the intrapartum period is said to be the sole problem in this case which caused the neonatal encephalopathy. This was said to be, according to professor Smith, an obstetric problem which is pathway A. Pathway B talks about distal risk factors. Distal means far away from the time of delivery whereas proximal risk factor would be closer to the time of delivery. A family history would be a distal risk factor. An antepartum distal risk factor would be something like if the mother develops hypertension during pregnancy, diabetes or HIV. The question in this case is whether there was a proximal risk factor, the intrapartum asphyxia. He explained that the delivery itself is a high risk situation.

[136] He testified that in his joint minute with professor Smith, the latter was arguing pathway A whereas he argued pathway B. He was making a case for genetic investigation. He referred to professor Andronikou's report. He said the genetic investigation was not sufficiently done. He said professor Andronikou had opened the door and was in fact recommending genetic investigation where he says “the patient requires evaluation by a paediatric neurologist and may have to undergo testing for metabolic disorders.” He then referred to what professor Smith says in his joint minute:

“Neonatal hypoglycaemia may injure the thalamic pulvinar. The pulvinar is injured in the present matter. The thalamic pulvinar has been highlighted as a

distinguishing factor when determining whether the HIE injury was compounded by neonatal brain injury related to hypoglycaemia.”

[137] Professor Rothberg further testified that there are typical occipital changes as well. In his opinion with regard to professor Andronikou’s report, the changes were not obvious and had the changes of hypoglycaemia been obvious, then professor Andronikou would not have just included it in the list of metabolic disorders but would have been specific about the hypoglycaemic changes being present.

[138] Professor Rothberg continued with his evidence explaining his argument for genetic testing. He referred to the plaintiff’s literature bundle in an article written by Cowen et al 4 published in 2003 which he said is frequently referenced in the discussion of whether an injury was intrapartum or antepartum. He said at page 261 the authors say:

“Our data do not exclude the possibility that antenatal factors could initiate a causal pathway for perinatal brain injury and that they, possibly together with genetic predispositions to hypoxic-ischemic injury, might make some infants more susceptible than others to the stressors of labour and delivery.”

[139] He testified that in his joint minute with professor Smith they are in agreement regarding the following. The baby developed and presented with mild hypoxic ischemic encephalopathy between birth and around 16:29 on 3 January 2016. They agreed that at 16:29 was the first time that the bilirubin results indicated an abnormality. Dr Mans did the bilirubin at that time.

[140] They agreed that the foetus suffered intrapartum hypoxic ischemia. However, professor Smith goes for pathway A and it means that it is the result of the insult during the management of labour. On the other hand, he testified that he could not exclude additional asphyxia as a result of obstetric management but he agreed that there was intrapartum hypoxia ischemia. They also agreed that the neurological condition (encephalopathy) worsened around 4 January 2016. Suddenly there was fitting and that changed the condition at 16:00 on the 4 January 2016 which was some 95 hours after birth. He explained that professor Andronikou’s report was

equivocal with “may” being highlighted. They also agreed on cerebral palsy based on the joint minute of the paediatric neurologists as they both did not see the child.

[141] He testified that 24 hours after birth there is a note that the baby was sucking well. Under those circumstances admission to a high care unit or ICU for intensive care monitoring would have been inappropriate and there were no features of a progressive HIE. The clinical progression between days one and five was not compatible with the progressive HIE. Consequently, other causes for the encephalopathy as manifested by the seizures must be considered. Professor Smith is of the opinion that substandard intrapartum obstetric care directly contributed to the child’s neurological disability and that timeous expedited delivery would have avoided the outcome. Furthermore, had proper neonatal care been afforded to the baby, the possibility of aggravating factors occurring and contributing to the outcome such as hypoglycaemia would have been avoided altogether.

[142] Professor Rothberg testified that in the absence of the family history in this case there would likely have been little to argue other than the presence or severity of foetal distress. However, the family history cannot be ignored. Significant factors include four pregnancies. Two males died late in pregnancy or shortly after birth. A female presented with HIE and subsequently developed cerebral palsy and global developmental delay. A second female is reported to have had seizures on the first or second day of life. This pattern of males being affected while females are less so affected, fits with recognised modes of inheritance, X-linked or mitochondrial disorder. The onset of seizures was late for a progressive HIE, therefore other causes for the late neonatal encephalopathy should be sought. He concluded his main evidence by saying that the distal risk factor or the genetic factor plus a proximal risk factor of asphyxia would have led to neonatal encephalopathy. The final outcome was cerebral palsy related to a combination of factors. It is not clear that expedited delivery would have altered the outcome.

[143] Professor Rothberg then testified under cross-examination. He said that indeed it was his evidence that the foetus suffered intrapartum hypoxia-ischemia. However, he was of the view that the intrapartum hypoxia-ischemia may have underlying mechanisms. In pathway A it is postulated that the intrapartum asphyxia

was largely the result of what happened during the intrapartum phase which would be an obstetric issue. On the other hand in pathway B which is what he postulates, the intrapartum asphyxia was also related or alternatively related to the normal asphyxia process as has been described very well by professor Anthony that every baby suffers asphyxia. In a situation of a primed foetus, that intrapartum asphyxia, that normal asphyxia may become pathological. Therefore, he was only in agreement that there was intrapartum asphyxia. While he accepted that the normal asphyxia does not result in HIE, A was not a normal baby, it was a primed baby in which the labour may trigger the pathology. He was aware that the neonatologists are in agreement that at birth there was neonatal encephalopathy which was the result of intrapartum hypoxia-ischemia which was assessed as mild HIE and he agreed with that. According to Volpe the sequence of events is that following the hypoxic-ischemic insult and a brief period of apparent improvement between 24 and 72 hours of age, the level of consciousness deteriorates in neonates whose HIE is progressing. After 72 hours, the stupor continues and abnormal sucking, swallowing and tongue movements prevent feeding. In A's case the problem was only poor latching.

[144] There was no deterioration in the level of consciousness between that time. Both the notes and the evidence refer to poor latching rather than poor sucking. In this case those who were observing the baby did not observe progressive signs of encephalopathy until 80 hours and more. Professor Smith agreed that in 24 to 72 hours there is progression in most cases. The evidence is that Dr Cilliers examined the baby at 18:30. At about 22:00 which was about five to six hours after birth, Dr Mans examined the baby and that was a systematic examination in which he looked specifically at the neurological status of the baby and he found the baby to be normal. After that the notes refer to poor latching. Dr Cilliers indicated positively on breastfeeding and said an assessment was necessary to assist with the feeding and then the note says sucking well. On the issue of a long weekend and understaffing that was mentioned, the evidence of Dr Mans was that there was a daily visit to ensure that the babies that could go home were able to go home with their mothers. Professor Rothberg testified that he agreed that there was no daily systematic investigation. However, he was of the opinion that a baby with a progressive encephalopathy and becoming more and more stuporous would be obvious to the

nursing staff who are constantly cup feeding the baby. Nurses were cup feeding the baby and the baby was able to feed. All of that is not a picture of a progressive encephalopathy with a progressive stupor.

[145] He accepted that subtle seizures may be missed. However, seizures occur in concert with other signs such as progressive deterioration in level of consciousness. It is impossible that if A was undergoing progressive encephalopathy with interference with the level of consciousness, level of conscious tone etc, the nurses would not be commenting about that. The comments of the speech therapist were about 94 hours of age which was well after 72 hours on 4 January 2016 at 15:30. The baby was not comatose or stuporous before 72 hours. Something happened which was why Dr Mans asked, "why now?". It is apparent that the child had been normal up until well into day 4 and suddenly there was a problem. He was of the view that the cause of the late onset of seizures might be bilirubin toxicity. The child was not manifesting signs of progressive HIE until the point when she was assessed by the speech therapist who made the observations she did. With regard to professor Andronikou raising the red flag of possible metabolic genetic conditions, he testified that conditions such as canavan disease, krabbe disease and wilsons disease are genetic conditions and those were examples of genetic conditions that he referred to. In light of the family history, the child required an evaluation. What professor Andonikou said was that the picture is not specific for hypoxic-ischemic injury and therefore there should also be a genetic evaluation or investigation.

[146] The last witness called by the defendant was professor Christianson who had been called as a subspecialist in genetics. He testified that he prepared a joint minute with Dr Gericke. At some point in their interactions as geneticists engaged in the matter they became aware of the fourth child who also had neonatal seizures. Dr Gericke suggested that a Whole Exome Sequencing (WES) be done in those circumstances with specific emphasis on cerebral palsy and epileptic encephalopathy.

[147] Based on the documentation that was available and A's clinical features he stated that he did not think that the first four days of A's life were consistent with neonatal encephalopathy 2 or 3 or overt neonatal neurological syndrome. He

therefore suggested that a neonatologist should be consulted. Then he became aware of the third pregnancy outcome and then the fourth pregnancy. The fourth child had been delivered apparently well until she fitted on the second day of life. He then recommended that the fourth pregnancy needed to be assessed by an obstetrician. Furthermore, the baby as well as other members of the family needed to be medically evaluated by a paediatric neurologist and a medical geneticist. He was of the view that if indicated, the fourth child should also have an MRI. If it became necessary, depending on what the MRI showed, a medical genetic testing would then be considered.

[148] He then testified about a report from Centogene in respect of A. He described the test that had been done as a deep analysis of a narrow field of metabolic disorders. It is the same type of testing that would be done in WES and WGS but it is confined to a specific number of disorders. On this test there were no metabolic disorders that were diagnosed. There may be other rare conditions that were not tested for but on the Centogene panel for metabolic disorders the results were negative. Centogene recommended WES as the next step. While he was considering and working on a WGS being done by Centogene, a letter was received from plaintiff's attorneys prohibiting any further genetic testing and prohibiting even the use of the blood sample that was already with Centogene⁷.

[149] He testified that when the said letter was received Centogene had some blood of A that was left and therefore there was no need for a further drawing of a blood sample. However, it may have become necessary to draw blood from the mother and father but in respect of A the further testing could have been done with the blood sample Centogene already had. He testified that metabolic disorder genetic testing had been done. He explained that basically genetic testing is a process in which one can test for a particular gene. The next test is for a panel of genes which cover a

⁷ The said letter reads "To the extent that your client and/or Centogene may be in possession of a portion of the blood sample, under no circumstances whatsoever is your client permitted to perform or undertake any further whole exome sequencing, genome sequencing or any other genetic tests of any kind or nature. In particular there is a cluster of our client's constitutional rights that would be fundamentally breached, viz, those of dignity, privacy, freedom of security, bodily integrity etcetera. In addition to the constitutional rights that may be infringed your client has already exhausted its remedy for medical examination in terms of Uniform Rule 36 (5). At common law it is well established that a person's right to bodily integrity and autonomy entitles him to refuse medical treatment or assessment and subjecting a person to unauthorized medical procedures to which he or she has not consented has been held to constitute invasion of privacy."

particular group of problems. There is then WES which covers up to 80% of genes that code for proteins needed to run the body. Finally, you have the WGS which covers everything covered by WES plus many genes known in the genome. There are many problem causing genes but the WGS will look at the genes and if an abnormality is found it will be pointed out. He added that increasingly more problem causing genes are being found and if one of them is found, it has to be related to the phenotype of the particular individual to see if it is relevant.

[150] He testified that his interpretation of professor Andronikou's MRI report on A was that there were four possible causes for A's condition. It was hypoxic ischemic injury of a combined acute profound and partial-prolonged, toxic causes and metabolic causes. Those would include canavan disease, krabbe disease and wilson disease as well as post infectious causes. He explained that professor Andronikou did not have a clinical picture. Therefore, in light of A's clinical phenotype and the significant family history including the 3rd and 4th children, a genetic etiology other than a chromosomal imbalance which was largely excluded by the Cento LCV or a metabolic disorder largely excluded by Cento metabolic testing must be added to the list of possible causes. He disagreed with Dr Gericke who said that A had no genetic predisposition.

[151] Under cross-examination professor Christianson testified that both professor van Toorn and Dr van Rensburg confirmed that A had mild dystonic cerebral palsy and he accepted that. He also accepted professor Rothberg's opinion that she had HIE grade 1. On day four after the seizures, the child developed neonatal encephalopathy grade 2 with seizures which was not typical of neonatal neurological syndrome as described by Volpe. He explained that there was a difference between what professor Rothberg and professor Smith said when they suggested that the primary etiology of A's injury was hypoxic ischemic in origin. His understanding was that professor Andronikou said the features on A's brain may be due to hypoxic ischemic injury of a combined acute-profound and partial prolonged nature meaning it was possible. However, the pattern of injury in A can also be seen in toxic, metabolic and post infectious causes. This means that it may not be hypoxic ischemic of acute profound and partial prolonged. It may be due to a toxic cause, or a metabolic cause or post infectious cause. He therefore did not consider professor

Andronikou's report to mean that hypoxic ischemic injury was the primary cause. After becoming aware of the third and fourth child he decided to add into the list of possible causes, the genetic causes.

[152] Professor Christianson was referred to a report of the defendant's radiologist, Dr Schwartzberg in which he inter alia opined that the features in the MRI scan in A are consistent with hypoxic ischemic encephalopathy due to perinatal ischemia. Professor van Toorn also compiled a report in which he agreed with professor Andronikou that the abnormalities seen in the MRI scan were those of chronic evolution of a global insult due to hypoxic ischemic injury of a mixed acute-profound and prolonged partial variety occurring in a brain of term maturity. In his evidence professor van Toorn also pointed out that the two radiologists, professor Andronikou for the plaintiff and Dr Schwartzberg for the defendant concurred that the pattern of injury is that of hypoxic ischemia. While he agreed that Dr Schwartzberg made the findings that he did and professor van Toorn made the statements attributed to him, he pointed out that professor Andronikou made it clear that the injury pattern "may" and bolded the word "may" which was critical in reading his report. But he accepted that Dr Schwartzberg's opinion was that it was hypoxic ischemic injury of a partial prolonged and acute profound type. Therefore, there were significant differences between the statements of the two radiologists. Professor Andronikou and Dr Schwartzberg differed and there was no joint minute between them and both of them were not called to testify.

[153] It was put to him that toxic and post infectious causes that professor Andronikou also referred to have been excluded and he agreed that they have been excluded. That leaves hypoxic ischemia and metabolic causes and he added a third which is genetic disorders or congenital disorders. Metabolic testing largely excluded metabolic causes. He explained that as a geneticist and in light of the additional information and the family history and having known that A had congenital macrocephally and the fourth child who also had some issues, he was of the view that genetic testing needed to be explored. He agreed that save for hypoxic ischemic injury all the possible causes that professor Andronikou mentioned have been excluded but he considered genetic testing to be necessary. He disagreed that evidence of foetal distress and a difficult labour would militate against doing further

genetic testing. He said he accepted that A has cerebral palsy and that both Dr van Rensburg and professor van Toorn say she has mild dystonic dyskinetic cerebral palsy. It was put to him that the neonatologists, professor Smith and professor Rothberg have agreed in their joint minute that the foetus suffered intrapartum ischemia and they are the correct specialists to make the diagnosis.

[154] He testified that he had decided that the Cento-LCV test be done and professor Andronikou had decided that the Cento metabolic test be done so that metabolic causes could be excluded. The Cento-LCV test was done to deal with his clinical diagnosis of macrocephaly. However, the Cento-LCV test did not detect macrocephally. He was looking at the macrocephally as part of the phenotype but when the discovery of the 3rd and 4th children was made he had to evaluate the situation because it revealed a significant family history. There were neonatal seizures in both A and the fourth child and all of this required further testing like WES or WGS. That is how he got to write to Centogene asking them for their suggestions. The LCV test which did not show congenital macrocephaly was the baseline test. There are many other tests that could be done including WES and WGS.

[155] Professor Christianson was asked with reference to Dr van Rensburg who stated in her report that A had dyskinetic cerebral palsy as well as a moderate degree of neonatal encephalopathy. He said that on his part he did not feel that the child had cerebral palsy but deferred to a paediatric neurologist. He deferred to an obstetrician. The issue of the fourth child became a relevant factor when on day two of her life, she developed seizures with no obvious reason when she had apparently been normal, having been born with normal apgar scores and had been breastfeeding. There were no questions of hypoxic ischemic encephalopathy in the first hours or day of life. According to Volpe in the first hours or day of life there was no overt neonatal neurological syndrome.

Negligence.

[156] Some of the facts about what happened on 31 December 2015 are largely common cause or cannot be disputed, at least not cogently. The plaintiff was at a local clinic at 06:50 having started feeling labour pains at about 02:00 am that

morning. The clinic decided to send her to hospital for the management of the labour after a vaginal examination had been done and the cervical dilation had been assessed. She was found to be 5cm dilated at the clinic. She arrived at Zithulele Hospital at 09h00 at which time the first entry in the partogram was made. Her cervical dilation was assessed and it was found that she was still 5cm dilated. There appeared to be no concern about the foetal condition. Perhaps it is important to note that at this time there had been no progress in her dilation as she had been assessed as being 5cm dilated at 06:50 at the clinic. Therefore, by 09h00 it had been two hours with no progress in dilation. At 12:00 another assessment was done. At this time the heart rate was 160 bpm which according to Dr Murray, was borderline high as the normal heart FHR is between 110 and 160 bpm. In Dr Murray's opinion regard, being had to the plaintiff's first child who was born at home as a still born, this slow labour progress was concerning. She testified that according to the partogram at 14:00 there was no cognisance taken of the slow labour progress and consequently no plans were made to remedy it. There was no documented heart rate for two hours from the last plotting at 12:00 which had shown abnormality. There should have been plotting at least every 30 minutes and this did not happen.

[157] It was also common cause that there was poor recording of the monitoring which therefore meant that the observations that were done were not always recorded to see the heart rate, accelerations, decelerations and variability at different times. What would have been observed from the CTG should have been recorded in the notes and the partogram. According to Dr Murray, the partogram showed that no monitoring was done for four hours from 12:00 to 16:00. When the plotting was done on the partogram the FHR baseline was 170 bpm at 16:00 and it dropped to 140. This means that the FHR baseline was higher and therefore there was tachycardia. The FHR was too high and then there was slowing after contraction, a pattern similar to 12:00 where it had been at 160 bpm. Dr Murray was of the opinion that on the reading of the partogram, it appears that foetal distress was on going between 12:00 and 16:00 as shown by the baseline FHR having gone up from 160 to 170. The recording of the FHR of 150 to 155 in the notes does not agree with what appears in the partogram.

[158] According to Dr Murray the report of professor Andronikou refers to acute profound and partial prolonged brain injuries. This means that he found evidence of brain injury that occurred suddenly and evidence of brain injury that would have occurred over a long period of time. The evidence of brain injury over many hours was in keeping with what appears in the notes where the first recording of foetal distress was based on decelerations on the partogram was at 12:00 but the baby was only delivered at about 17:00. The five hour period was enough period for the baby to sustain brain injury as the baby would have been struggling to maintain normal oxygen levels for five hours while in distress. That made the foetus more vulnerable to injury so that even the normal birth process might have been the last straw which the baby could not cope with.

[159] Based on the maternity case records, Dr Murray summarised her evidence on the active phase of labour. I must again point out that most of this evidence is common cause, having been gleaned from the maternity case records. At 12:00 the partogram suggested decelerations but assessment no.2 does not and the recorded FHRs are different. At 14:00, according to the partogram, the cervix was 5cm dilated with no recorded FHR. However, according to assessment no.3, the cervix was 7cm dilated which showed a discrepancy regarding the cervix. At 15:00 the cervix was 7cm dilated but there was no recording of the heart rate. At 15:00 an order was given for syntocinon infusion but there is no record of a reactive CTG at 15:00. At 16:00 the cervix was fully dilated according to the partogram with two strong contractions in 10 minutes. The FHR was 170 bpm before contraction and 140 after a contraction with late decelerations. However, according to assessment no.4 at 16:00, the cervix was 10cm dilated with strong contractions with a FHR of 150-155 bpm with a reactive CTG. Therefore, what was on the partogram and the assessment notes were completely different.

[160] She also testified about the faded CTG tracings which she had taken pictures of with her cellphone and enhanced them to improve their legibility. The admissibility of such evidence to which the defendant objected very strongly with the defendant's expert refusing to have regard to the faded CTGs saying he could not read them is being questioned and objected to. I will deal with this issue pointedly later in this judgment. Dr Murray further testified that if the partogram had been plotted correctly,

the labour progress would have crossed the action line at 13:00 indicating a need for a doctor to make an assessment and decide on the required intervention. This could have been allowing more time, rupturing the membranes which, in any event, had ruptured spontaneously at 12:50, giving oxytocin or performing a caesarean section.

[161] Her opinion was that there were several significant labour related risk factors for hypoxic brain injury. These were the prolonged labour, the use of oxytocin, substandard foetal monitoring especially during oxytocin administration and the complicated second stage of labour which necessitated instrumental delivery. With regard to whether or not the oxytocin was administered, she was of the firm opinion that it was administered. She based her opinion in this regard on the contraction pattern which showed hyperstimulation. There should also have been a nursing note explaining why it was not given as the doctor had ordered it, if it had not been given. If the pre-requisites for it were met, which were a reactive CTG and a catheter, logic dictates that it would have been given as the doctor had ordered it. If it was not given it could only be because the CTG was not reactive meaning the FHR was not normal.

[162] She testified that at 12:00 there was an indication for a caesarean section as there was a delayed labour progress. At 14:00 the labour progress continued to be poor and therefore due consideration should have been given to performing a caesarean section. In this case the cervix was dilated at 5cm at 06:50 and was 7cm at 12:00. Therefore, the plaintiff had progressed only 2cm in five hours. By definition labour progress was poor. There was substandard care in her opinion in the failure to timeously diagnose failure to progress, the failure to plot the partogram accurately which led to the failure to diagnose the poor labour progress, the failure to react to probable foetal distress from as early as 12:00, the failure to react to foetal distress at about 16:10, and the probability that oxytocin was given in an unmonitored manner. That would have led to severe uterine tachysystole which was also unrecognised with the foetal distress that came with it. All of this happened to a patient who was a high risk as she had already lost another baby. The contraction pattern of 10 to 11 contractions in 10 minutes suggested the use of a uterine stimulant and tachysystole was in keeping with the use of syntocinon. It was

contended on behalf of the plaintiff that the essence of Dr Murray's evidence was not disputed by any of the defendant's witnesses.

[163] Professor Anthony testified that the hospital notes started at 09:00 and that entry was incorrectly plotted on the latent phase part of the partogram graph. A patient who is not progressing in labour needs to be carefully assessed and the reason for the slow progress needs to be found. In a parous woman the diagnosis of the slow progress should lead to the critical assessment of the labour to look for signs of pelvic disproportion. In such circumstances oxytocin should only be prescribed with great caution after such examination. The FHR on the partogram was recorded only at 12:00 and at 16:00. The expectation is that during the active labour phase, the observation of the FHR should be done before and after contraction every half an hour. Therefore, the number of observations were clearly not according to the prescripts even for patients who are completely normal. The foetal well-being was inadequately assessed. At 12:00 it was the first time in which the partogram was correctly plotted before and after contractions as it is shown by the different heart rates before and after contractions. From 12:00 onwards the FHRs were slower after each contraction compared to the baseline rates. The difference in the observed FHRs is significant and should have led to the suspicion of foetal hypoxia. This should have led to the introduction of continuous CTG monitoring with re-evaluation of the tracing every 30 minutes.

[164] There does not appear to have been a consideration of foetal hypoxia or foetal distress as a possibility. At no stage did anybody take cognisance of the fact that the baby might be hypoxic which was substandard care. At 12:00 the disparity between the pre and post contraction heart rate was observed but the significance thereof was not taken into cognisance by the attending staff. The plaintiff reached full dilation at 16:00 with the nursing staff still recording a large disparity between pre and post contraction rates. The CTG tracing at this time was pathological even though it is not known for how long that tracing was pathological. What is known and discernible is that that tracing was abnormal from the beginning. The tracing he received was faint but he examined it with a good light and a magnifying glass and used a felt pen to highlight the tracings. He was able to make out a discernible tracing. He enhanced it by tracing what was available in order to determine the

pattern of the abnormality. The tracing begins at 16:00 and for the first two minutes it shows a baseline of about 145 bpm followed by a FHR tracing that decelerates progressively down to sometimes as low as 55 bpm. When it reverses the baseline heart rate is 145 bpm. Once it gets back up to the baseline there is a brief period of tachycardia which means a heart beating very, very faint going up to about 170 bpm.

[165] This is followed by a decline with further decelerations being evident for the rest of the tracing. There were repetitive decelerations until the tracing ended at about 16:25 and the baby was delivered at 17:10. He could see nine contractions which is tachysystole. With such frequent contractions there was not enough relaxation time between them for the baby to get more oxygen to maintain normal metabolism. That tachysystole is a recognised complication of an oxytocin infusion. Its evidence in the tracing starts from 16:00 to the end of the tracing. If the tracing falls into a pathological category there is a high probability that the baby is hypoxic and acidotic. When there is foetal distress especially in the second stage of labour oxytocin must be stopped. The mother must stop pushing and the contractions must be stopped and intrauterine foetal resuscitation must be done. Tocolytic drugs should have been used to relax the foetus. Both the contractions that were being augmented by the use of oxytocin infusion and the maternal bearing down effort should have been stopped to allow restoration of foetal oxygenation through intrauterine resuscitation prior to the expedited delivery. According to the available hospital records the pathological tracing was not recognised and the necessary interventions did not take place. That was substandard care which was directly linked to an increased likelihood of an adverse outcome. More than 5 contractions in 10 minutes constitute tachysystole. The decision to allow the mother to push in the face of a pathological tracing was substandard care which would have increased the likelihood of foetal hypoxia.

[166] The patient remained at 7cm of dilation for three hours up to 15:00. At 12:00 disproportion and foetal distress should have been considered and critically evaluated. Not doing it was substandard care. By 14:00 the progress of labour was non-existent for the preceding two hours and there is no recording of the FHR charted on the partogram despite a previous abnormal finding. There should have been an intervention by means of caesarean delivery as early as midday as there

was an indication for it. However, the baby was delivered after 17:00, some five hours later. The indication for caesarean delivery at 14:00 was shown by the ongoing failure to show adequate progress of labour. The same indication existed at 15:00. The failure to correctly complete the partogram led to the late recognition of slow labour progress.

[167] In the partogram only two contractions per 10 minutes were recorded throughout the entire course of the labour. On the available CTG tracings there were quite clearly numerous contractions every 10 minutes and not two per 10 minutes as depicted in the partogram. The problem of the FHR from 12:00 went unrecognised by the nurses. The decision to use oxytocin should have followed a very careful assessment of the labour which did not happen. Foetal distress was not excluded before the introduction of oxytocin. It was prescribed for a parous patient. The foetal distress went unrecognised and no intervention took place. On the contrary, the mother was encouraged to push and she was given oxytocin. Encouraging her to push and the use of oxytocin were contra-indicated because of the pathological tracing. All of that was substandard care.

[168] There was a period of 5^{1/2} hours during which there was evidence that the baby was trying to compensate by slowing her FHR after contractions because it was becoming hypoxic. It was likely that there was a gradual worsening hypoximia. In addition to that, oxytocin was introduced and the mother was encouraged to bear down in the second stage of labour which more than likely led to the sudden intensification of the hypoxic stress as evidenced in the pathological tracing. The neuroradiological diagnosis provided by professor Andronikou was that of acute profound hypoxic injury and partial prolonged injury to the foetal brain. That is consistent with what appears to have happened during the course of this labour. The apgar score of 7 at 5 minutes did not necessarily exclude the possibility of acidosis. As an obstetrician, his view was that there was evidence of foetal distress. The baby needed some support at the time of delivery as the neurological syndrome took place. The prerequisites for a diagnosis of neonatal encephalopathy due to intrapartum asphyxia are present in this case with no obvious explanation.

[169] His conclusions were that this was an uncomplicated pregnancy. The mother went into spontaneous labour at term. The problem arose with the management of the labour which was characterised by substandard care in a number of respects. The FHR monitoring was infrequent and incorrectly interpreted or at times not interpreted at all during the first stage of labour. There was inadequate monitoring of the active phase of labour which included the inadequate use of the partogram. Oxytocin was used without a proper prior assessment to exclude disproportion and without excluding the possibility of foetal distress. The second stage of labour was not adequately managed. It was allowed to continue despite the fact that the tracing was abnormal and the mother was allowed to push. The oxytocin was allowed to continue. His final conclusion was that *“the adverse outcome was consistent with intrapartum hypoxia which would have been avoidable with a proper standard of intrapartum care leading to delivery at several points where intervention might have been indicated well before the actual delivery at 17:10.”*

[170] Sister Mbada’s evidence on negligence was that she attended to the plaintiff’s labour. She testified that the order for syntocinon meant that it should be given 30 minutes after a reactive CTG. If it is administered a drip is inserted with a catheter also applied. The syntocinon is added into the drip. The prescription from the doctor for syntocinon was made at 15:00. At that time the plaintiff was still 7cm dilated. The instruction was that the CTG must be reactive before the syntocinon was infused and the catheter was applied. When infusion is started a tick is made every time it is infused. She confirmed that if there are changes in the intensity and frequency of the contractions syntocinon is not started. If infusions continue a tick is made on the prescription form. If it is discontinued she would write “infusion stopped.” Because there were no ticks it meant that syntocinon infusion was never started at all. She did not have a recollection of what happened on that day and for her evidence she relied on the maternity case records.

[171] She took over monitoring the plaintiff at 12:00 and at the time she did not realize that the partogram was started incorrectly. She confirmed that the completion of the partogram is essential in the management of the labour. She confirmed that she made a mistake on the partogram at 14:00 when she recorded 5cm dilation whereas in the clinical notes she recorded 7cm. She accepted that it

was a mistake on a very important aspect of the monitoring of the labour. She accepted that the CTG drawn by professor Anthony and shown to her was abnormal with at least 8 contractions per 10 minutes at 16:00. However, nowhere in assessments no.4 or 5 did she indicate that she suspected foetal distress. At no stage did she suspect that the foetus was in distress and that action should be taken to remove the stress or to do an emergency extraction or a caesarean section. The second stage in this case was prolonged but she did not write the time when the plaintiff was fully dilated. She did not accept that the fact that dilation improved from 7cm at 15:00 to 10cm at 16:00 which was 3cm in one hour was way faster than the norm and indicative of oxytocin having been administered.

[172] Dr Linde's evidence was that she made an entry on the partogram at 15:00 when she performed a PV examination on the plaintiff. She assessed the cervix to be 7cm dilated and the head of the baby was three to four fifths above the pelvic brim. At this time the plaintiff had crossed the action line on the partogram. She put her on a drip with Ringers Lactate and inserted a catheter. If there were signs of foetal distress syntocinon would not be administered. Ringers Lactate is used to infuse syntocinon. The prescription did not confirm that syntocinon was administered. There would be a tick if it was administered. She was therefore uncertain that syntocinon was administered.

[173] Under cross-examination she testified that when she saw that there was poor progress in labour her decision was to prescribe syntocinon. When she made the entry and the assessment of the plaintiff, she did not record the FHR on the partogram. There was no evidence on the partogram of FHR at about 15:00. There was no evidence of a reactive CTG or a satisfactory FHR for at least an hour before her entries and syntocinon instruction. She said that Ringers Lactate could have been put up for rehydration and not only for syntocinon administration. She intended to have syntocinon administered. When she assessed the plaintiff she was satisfied that there was no foetal distress and there was no indication of CPD. The partogram was incorrectly plotted. The progress of labour was even slower than she appreciated and that by all accounts the labour was severely delayed. The contractions were inadequate from 14:00 which would necessitate the augmentation of the labour with syntocinon. At 16:00 and assuming that it was administered, there

was no indication for it to be stopped. The entry at 14:00 was probably falsified as she only gave instruction for syntocinon at 15:00. There was nothing in the records to indicate that sister Mbada had any reason to believe that there had been a change in the circumstances which rendered the administration of syntocinon unnecessary. If there was tachysystole it would be on overwhelming indication that syntocinon was administered.

[174] Dr Koll's evidence was that he is a semi-retired obstetrician and gynaecologist. He agreed with Dr Murray that there were several significant labour related risk factors for hypoxic brain injury. These were the prolonged labour, the oxytocin infusion, substandard foetal monitoring especially during oxytocin infusion and the second stage of labour was complicated leading to instrumental delivery. However, there was a difference between risk and cause. The definition of tachysystole is more than 5 contractions in a 10 minutes period. That indicates that the uterus was being overstimulated. If contractions were too frequent with no sufficient gap in between the contractions the baby is in a greater threat of having hypoxia. For that reason tachysystole has to be managed on a fairly urgent basis. He was not prepared to comment on the CTGs or that there was evidence of foetal distress as he simply was unable to read those CTGs. He was of the opinion that if the baby was subjected to that degree of hypoxic stress as depicted in those CTGs for an hour before birth, she would have been severely compromised at birth. The baby was clearly not severely compromised. He agreed that tachysystole would increase the risk of an abnormal heart rate and increased hypoxic stress on a foetus but it does not always lead to a problem. He agreed that whether there is a contraction putting stress on the baby or whether there is bearing down putting stress on the baby or both, the fact of the matter was that there should not be more than 5 contractions per 10 minutes as that would increase the risk of hypoxia.

Plaintiff's submissions on negligence and causation.

[175] The following submissions on negligence were made on the basis of which it was argued on behalf of the plaintiff that the defendant was negligent in the care of the plaintiff when she delivered A. It was submitted that sister Mbada's evidence was that she did not have a clear understanding of the completion of the partogram

and made mistakes in the completion of the records. She did not make notes of everything that happened to the plaintiff during labour and did not make notes of what the CTG traces showed at any stage. As there was delayed labour progress, action was mandatory in terms of the guidelines whether it was administering oxytocin or performance of a caesarean section. She was not allowed to override the doctor's orders regarding the syntocinon infusion unless there was a clear change in the circumstances of the plaintiff. If she did not comply with the doctor's orders to infuse oxytocin she should make notes and there was no note made. There were no circumstances recorded that allowed her to ignore the doctor's orders. She accepted that the CTG drawing by professor Anthony was the CTG belonging to the plaintiff and A. It showed foetal distress. She never thought that the baby was in distress. On the basis of all her evidence, it was submitted on behalf of the plaintiff that sister Mbada contradicted herself. She admitted not doing the monitoring of the mother and foetus as required.

[176] About the evidence of professor Anthony and Dr Murray it was submitted that they are both recognised experts in the field of obstetrics. Professor Anthony is a worldwide renowned expert and a South African representative in FIGO, the worldwide body setting standards for foetal monitoring. Both professor Anthony and Dr Murray can read and interpret CTGs. It was submitted that Dr Murray showed a photograph of the original CTG on a screen in court. It was never suggested to her in cross-examination that what she showed as the heart rate and contractions on screen with her cursor did not appear clearly on the CTG tracings. That CTG was clearly pathological and contractions were clearly abnormal and both the pathological heart rate and grossly abnormal contractions could be seen on the screen in court when she testified. Professor Anthony had enhanced the same CTG tracings and confirmed that they were pathological and the contractions were in excess of what normal contractions should look like. Therefore, they deprived the foetus of the rest time to oxygenate properly.

[177] It was submitted that Dr Koll indicated in the joint minute that he could not see what was on that CTG. However, when he testified his evidence was that he could not see the cardio portion (heart rate) section of the CTG but could see the tocograph (contraction) section. Even though he could not see the cardio portion he

then said that one could not interpret the CTG during the second stage because the mother would be pushing. On this basis, it was submitted on behalf of the plaintiff that Dr Koll was not an objective witness. He suggested that the contraction pattern was not clear and normal because the mother was pushing. Professor Anthony and Dr Murray explained why Dr Koll was wrong and what the effect of pushing would show which would be the jaggedness of the lines which was not the case. Dr Koll's evidence was that the tocograph showed the mother pushing uncontrollably. It was submitted that it was irrelevant whether the pattern in the tocograph was produced by contractions or pushing as it remained dangerous to the baby and the nursing staff observing uncontrolled pushing should record it and call a doctor which sister Mbada did not do.

[178] Professor van Toorn, a paediatric neurologist, testified on causation. Submissions were made with regard to his evidence on causation. His evidence was that he assessed the hospital records in this case. He testified that MRI scans do not time the injury. Therefore, an MRI scan cannot tell when the injury happened, whether it was within hours or days but it can tell what the cause of the injury was. In order to time a brain injury a comprehensive approach was required. This would entail looking at the risk factors that could cause brain injury. You look at things like the onset of labour and the period before birth. You look at the growth of the baby, the head circumference, whether there was meconium stained liquor, whether the mother had diabetes. During the intrapartum period and during the process of labour, risk factors are things like whether the labour was prolonged and whether it was properly monitored and whether there were bleeds etc. After birth you look at what could have compromised the baby, lack of oxygen, did the baby collapse, was there infection, was there low blood sugar. All of this is a comprehensive approach to establish causation. He testified that FHR monitoring is vital because it is a sensitive way. If the baby is in trouble, you will pick up abnormalities in the FHR. If a FHR monitoring is done, you will pick up if the baby is in trouble. This is when there is a lack of oxygen to the brain or lack of oxygen to the heart as there will be abnormalities in the FHR.

[179] Professor van Toorn made reference to the guidelines. When there is no adherence to them there is a potential of harm and the guidelines provide best

practices. If there is clear evidence of foetal distress you have to intervene. This is because, the longer the baby remains in a distress environment, the higher the risk of brain injury. What happens is that the baby tries to compensate by shunting blood from the heart and if the distress is too long then it decompensates which leads to brain injury. He referred to the reports of the radiologists, professor Andronikou and Dr Schwartzberg. He pointed out that they both agreed that the pattern of injury on the scan was that of hypoxia ischemia. This means that there was lack of oxygen and diminished blood to the brain. If there is lack of blood to the brain, it causes damage because brain cells require oxygen.

[180] The radiologists concurred that the pattern of injury is hypoxia ischemia and the injuries were of a combined nature. This means that there was a prolonged partial, meaning that it happened over a long time. During that prolonged partial period, the brain tries to protect the core, that middle part, because that is where critical functions occur during respiratory rate consciousness. If there is diminished blood flow to the brain, blood going to the outer surface of the brain is shunted to the inner core to try and protect the middle part which is crucial for life. The scans in this case reflect that. There was prolonged partial happening over hours and at some stage the brain could not direct the blood anymore to protect the core as there was significant compromise. This led to the injury to the centre, the middle part of the brain called the thalamus and peri-rolandic cortex. The MRI imaging showed that there was exposure to hypoxia and lack of oxygen for hours whereafter the compensation probably could not help and there was decompensation which then led to the damage to the middle part, the core of the brain.

[181] Professor van Toorn also referred to a suggestion by professor Andronikou that some of the metabolic causes had to be excluded mentioning three conditions that had to be excluded. He understood professor Andronikou to be saying that hypoxic ischemia was the most likely diagnosis of the brain injury but metabolic causes had to be considered. Upon the blood becoming acidotic through lack of oxygen, the baby tries to protect itself. If hypoxia continues, you get the cardiovascular stage. This is when blood is taken from organs that are not essential like the gut, the skin and the liver and is sent to the heart and the brain to protect these organs. During those two stages there is no injury to the brain even though there is an insult. The

third stage is when there is not enough blood going to the brain. The brain sends blood from the brain's outer surface just to protect the core, the inner surface. Then you get a pattern of injury where there is damage to the outer surface of the brain but the inner core, the middle part is not damaged.

[182] This is called prolonged partial and it usually takes hours. This is because when the uterus starts to contract, it tries to push the baby down which diminishes blood flow to the brain. If there are strong contractions you listen to the FHR every half an hour before, during and after contractions. If that is not done, drops in the heart rate may be missed. When the mother is ready to deliver which is the second stage of labour you listen to the FHR every 5 minutes or after every second contraction. The reason it is done more frequently is because the risk is higher because during all these contractions the baby becomes exhausted especially if labour is prolonged and tired and then there is collapse. During collapse the heart does not pump blood anymore leading to inadequate blood flow to all parts of the brain. This is why there is mixed or combined picture in which there is prolonged partial followed by acute profound injuries.

[183] He referred to the report of Dr Murray in which she said that foetal distress was not acted upon. There was also a prolonged second stage in which the baby was exposed to prolonged contractions than was necessary. Guidelines provide that if the action line is crossed for 5 hours the baby is being put at risk. If a potentially dangerous drug such as oxytocin is administered to an exhausted baby who is exposed to prolonged contractions thus causing the uterus to contract even further that can potentially harm the baby. He testified that even though he did not see the CTGs he had no doubt that there were FHR changes because of the severity of the brain injury. When the baby came out it was flat and needed resuscitation. During the first examination it was said that the baby was lethargic. This means that there was depressed level of consciousness. She was sleepy and was not able to be raised at some stage. She was not sucking and had no suck reflex.

[184] Professor van Toorn referred to Dr Murray's report which referred to decelerations and tachycardia which means the FHR was high. There were hours where there was no recording at times for two to three hours. He testified that where

cerebral palsy was caused by genetics, it would cause brain malformations in most cases. The brain looks abnormal in this case and often where there are genetic causes of cerebral palsy, the image is often normal. He considers genetic causes if a child has cerebral palsy but the scan is normal. In this case with this type of injury, the pattern showed hypoxia ischemia and it was unlikely that there were genetic causes that made this baby vulnerable to hypoxia ischemia. There were none of the risk factors of infection of the placenta or premature labour or the baby being growth restricted. The antenatal period was unremarkable. He agreed with professor Andronikou that the abnormalities are those of chronic evolution of a global insult due to hypoxic ischemic injury of mixed acute profound and prolonged partial variety occurring in the brain of a term maturity. This happened during the labour process based on all the considerations and all the available evidence.

[185] The baby has dyskinetic cerebral palsy as Dr van Rensburg agreed and there were no dysmorphic features and therefore genetic testing was not indicated. He examined the child and could find no abnormalities in the other systems because if you have metabolic conditions sometimes they affect not only the brain but also the heart or the kidney or liver. Other possible conditions were excluded. He was also of the opinion that the baby's head growth was within normal limits. He found no congenital brain abnormalities or metabolic abnormalities. This baby was critically ill and had seizures and required multiple medicines. However, four years later she had no further seizures. There was no evidence of the baby declining or developing new neurological symptoms. When he examined her, the profile he got was consistent with the child's damage during labour from hypoxic ischemia, not metabolic causes. He looked at the evidence of infection of the brain. The CT scan did not report that. The radiologists did not report that there was an infection. Because the baby became very ill and started having fits, the doctors looked for infections and even looked at the possibility of meningitis or blood infection. Blood tests came back negative. The baby was well grown and the head circumference was normal and at the onset of labour the FHR was reported as normal.

[186] He testified that according to Volpe, prolonged labour is a well recognised risk factor. Secondly, the baby needed resuscitation and was reported as being flat. Thirdly, she had neurological syndrome within the first hours or days of life. The first

examination showed the baby being lethargic with no suck reflex. The next morning there was excessive crying, poor feeding and on day four the baby started having fits. Subsequently she had to be hospitalised for 11 days. She needed to be tube feed through the nose because she was not sucking and she was unconscious. The neonatal records support that the baby was born in a compromised state and required resuscitation. That supports foetal distress. He testified that if the baby had a metabolic condition that caused seizures one would expect the baby to have had seizures for her whole life because metabolic problems do not disappear. The baby had no seizures since being discharged from hospital. His opinion was that the way the child developed in the last four years to the time he saw her was consistent with being damaged during labour because of hypoxic ischemia. When there is cerebral palsy, the lesion to the brain is static.

[187] Professor van Toorn's evidence on macrocephaly was that A had a cephalic haematoma which is a bleed outside the brain between the scalp and the bone which could increase the head circumference. However, the ratio between the head circumference and the weight is within limits. If there was a genetic macrocephaly, the macrocephaly would persist. However, that was not the case with A as her head circumference became normal later on. The megalcephaly postulated by professor Rothberg is one of the causes of macrocephaly. Macrocephaly simply means an enlarged head circumference whereas megalcephaly implies the enlargement of the brain. It is a developmental disorder but the MRI imagery did not report it and therefore it can be discarded. Therefore, both macrocephaly and megalcephaly can be discarded.

[188] On apgar scores, professor van Toorn said they should not be interpreted in isolation as they are generally poor predictors of outcome. The apgar score which was 5 at 1 minute is poor as it should be 7 or more. Even the condition of the baby was not in keeping with good apgar scores. He disagreed with Dr Koll that the baby was not severely compromised at birth. All the evidence including notes from the examination of the baby and the brain scan suggest a compromised baby. There was also secondary apnoea which is indicative of a foetus that was deprived of oxygen during the labour phase. On day 2 or 3 there was no serial neurological examination which made it difficult to tell how severe the encephalopathy was. The

absence of a suck reflex which is considered as moderate was not normal. Lethargy is also a sign of moderate neonatal encephalopathy. On seizures his evidence was that young babies do have subtle seizures in which the babies become still or have subtle jerks or myotonic seizures. These seizures can be very difficult to diagnose at an early age even for trained medical staff. Blood sugar was not recorded in the critical first three days of life and yet the baby was at risk of low blood sugar due to the abnormal neurological signs. The baby's brain injury pattern was consistent with exposure or injury due to low blood sugars.

[189] He and Dr van Rensburg agreed that the baby's brain injury was not due to high levels of jaundice at birth. Her clinical picture was not in keeping with a genetic epileptic disorder as the seizures were confined to the period of birth and there was oxygen deprivation and possible sugar and the baby did not have seizures later in her life. The condition of the baby at birth, the intrauterine environment of the baby was not optimal because of secondary apnoea. The suck reflex that should be present shortly after birth was not there at first examination of the newborn. This is therefore a textbook sign of moderate encephalopathy. He was of the opinion that there was a worsening of encephalopathy up until day 3 or 4 after which there was gradual improvement until discharge on day 11. The absence of written notes implies poor management because note keeping is a vital part of management. On day two or three there were no vital signs recorded. There is no evidence of doctors performing a neurological examination and blood sugar was not monitored.

[190] The dietician was concerned about the baby's inability to suck and the speech therapists stated that the baby could not be woken up and had difficulty keeping her awake and had no suck reflex. There was a tongue thrust and she advised against breast feeding. Feeding was to be done with syringe. The baby went into extensions intermittently. Prof van Toorn said that all these were very abnormal severe neurological signs and in his opinion they were part of the evolution since birth. Metabolic and genetic investigations as recommended by professor Andronikou were done and were negative. These investigations were sophisticated. Sophisticated genetic investigations have to be targeted meaning that the laboratory should be told what the suspected underlying cause is so that they know where to look. He has experience and does perform genetic tests with a geneticist. This baby

had destructive, hypoxic ischemic brain changes and there was no neurometabolic condition that could fit the evolution of this picture. He was of the view that the tests that have been done cover any metabolic condition that could cause compromise to newborns.

[191] Professor Smith is a neonatologist who has done extensive research and is a world a claimed neonatologist with considerable expertise in birth asphyxia of babies. He testified that the plaintiff's pregnancy was a high risk pregnancy because the previous baby died after birth following a home delivery. In his opinion A's condition was caused by intrapartum asphyxia for the following reasons. The antenatal period leading up to the labour on 31 December 2015 was unremarkable. The maternity case records state that at 12:00 the FHR was 160 bpm. It was therefore on the verge of tachycardia which is non-reassuring because a normal FHR varies between 110 and 160 bpm. When it reaches 160 you should keep an eye for the non-reassuring foetal condition. He had seen a copy of the CTG trace of around 16:00 which was in Dr Murray's report. He can read CTGs and that trace showed a grossly pathological CTG implying that the foetus was hypoxic. In other words, there was oxygen deficiency. A foetus can withstand quite severe and extended episodes of hypoxia before injury to the brain occurs. What is seen in the CTGs at 16:00 is a foetus exposed to an insult of hypoxia and if that hypoxia goes uninterrupted it will eventually change to acidosis. This means that the tissues of the foetus become deficient in oxygen, cannot operate properly and therefore start making acid which then spills over into circulation which affects organ function especially the heart.

[192] When a CTG is like that, foetal distress must be interrupted quickly to avoid injury. In this case there were no expedited interventions to interrupt foetal compromise which was substandard care. The CTG also showed an excessive number of uterine contractions which means the womb was stimulated to produce the excessive contractions. A foetus needs about 60 to 90 seconds to recover from a deceleration between contractions. His conclusion was that the foetal distress at about 16:00 implied a compromised foetal oxygenation status because there was insufficient time between uterine contractions during which the foetus could be re-oxygenated. Hypoxia resulting in acidosis explains the loss of variability of the FHR

during contractions. This warranted urgent attention. There is no record that the nursing staff recognised the pathological CTG tracing. Therefore no interventions were made such as intrapartum resuscitation and no expedited delivery was planned or performed. Intrapartum resuscitation would have entailed administering maternal oxygen, putting the plaintiff on her side, the discontinuation of the infusion of oxytocin and consideration being given to administering tocolysis. When the midwife completed the assessment of the newborn form she recorded that there had been no foetal distress before birth. This contradicts the doctor who performed the vacuum extraction at about 17:05-17:10 who recorded a FHR abnormality which was initially good and then tachycardia. Tachycardia is one of the signs of probable foetal distress. The second stage of labour was prolonged. At birth the baby was described as being born flat but recovered after bag mask ventilation and stimulation. She was described as lethargic with weak grasp reflex, and absent suck reflex. The baby's breathing was inadequate for the first five minutes or more because the apgar score notes of 1 for breathing at 1 minute and 1 for breathing at 5 minutes. The baby was probably born in the state of secondary apnoea because the baby was flat at delivery, being limp without any efforts of breathing and then required manual breathing support. The one minute apgar more for the heart rate was changed to 2.

[193] Professors Smith and Rothberg agreed that the baby suffered intrapartum asphyxia as stated in their minute. At 17:30 the baby was on nasal prong oxygen. The baby had tachycardia which is a pulse rate of more than 160. The baby's blood sugar level was not checked. New born babies who suffer probable intrapartum asphyxia are prone to develop early neonatal hypoglycaemia which is an independent brain injury factor. Therefore, hypoglycaemia has a contributory role for the injury. That was substandard treatment. The baby's care following a successful resuscitation should have been escalated to a higher level of care. That was not done in the first hours after birth. The records saying that the baby was showing poor latching and hungry were related to irritability as a result of developing cerebral irritation several hours later which is why the baby was crying. The doctors interpreted the baby as crying because she was hungry. The records of inability to latch and crying at around 19:30 were in keeping with stage 1 encephalopathy. The doctors recorded diagnosis of HIE at discharge was in keeping with his assessment.

[194] The condition of the baby after birth included cup feeding which was abnormal. Babies who sustain intrapartum asphyxia typically have the inability to latch, suck and swallow properly. If the baby did not have good sucking and swallowing which persisted, it is encephalopathy that persisted and started to deteriorate at some point but was not recognised until the speech therapist came on the scene at about the 4 January 2016. The speech therapist recognised that the infant could not suck and swallow, had abnormal tongue movements and thrusting. She observed that the infant had back arching movements. That could be in keeping with probable tonic seizures. The infant was jaundiced and none of that was recognised between the 31 December 2015 and the 3 January 2016 by the attending medical staff. Professor Smith opined that until the 3 January 2016 the baby's care was substandard as it should have been escalated to a higher level of care than a normal nurse after the resuscitation. That did not happen which was substandard care.

[195] Blood pressure was never checked, erecting venous fluid was not done, frequent assessment of fluid balance, that is intake and output was not done, restricting fluids was not done, excluding renal failure was not done, maintaining serum electrolytes, calcium, magnesium and acid-base status within a normal physiological range was never done. Calcium was done once. The aim of performing these vital functions is to prevent secondary insults to the brain. The priority was to prevent secondary aggravated brain injury and they did not do that. The baby had been stressed by hypoxia. It will outstrip its glucose supply and develop hypoglycaemia. There is no evidence that the baby's sugar level was checked until day 4. Hypoglycaemia may injure the thalamic pulvinar and the MRI scan shows injury to this part of the brain. The fact that they did not check the blood level sugar during the first two or three days was inexcusable. On 04 January 2016 the baby's level of consciousness was depressed. The baby did not have a rooting reflex, that is, she did not turn her head to suck. The baby reached a level of 313 jaundice which means that she was developing jaundice at least a day or two before starting to become yellow. Yellowness is observed when it reaches a level of 120. There is no indication that it was picked up that she was developing jaundice.

[196] Professor Smith's opinion was that regard being had to the probable seizures, abnormal sucking pattern, level of consciousness, inability to latch, he concluded that at approximately 94 hours, the features were in keeping with stage 2 neonatal encephalopathy or a moderate degree of encephalopathy. The baby's condition followed a rather typical course of birth asphyxia leading to an early onset neonatal encephalopathy, namely a mild encephalopathy. Very inadequate or no assessments were done between the 01 January 2016 and the 3 January 2016. During this period, fluctuations in the baby's level of consciousness and seizures were likely to develop. That was probably missed because of inattention being paid to the clinical condition. The staff would not have picked up the deterioration of the baby's neurological status because they did not perform proper assessments.

[197] They did a cranial ultrasound on 4 January 2016 and found brain oedema. The recorded brain swelling was in keeping with intrapartum sustained asphyxia because brain swelling develops three to five days after the insult and this is exactly what happened. The brain swelling strongly supports an injury of hypoxic ischemia in close proximity to the actual birth, that is labour. Professor Smith's opinion was that the baby exhibited the typical course of an early onset neonatal encephalopathy of moderate degree. The negative metabolic and genetic screening results lead him to conclude that the probable cause for the baby's neurological impairment is directly related to intrapartum asphyxia. The probability of undiagnosed neonatal hypoglycaemia as a contributing factor to the patterns of injury described by professor Andronikou.

[198] Dr Gericke's evidence was for purposes of expressing an opinion on whether there was a pre-existing genetic hereditary factor which may have contributed to an intrapartum injury or cerebral palsy and/or intra neurodevelopmental outcome as currently manifesting in A. He regarded the baby's measurements as normal growth measurements for a new born baby. There were no indications of a family history, MRI findings or clinical findings during assessment which indicated a progressive disorder. There were no external features which indicated a possible existence of an underlying clinical genetic syndrome or chromosome disorder. He described A as having no progressive evolutionary course of neurological features. Hidden genetic conditions have their own characteristic neuroimaging findings and these are either

associated with progressive metabolic disorders, neurodegenerative disorders or intrauterine infections related calcifications. The neuroimaging analysis did not record these. He did not see any atypical features that were present or indicated the need for metabolic and genetic testing. There were no specific clinical or radiological findings to seriously consider infections or toxic exposures during pregnancy.

[199] Genetic evolution needs to take into account a family pedigree, personal history of the child, the course of the development of the child and the findings in a specific instance. The targeted sequencing of several areas of the expressing genes which is the exome – are looking for a number of indications which could be considered relevant in A's case have been found to be negative. The second consideration is that the indications for genetic testing is the absence of an incriminating birth history. The obstetricians have indicated that there was a prolonged second stage and foetal distress before delivery and the baby was delivered with a neonatal encephalopathy. 166 genetic metabolic conditions have been excluded. There is no 100% end point in the extensive GWS and WES. The further you go the more uncertain variants you pick up whose clinical significance is not yet known. Therefore, there is no point where it can be said that everything has been tested. The further one goes down the line, it is the law of diminishing returns.

[200] Dr Gericke was invited to comment on Dr van Rensburg's opinion in which she said that A was the sole survivor of three pregnancies. She developed a moderate degree of neonatal encephalopathy and appears to suffer from dyskinetic cerebral palsy and associated developmental delay in all aspects of functioning. She appears to be developing slowly and is still making progress and has not shown any degeneration or deterioration in functioning and is totally mobile without any help. His response was that all of this means that almost all genetic conditions associated with cerebral palsy can be excluded because they are progressive in nature and are associated with pervasive encephalopathy whereas cerebral palsy is a static encephalopathy.

[201] Dr Cilliers was the second doctor on call and assisted Dr Philips in performing a vacuum extraction. Her evidence was that the CTG was initially good and then there was foetal tachycardia. She performed a bag mask ventilation on A at birth.

After the 31 December 2015 she next saw A on 3 January 2016. On her assessment on 3 January 2016 as recorded in her notes, the baby was breastfeeding. Under cross-examination Dr Cilliers conceded that she only had a vague recollection of certain events and did not remember all the details. The clinical records did not indicate that the baby cried after birth. At 18:30 the baby was still on nasal prong oxygen. The baby's apgar scores were assisted scores. There was lack of the latch reflex. The apgar scores for heart rate were altered from an initial score of 1 to 2. On 5 January 2016 the baby was still on a nasal gastric tube.

[202] Dr Mans was on night duty call on 31 December 2015 and assessed the baby at 22:34. He saw the plaintiff again on 01 January 2016 at 07:30. On 04 January 2016 when he saw the plaintiff again, he recorded a total serum bilirubin of 313. He also recorded that the baby was not breast feeding nicely. At 16:22 on 4 January 2016 he recorded jaundice and c-reactive protein zero which implies no significant infection and inflammation. He then questioned why there was a deterioration and queried hypoxic ischemic encephalopathy. He discussed the matter with a senior colleague Dr Gord. Recommendations for the cranial ultrasound and lumbar puncture were made. This was to make sure that the brain of the baby was well. At this stage hospital records showed that the baby had had a seizure. At 18:21 another seizure was recorded by a nurse. Dr Mans performed a cranial ultrasound which showed only oedema. There was no evidence of infection and bleeding based on CSF results. He performed an HIE score and recorded 6/[22] on 5 January 2016. He prescribed phenobarbitone and midazolam for the seizures.

[203] Under cross-examination Dr Mans conceded that he was not aware of what happened between 01 January 2016 and the 4 January 2016 when he returned. He testified that when he initially assessed the baby she was not on nasal prong oxygen but it was normal to put a normal baby on nasal prong oxygen. Blood glucose level is usually assessed after birth because the baby may have suffered a hypoxic ischemic injury and to assess for hypoglycaemia. A reflex will not just disappear and will only change if there is an insult. Hypoxic ischemic encephalopathy means that the baby was suffering from hypoxia and ischemia which was intrapartum. He agreed that the baby had HIE and ended up with sucking problems. He wrote HIE as a working diagnosis.

[204] With regard to the defendant's expert paediatric neurologist, Dr van Rensburg the following submissions were made for the plaintiff. Her evidence was that Professor van Toorn and herself examined the plaintiff and A. Her information was the third baby was delivered in 2018 by means of a vacuum extraction. It was a male baby. She and professor van Toorn agreed in their joint minute that A was GMFCS level 1. She accepted the reports of the radiologists. She and professor van Toorn agreed that canavan's disease and krabbe's and wilson's diseases were not applicable to A. She regarded the plaintiff's pregnancy outcomes history as a very serious risk factor. The hospital records did not indicate clinical seizures in A for the first 72 hours of life. She and professor van Toorn agreed that there was a neonatal encephalopathy of a moderate degree. They agreed that there was partial prolonged hypoxic ischemic dysfunction and that it can cause acute profound changes. The lumbar puncture performed on A did not indicate a bacterial infection or meningitis. According to her, the HIE score performed on A was done when A was on a midazolam infusion, which cannot be done. She was of the opinion that A displayed an atypical history in the peripartum period and there was a very unusual neonatal encephalopathy.

[205] Under cross-examination Dr van Rensburg testified that she and professor van Toorn agreed that there was neonatal encephalopathy in A which turned into moderate neonatal encephalopathy on 4 January 2016. She had assumed that on the findings of Dr Gericke, there was a change in the motor symptoms of A. During the period 1 January 2016 to 4 January 2016 there were few examinations done on A. On probabilities, if there was intrapartum hypoxia ischemia, then the cerebral palsy was an etiological factor, hypoxia during labour. She did not know whether hypoxia was imposed upon another condition and it was one of the possibilities. With proper monitoring, if there was a genetic vulnerability to hypoxia, it may be detected if that was when the injury was suffered. It was a possibility that in the first three days in hospital with skeleton staff and with relatively few observations, it is highly likely that seizure activity may have taken place while the child was with the plaintiff. There was no indication that toxic conditions brought about A's condition but she was of the view that they had not been totally excluded. There was no evidence to suggest toxic conditions and there were no post-infective causes for the

child's condition. Metabolic conditions have been excluded but not all genetic conditions have been excluded. There was no prematurity and there was no intracranial haemorrhage. There was no evidence of infection, stroke or kernicterus. There was no evidence of oscillated generalized hypotonic, prominent ataxia, signs of peripheral neuromuscular disease, reduced or absent reflex, sensory loss or eye movement abnormalities.

[206] Professor Rothberg was the defendant's expert paediatric neonatologist. His evidence was that he would regard this case as a poor pregnancy outcome. Genetic risk factors must be considered. The court must decide on the proximal risk factor, the labour itself, the labour stress or mismanagement. Clinical risk factors could act as triggers and a clinical risk factor could be intrapartum asphyxia for cerebral palsy where there is genetic susceptibility. A developed and presented with mild hypoxic ischemic encephalopathy between birth and around 16:29 on 3 January 2016. This child suffered intrapartum hypoxic ischemia. The points of disagreement between professor Smith and himself related to the interpretation of the head circumference measurement at birth and the possible implications or relevance of the matter. At the time of birth, A was in primary apnoea and the bag mask ventilation is not an indication that the baby was in secondary apnoea. He and professor Smith agreed that A had an intrapartum hypoxic ischemic brain injury. They agreed that at birth there was a neonatal encephalopathy which resulted from the intrapartum hypoxic ischemia and was assessed as mild HIE. He disagreed with professor Smith that genetic causes have been excluded particularly because a female child born in 2020 also had a postnatal seizure. He did not consider A to have an epileptic syndrome. In his view, the family history could not be ignored and there were significant factors which included four pregnancies, two males died late in pregnancy or shortly after birth, A presented with HIE and subsequently developed cerebral palsy and later developmental delay. He was of the view that one cannot confidently state that an expedited delivery would have avoided the ultimate neurological disability.

[207] Under cross-examination he conceded that A suffered intrapartum hypoxic ischemia. However, he believed that A was in the situation of a primed foetus such

that normal asphyxia became pathological. Volpe and Wassmik⁸ differed about the latent phase improvement. Volpe describes 24 to 72 hours' deterioration. Wassmik describes 6 to 15 hours and after that several days. There was no systematic evaluation of A during the first three days. Recognition of seizures in the new born period can be difficult because of subtle or absent clinical manifestations. Clinical manifestations of neonatal seizures may be overlooked, even by skilled observers and neonatal seizure identification by clinical observation was suboptimal. He disagreed that that the progression of HIE would be typical but agreed that there was a deterioration which was observed by the speech therapist and the nurse when the child suffered a seizure. The head circumference of A was 97th percentile but he was of the view that the big head of A was significant but was part of the phenotype and a red flag. The Ethiopian study would place A at the 92nd percentile. He said that absent the family history in this case there would be little to argue other than the presence and/or severity of foetal distress.

[208] Professor Christianson is qualified as a paediatrician and specialises as a geneticist. He testified that A had congenital macrocephally which is an abnormal enlargement of the head. It is frequently familial, meaning if one of your parents has a large head, you may have a large head. In cases where it is familial it is usually benign because the parent is normal and the child is normal. But it can be genetic in origin associated with metabolic disorder and syndromes. This is another pointer in the phenotype of A to a possibility of a genetic issue. When he examined the child he thought she was GMFCS1. She was not obviously dysmorphic and had no cataracts and no neurocutaneous syndrome. He did not consider that she had cerebral palsy and if she did, it was subtle which he said must be assessed by a paediatric neurologist. He referred to the report of professor Andronikou and his comments that the features may be due to hypoxic ischemic injury of a combined acute profound and partial prolonged nature. He said that the word "may" should not be ignored. Professor Andronikou stated that the pattern of injury may also be seen with toxic metabolic and post infectious disorders. He gave examples of krabbe, canavan and wilsons diseases but it is not an exclusive list.

⁸ Wassink G et al: The Mechanisms and treatment of Asphy encephalopathy: Volume 8 Article 40 February 2014: www.frontiersin.org.

[209] That is why he did the Cento metabolic tests and he considered that the child should be seen by a paediatric neurologist and may require testing for metabolic disorders. On his clinical assessment, A was born with congenital macrocephaly. With regard to the fourth pregnancy, he suggested that this pregnancy should be assessed by an obstetrician. The baby with other members of the family needed to be medically evaluated by a paediatric neurologist and a medical geneticist. Further medical genetic testing on this family needed to be considered. He then referred to the genetic tests that were done and said that the Centogene metabolic test is next generation sequencing based on copy number variation. This is a deep analysis but of a narrow field, just in the field of metabolic disorders. The result was negative which means there were no metabolic disorders diagnosed on this test. The WES can find 80% of problems but if you do a WGS you might get a 20% increased yield of problems or abnormal sequences. However, it is not the full way.

[210] Then Cento-LCV test was done, that is a Cento metabolic test. It is a whole genome in next generation sequencing based on large copy number variation analysis. This also produced a negative result as well. He stated that WES should be done but said doing WGS would even be better as there is nothing beyond it currently. He said if WES comes back negative and there is still a need for genetic investigation you do the WGS. He believed that the family history, the macrocephally, the first four days of life of the baby not being typical of neonatal encephalopathy grade 2 or 3 makes this case qualify for Cento genome. He referred to the correspondence between professor van Toorn and Dr van Rensburg in which it is stated that the plaintiff's obstetrician gave a feedback on the fourth pregnancy which was complicated by obstructive labour and a prolonged second stage. The pelvis of the mother was reported as being insufficient and the maternal height is 1.5 metres. A family history of consanguinity or a sibling or other relative with similar neurological symptoms may suggest a possibility of a genetic condition.

[211] The MRI scan in respect of A was abnormal. He did Cento metabolic and Cento-LCV genetic testing that was limited based on facts that were available after he saw A. Now there are further findings that he cannot explain from a geneticist point of view. The test was not only looking for what is known. It was also looking to the future to find genes that are associated with or can cause CPs that are not yet

known. WES is a powerful tool to identify deficiencies, the likely cause of genetic variants, in particular sequencing of multiple family members, which can reduce a number of candidate gene DNA variants to one or two and thus lead to finality and precise diagnosis.

[212] Under cross-examination professor Christianson stated that A was not dysmorphic, that is she had normal features. There were no neurocutaneous lesions which means birth marks, different types of birth marks which can be associated, and they are dysmorphic features because they are associated with genetic disorders and syndromes. He accepted that she had cerebral palsy but did not have neonatal encephalopathy type 2 or 3. He accepted professor Rothberg's opinion that she had grade 1. His understanding was that on day four, after seizures A developed neonatal encephalopathy grade 2 with seizures. That is not typical of neonatal neurological syndrome as described by Volpe. He would like to see where the neonatologist described hypoxia ischemia enough to cause neonatal encephalopathy grade 1. Professor Christianson was referred to a minute between professor Smith and professor Rothberg in which with reference to the MRI report of professor Andronikou, they say:

“Professor Andronikou described features consistent with combined partial prolonged and acute profound and hypoxic ischemic injury. However, professor Andronikou also indicated that the MRI pattern may be seen with toxic metabolic and post-infectious causes.”

[213] He questioned the way they have worded it with reference to the report. He said what professor Andronikou said about the features **may** be due to hypoxic injury of a combined acute profound and partial prolonged nature. He said that there was therefore a difference between what professors Rothberg and Smith were saying and what professor Andronikou said which he echoed. He did not think that hypoxic ischemic injury was the primary cause. He considered that there may be a hypoxic ischemic injury, there may be metabolic causes, there may be toxic causes, and there may be post-infectious causes. After learning about the third and fourth child, he then considered to add into that list, genetic causes. He was then referred to the report of Dr van Rensburg in which she refers to the defendant's radiologist, Dr

Schwartzberg's report in which the latter said there were no congenital abnormalities, neuronal migration disorders, cysts, hydrocephalies, masses, haemorrhages, blood breakdown products, calcification, midline shift, perfusion restriction. He said from his perspective as a geneticist he saw no neuronal migration disorders or congenital disorders.

[214] He disagreed that both radiologists concurred that the pattern of injury was hypoxic ischemia arguing that professor Andronikou used the word "may" and therefore what Dr Schwartzberg and professor Andronikou said were different. He conceded that toxic and post-infections cause have been excluded to the extent that they could be. Two conditions remaining were the hypoxic ischemia and metabolic causes. The third condition is genetic disorders or congenital disorders. He conceded that in professor Andronikou's report there were only two conditions that remained and metabolic testing has largely excluded one of those two conditions. He agreed that all the possible causes mentioned in professor Andronikou's report have been excluded except for hypoxic ischemic injury. He insisted on the macrocephally which he said was frequently familial.

[215] He was referred to sister Mbada's evidence who said that she took the measurement over the haematoma. He said he did not accept that the haematoma would have been part of the measurement of the head circumference saying that where the cup is placed was not where you would normally do the measurement. Notwithstanding the evidence of sister Mbada, he said that the measurement could not be doubted. The macrocephally was part of what he used to suggest that WGS should be explored. Notwithstanding the evidence of professors van Toorn and Smith that the measurement was unreliable he insisted that A had congenital macrocephally. However, the Cento-LCV test targets, inter alia, macrocephally and it did not detect macrocephally.

Submissions on causation.

[216] It was submitted that the defendant had pleaded a general denial and did not plead that the agreed cerebral palsy was caused by any specific condition. The MRI scans were assessed by professor Andronikou, Dr Schwartzberg and professor van

Toorn. Professor van Toorn and Dr Schwartzberg stated that the scans were consistent with hypoxic injury of a partial prolonged and acute profound type (mixed). Professor Andronikou also stated that:

“However, the pattern of injury in this patient can also be seen with toxic, metabolic and post-infectious causes. Possible metabolic conditions that can have this appearance include Canavan disease, Krabbe disease and Wilsons disease. The patient requires evaluation by a paediatric neurologist and may have to undergo testing for metabolic disorders to distinguish these potential causes.”

[217] On the basis of this statement, the plaintiff argues that nothing further was suggested by professor Andronikou about any further conditions that may provide the picture seen in A’s scan. Professor van Toorn stated that professor Andronikou’s primary aetiology was hypoxic ischemic damage but other possibilities had to be excluded. Metabolic conditions were excluded by the Cento metabolic (sequencing) including NGS based CNV analysis and Cento-LCV – Whole genome NGS large copy number variation analysis. Metabolic conditions were also clinically excluded by professor van Toorn, professor Smith and Dr Gericke. Professor Christianson conceded that genetic metabolic causes have been excluded. Professors Smith and Rothberg agreed that A suffered intrapartum hypoxic ischemia which resulted in encephalopathy after birth which progressed to stage 2 by day four with seizure activity. Speculation about epileptic encephalopathies are irrelevant and were in any event excluded by professor van Toorn and professor Christianson conceded that. No defendant’s expert suggested that epileptic encephalopathies could be the cause of A’s brain injury. Professor Rothberg also conceded that there is little to argue about in this case save for the extent and duration of the hypoxia other than the genetic issue on which he is not an expert.

[218] It was further submitted on behalf of the plaintiff that when one puts everything together, the clinical factors and the events of the relevant period, all fit perfectly into the picture of intrapartum hypoxia ischemia. The factors that are considered important by the plaintiff are the following. There was no adverse antenatal history. The foetus and the plaintiff appeared in good condition during admission to hospital. There was prolonged active phase of the first stage of labour without normal

progress, which is a risk factor for foetal distress. There was a deceleration recorded at 12:00 which is a serious risk factor for foetal distress. There was significant substandard monitoring which is a risk factor that foetal distress will not be detected. There was delayed progress of labour which called for intervention by way of caesarean section or administration of syntocinon subject to careful monitoring as it is dangerous and a risk factor for tachysystole. There was a seriously pathological CTG at 16h00 with bradycardia (decelerations below 110) and tachycardia (heart rate above 160) as well as severe tachysystole with up to 11 contractions in 10 minutes. Only up to 5 contractions are acceptable. This is a serious risk factor for foetal distress and hypoxia ischemia.

[219] The second stage of labour was delayed and again this is a risk factor for foetal distress. The mother was pushing in the presence of tachysystole during the prolonged second stage. Once again this is a serious risk factor of hypoxia. The baby was born flat in a state of secondary hypoxia, ie. not breathing and had to be resuscitated firstly with an ambu bag and then with nasal prongs for at least 1^{1/2} hours. The baby had an absent suck reflex and poor grasp reflex. The baby had to be cup-fed for 4 days at least and developed seizures. An ultra sound scan showed oedema (swelling of the brain) on day 5 which is a typical consequence of hypoxia ischemia. The baby probably developed hypoglycaemia as a result of hypoxia ischemia. The baby's treatment in hospital was suboptimal as the baby was not escalated to a higher facility and not properly tested as she should have been according to professor Smith. The mixed picture of hypoxic ischemia would have developed over hours which is consistent with the clinical picture of probable distress in the foetus. The mechanism of damages as explained by professor van Toorn then fits in with brain damage of partial prolonged and acute profound type as seen on the MRI scan. The child has dyskinetic cerebral palsy.

[220] It was argued that all the above factors constitute a prototype that is normally seen for cerebral palsy caused by hypoxic ischemic intrapartum. All other reasonable causes for the cerebral palsy have been excluded by all the experts. Only professor Christianson was of the view that further genetic testing (WGS) should be done on the family. He cast doubt on what other experts agreed upon. For example, he did not think that A had cerebral palsy but still referred the issue to

paediatric neurologists who both found dyskinetic cerebral palsy. He did not accept that there was encephalopathy which he should have deferred to neonatologists as he is not an expert on that issue. The neonatologists did find positively that there was encephalopathy. Professor van Toorn and Dr van Rensburg also agreed that there was encephalopathy. However, professor Christianson maintained his denial and sought to rely on Volpe. His refusal to concede when he should indicate his bias.

[221] He drove genetic testing for metabolic disorders. The results excluded genetic metabolic causes but still he did not accept the results and insisted on further testing. However, he acknowledged that further testing may not bring about a conclusive result as there are five classifications of variants which may be inconclusive whose clinical manifestations are unknown. To arrive at a conclusive finding will take years requiring input from various experts in medical and genetic fields. This would cause unnecessary protraction to the trial with taxpayer funded cost implications. He did not accept the literature of Pearson et al which postulates that genome testing is contraindicated in cases of a poor obstetric history which happened in A. He made a diagnosis of macrocephally and was the only expert to do so. At the same time he concedes that macrocephally can be benign with a parent having a large head and the child having a large head with no consequences. He would not accept the inaccuracy of the head measurement and ignored the direct evidence of sister Mbada that she took the measurement over the haematoma. The evidence of professors van Toorn and Smith was that they doubted the correctness of the measurement. He did not accept their evidence that the head must be assessed in relation to the length and weight which is normal.

[222] He would not accept professor Smith's view, a world renowned neonatologist, that his personal experience was that head circumferences recorded in hospital are notoriously wrong and over-assessed. Instead he persisted with his opinion that there was macrocephaly even when A's head had proportionally reduced at 5 years. This usually happens when there was a hypoxic injury and the brain does not grow as it should. He conceded that there was no megaloncephally without saying how macrocephaly would be related to the destructive brain damaged that can be observed on the scan. Very significantly, the Cento-LCV test that was done which

inter alia targets macrocephaly came back negative for it. He still persisted that there was macrocephaly. All of this illustrates his bias. Another reason for him going beyond what professor Andronikou suggested, the testing or exclusion of metabolic, toxic or infectious causes, by seeking to do further genetic testing was because of the macrocephaly that he diagnosed. This is contrary to the facts. He made no contribution to resolving the question whether negligence was closely related to the cerebral palsy. The court will assess the evidence on that question on probability, not certainty.

[223] In the final analysis, it was submitted on behalf of the plaintiff that there was only one reasonable explanation for A's MRI picture and that is hypoxic ischemia of a partial prolonged and acute profound type. The defendant does not suggest an alternative reason. In *Goliath*⁹ the court said:.

“When an inference of negligence would be justified and to what extent expert evidence would be necessary would no doubt depend on the facts of the particular case. Questions of absolution from the instance at the close of the plaintiff's case aside, a court is not called upon to decide the issue of negligence until all of the evidence is concluded.... Thus any such explanation as may be advanced by a defendant forms part of the evidential material to be considered in deciding whether a plaintiff has proved the allegation that the damage was caused by the negligence of the defendant or its servants. Here although the procedure performed on Ms Goliath was under the control of the MEC's employees and what they did or did not do was exclusively within their direct knowledge, none of those employees were called to testify. In *Ratcliffe v Plymouth and Torbay Health Authority* (para 48) Lord Justice Brooke made the point that:

‘It is likely to be a very rare medical negligence case in which the defendants take the risk of calling no factual evidence, when such evidence is available to them, of the circumstances surrounding a procedure which led to an unexpected outcome for a patient. If such a

⁹ *Goliath v MEC for Health, Eastern Cape* 2015 (2) SA 97 (SCA) para 17-18

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

Lowe J appears to have allowed himself to be diverted from the obvious inference of negligence dictated by the evidence in this case by virtue of his heightened focus on the applicability of the maxim *res ipsa loquitur* to cases based on alleged medical negligence. He appeared not to appreciate that:

'At the end the trial, after all the evidence relied upon by either side has been called and tested, the judge has simply to decide whether as a matter of inference or otherwise he concludes on the balance of probabilities that the defendant was negligent and that that negligence caused the plaintiff's injury. That is the long and short of it.'

Defendant's submissions.

[224] In its simplest expression the defendant's case is that negligence is denied in that on admission at the hospital at 09:00 on 31 December 2015, having been referred by a clinic, the plaintiff was soon put on CTG monitoring which was ongoing. To the extent that some of the activities of the continuous monitoring of the plaintiff during labour may not have been properly recorded, that did not translate into a poor management of her labour which was, throughout, within the acceptable norms and standards especially as provided for in the guidelines. With regard to causation, the defendant's case is that in the first instance, even if it were to be found that the defendant was negligent, such negligence did not cause the child's poor outcome. The defendant contends that in light of the poor pregnancy history of the plaintiff, the macrocephaly that the defendant alleges the child suffered from together with the possible genetic causes, the child's condition was not caused by the alleged poor monitoring but by a predisposition to a poor outcome. This required further genetic investigation which was prevented by the plaintiff who refused genetic testing despite a viable blood sample that was readily available.

[225] For its defence, the defendant relied on the evidence of four factual witnesses and four expert witnesses which has largely been referred to above. Some of it is again referenced below in order to contextualise the defendant's contentions in some respects. The defendant submits that the plaintiff was first seen at the clinic at 06:50 and consequent upon being referred to Zitulele Hospital by the clinic, she was attended to shortly after her arrival at 09:00 at the said hospital where preliminary examination was done as recorded in the maternity case records. Sister Mbada's own assessment was at 12:50 at which stage the plaintiff had a spontaneous rupture of the membranes and was draining clear liquor. The FHR was between 136 and 150 bpm and the monitoring was done using CTG. During the next assessment at 14:00 the FHR was noted as 145 to 150 bpm. Sister Mbada noted that the labour progress was slow. As a result, she notified the doctor who ordered syntocinon which was to be administered if CTG was reactive. She understood this to mean that the CTG would be monitored over a period of 30 minutes to check if its baseline was between 110 and 160 bpm and ensuring that there were no decelerations in line with the guidelines. Only in those circumstances would the syntocinon infusion be done.

[226] However, syntocinon infusion was not done because if it had been administered, the syntocinon administration document which is clipped in the patient's file is ticked each time the required amount of infusion is administered as prescribed until the infusion is stopped. That document would be signed at the start of the infusion process. Even the stopping of the infusion would similarly be indicated and documented. Based on the absence of any of these indicators, she concluded that it was never administered even though it had been prescribed because the labour progressed naturally. The defendant contends that the fact that labour progressed naturally was evident from the assessment at 14:00 and the partogram. The contractions as is evident on the partogram became stronger, making it unnecessary to administer it. At 15:15 the contractions were serious and strong and therefore it was unlikely that she would have gone ahead and administered syntocinon. Only Ringers Lactate was administered in preparation for possible syntocinon administration and the hydration of the patient.

[227] The submissions of the defendant on Dr Linde's evidence, one of its factual witnesses was that she was on duty on 31 December 2015 and her duties included attending at the maternity ward. At 15:00 she made an entry after having assessed the plaintiff. Consequent upon that assessment she ordered that if the CTG was reactive the plaintiff should be given syntocinon to augment her labour and a catheter should be inserted to empty her bladder to prevent labour obstruction. She allowed labour to continue as there was no risk in allowing it to progress naturally. It was not necessary to perform an urgent caesarean section or any other urgent intervention. It was further noted that she should be called if there were any problems. Her assessment of the foetal wellbeing was through the CTG through which she checked for any signs of foetal distress. She made partogram entries at 15:00 where she plotted on the lower part that the dilation was 7cm. She explained that the Ringers Lactate was often used in the labour ward as it contains electrolytes and lactate. It is a supportive fluid commonly used in rehydrating patients. Therefore, it was denied that the administration of Ringers' Lactate was suggestive of the syntocinon having been administered.

[228] Her evidence was further that at Zithulele Hospital they had a prescription sheet which indicated dosage rates for syntocinon administration as well as time intervals for its administration. Therefore, its mere prescription did not equate to its actual administration, maintaining that it was never administered. The defendant argued that Dr Linde's evidence was that tachysystole was not only attributable to syntocinon infusion but does happen naturally during labour. Therefore, it was denied that the occurrence of tachysystole, even if it were to be accepted that it occurred, was indicative of syntocinon infusion. However, to the extent that the tachysystole hypothesis was premised on syntocinon infusion based on disputed CTG tracings and further premised on disputed enhanced CTG drawings, it was vehemently denied, it being the defendant's case that syntocinon was not administered. It was further argued that the foetal wellbeing of the baby was based on a running CTG even though the recordal of the findings of the observations of the CTG might have been suboptimal.

[229] The defendant's case was further based on the evidence of Dr Cilliers, one of the defendant's factual witnesses who was on duty at Zithulele Hospital on 31

December 2015 as the second doctor on call. Her evidence was that the vacuum extraction began at 17:05 and was completed at 17:10. She was called by Dr Philips to assist him in delivering the baby. On her arrival Dr Philips explained that the plaintiff was 16 years old with para-zero grav2 with a delayed second stage. Dr Philips was a junior doctor and she decided to upskill him by allowing him to do the vacuum extraction under her supervision. When she arrived the plaintiff was in labour with CTG that was running and the midwife was also present, who presumably called Dr Philips. She assessed the CTG tracings to see if it was normal, suspicious or pathological and found it reassuring. On that basis she excluded foetal distress and allowed Dr Philips to complete the vacuum extraction which was uneventful.

[230] Another factual witness of the defendant was Dr Mans. On 31 December 2015 he was on duty between 19:00 on 31 December 2015 and 08:00 on 01 January 2016. He examined the plaintiff and the baby when he did his postnatal rounds at about 22:34. He noted a third degree tear on the plaintiff and found that she had no clue on how to breastfeed. He noticed that the baby was hungry and there was poor latching. He based his view that the baby was hungry on the fact that the baby was crying and that babies cry a lot when they are hungry. His evidence was that there was nothing that concerned him about the baby who was basically lodging with her mother as she had been admitted for her third degree tear. Ordinarily babies were allowed to go home after six hours but this baby had to stay with her mother. He was adamant that the child was hungry and displayed the primitive reflexes necessary for breastfeeding. However, the mother was unable to initiate breastfeeding. He saw the mother and the baby again on 01 January 2016 at 07:30. He noted that the baby was hungry but could not feed because of the lack of feeding connection with its mother. He proposed cup feeding to deal with the child's nutritional needs in light of the mother's inability to breastfeed the hungry baby.

[231] He next saw the baby on 04 January 2016 at 16:00. There was a nurse's note that the baby had started fitting. This was the first occasion that the child started having fits. It was argued that this was not in keeping with a classical case of neonatal encephalopathy as the onset of seizure was after four days. He testified that at 18:21 on 4 January 2016 the nurses observed further seizures. It appeared

that there were no further seizures on 05 January 2016 at 17:29 as there was a note that there had been no seizures since 21:26 the night before. Under cross-examination he testified that in ascribing poor latching to the inability of the mother to breastfeed he had not made a mistake as he had watched, as per the notes, the mother breastfeeding. He never diagnosed HIE but merely queried it. It was argued on behalf of the defendant that Dr Mans' evidence supported the defendant's contention that the child did not display any features that were in keeping with a typical HIE.

[232] This brings me to the defendant's contentions on its expert witnesses' evidence. Dr Koll was the defendant's obstetrician and gynaecologist. He concluded a joint minute with Dr Murray, the plaintiff's obstetrician and gynaecologist. His evidence was that initially he and Dr Murray believed that syntocinon had been administered. He and Professor Anthony did a joint minute in which they agreed that there was nothing detectable antenatally which could have affected the outcome in this case. He further testified that there were discrepancies in the assessment of cervical dilation. He agreed with Professor Anthony that in monitoring FHR, one must listen after a contraction in that a random measurement of the FHR does not tell anything. Neither he nor Professor Anthony could explain the 5cm dilation at 14:00. There was another assessment at 15:00 by Dr Linde who ordered that CTG should be monitored and that the labour should be allowed to progress. She had excluded CPD and foetal distress and with the membranes having ruptured at 12:50 and draining clear liquor, she was assured of the foetal condition.

[233] He agreed with the doctor who, in light of the clear liquor, ordered catheter to drain the bladder, putting up a drip for Ringers Lactate which was important for rehydrating the plaintiff and ordering syntocinon which could be infused if the CTG was reactive. He would not fault the management of the labour until 16:00 which was about an hour before delivery. During assessment no.4 the FHR was 150 bpm which was reactive and reassuring of the foetal wellbeing and the plaintiff was allowed to bear down. Dr Koll found that to be reasonable. He would not comment on the CTG tracings which he said were unreadable as they were illegible. His evidence was essentially that besides poor record keeping, his assessment of the

actual management of the labour was that he could find no fault in how the defendant's staff managed the plaintiff's labour in that she was kept on a continuous CTG which was reactive and there was clear liquor which was indicative of the absence of foetal distress.

[234] The defendant argued its case for genetic testing on the evidence of Dr van Rensburg, professor Rothberg and professor Christianson. Dr van Rensburg signed a joint minute with professor van Toorn in which they agreed on the need for metabolic and genetic testing following the discovery of the information that the plaintiff had given birth in 2014 and a third one in 2018 both of which were still born births. They agreed that the fact that both children who were still borns were males was an indicator for genetic testing. She had classified A as GMFCS 1 which was an indicator of her motor functionality. She however, later discovered that Dr Gericke had classified A as GMFCS 4 which is severely disabled. This baffled her as it meant that the child's functioning was fluctuating which was not in keeping with cerebral palsy. This was an indicator for a genetic predisposition and not negligence or substandard care which might have been responsible for the child's condition.

[235] The defendant argued that Professor Andronikou's report was that metabolic and genetic causes may be responsible for the MRI findings. Therefore, it would be unjust to find in plaintiff's favour as the plaintiff actively prevented the defendant from pursuing what it considered to be a viable defence by her refusal to have further genetic testing done. The defendant argued that it is incorrect to distinguish metabolic disorders from genetic disorders. This is because metabolic disorders including the possible ones mentioned by professor Andronikou, are usually genetic in nature.

[236] Reference was also made to Dr van Rensburg's evidence that on 4 January 2016 A qualified as having moderate encephalopathy. According to Volpe seizures usually start within 6 to 12 hours and before 72 hours in a hypoxic ischemic brain injury. In this case there was a deviation from that norm. It was pointed out that the most common pattern of deep grey matter injury in a term infant with cerebral palsy is the involvement of the thalamus and globus pallidus. One usually sees a ventrolateral thalamic involvement. There can be involvement of the posterior of the

internal capsule and the posterior part of the putamen. But if there is a severe injury the whole thalamus, the whole putamen including the globus pallidus and the head of the caudate nucleus can be involved.

[237] It was submitted that it is noteworthy that in plaintiff's case the injury was on the medial aspect of the thalamus and the head of the caudate nucleus. It is not the typical basal ganglia involvement that is seen in a full term hypoxic ischemic brain injury. Therefore, one cannot say what the exact cause of the injury is and the question remains open. It was argued that Dr van Rensburg's opinion was that A displayed atypical history in the peripartum period and there was an unusual neonatal encephalopathy. Distal factors needed to be considered which included the very poor pregnancy history which complicated the situation. Radiological findings were atypical of hypoxic ischemic brain injury. Dr van Rensburg would not accept the suggestion that Dr Gericke made a mistake about the child's GMFCS level in the absence of clinical notes of his assessment on the day he examined the child which he failed to produce.

[238] The defendant's case is also that professor Rothberg and professor Smith differed on the pathways for cerebral palsy. Professor Smith argued for pathway A which is that as a result of the insult during the management of the labour, the injury was caused. Professor Rothberg on the other hand, was of the opinion that while he could not conclude whether there was additional asphyxia as a result of the obstetric management, he agreed that there was intrapartum hypoxia ischemia. The neurological condition (encephalopathy) worsened around the 4 January 2016 which was after 89 hours. He agreed with professor Anthony that infection has been reasonably excluded. They agreed that their diagnosis of cerebral palsy was based on the joint minute of the paediatric neurologists as neither of them had seen the child. He was also of the view that on the child's assessment at 22:34 by Dr Mans, the latter was so unconcerned about the condition of the baby that he made a note to review the child on discharge. The deterioration when the seizures occurred at 16:00 and he was called and saw the child at 16:22 some 95 hours from birth, was atypical for a baby to deteriorate with seizures. He was of the view that the fact that the plaintiff had four pregnancies, two males dying late in pregnancy, one female with HIE which developed into cerebral palsy and global developmental delays, the

second female also had seizures on the second day of life were all good indicators for genetic testing.

[239] All of these poor pregnancy outcomes spoke of an underlying problem in the family rather than bad obstetric management, which also called for genetic testing. The onset of seizures in A was late for a progressive HIE. According to Volpe, other causes for the neonatal encephalopathy should be sought. Even the equivocal nature of professor Andronikou's report in which hypoxia-ischemia and the possibility of other conditions including conditions with a genetic basis pointed to the necessity for genetic testing. His view was that definitive tests have not yet been done to conclusively rule out a genetic condition that may have predisposed A to intrapartum asphyxia. On the other hand, intrapartum asphyxia may have been an independent factor that was superimposed on a genetic condition. Therefore, one cannot state that expedited delivery would have avoided the ultimate neuro-disability.

[240] His evidence was that pathway A looked only at intrapartum asphyxia as a result of obstetric mismanagement. Pathway B is when there is a genetic risk factor and other distal factors. He agreed that there was intrapartum asphyxia which may have been related to the usual stress of normal birth but it acted adversely in this case because the brain had been primed by the underlying genetic condition. In this case there was a problem that occurred at the time of conception in the form of genetic abnormality regard being had to the history of the four affected children. There are always elements of hypoxia and ischemia in every pregnancy. In a baby that is susceptible, that is primed, the brain may be triggered to undergo energy failure and present with neonatal encephalopathy and ultimately cerebral palsy which is what would have happened in pathway B.

[241] The last expert witness for the defendant on which the defendant argued its case on causation was professor Christianson. On his evidence it was argued that there are other genetic epilepsies which have not been excluded by the gene sequencing by the Cento metabolic panel. Other panels could be tested but it was better to just do the WES or WGS. He added that at present there was nothing beyond the WGS.

The evaluation of the evidence.

[242] The legal position and guidance on how medical evidence should be evaluated was stated as follows in *Oppelt*¹⁰ by the Constitutional Court from which I quote generously:

“The correct approach to the evaluation of medical evidence is the one laid down by the Supreme Court of Appeal in *Linksfeld* where it held that –

‘it is perhaps as well to re-emphasize that the question of reasonableness and negligence is one for the court itself to determine on the basis of the various, and often conflicting, expert opinions presented. As a rule that determination will not involve considerations of credibility but rather the examination of the opinions and the analysis of their essential reasoning, preparatory to the court’s reaching its own conclusion on the issues raised.

...

Although it has often been said in South African cases that the governing test for professional negligence is the standard of conduct of the reasonable practitioner in the particular professional field, that criterion is not always itself a helpful guide to finding the answer.

...

That being so, what is required in the evaluation of such evidence is to determine whether and to what extent their opinions advanced are founded on logical reasoning. That is the thrust of the decision of the House of Lords in the medical negligence case of *Bolitho v City and Hackney Health Authority* [1997] UKHL 46; [1998] AC 322 (H.L.C (E.)). With the relevant *dicta* in the speech of Lord Browne-Wilkinson we respectfully agree. Summarised, they are to the following effect.

The court is not bound to absolve a defendant from liability for allegedly negligent medical treatment or diagnosis just because evidence of expert opinion, albeit genuinely held, is that the treatment or diagnosis in issue

¹⁰ *Oppelt v Head: Health, Department of Health Provincial Administration: Western Cape* 2016 (1) SA 325 (CC); 2015 (12) BCLR 1471 (CC) para 36.

accorded with sound medical practice. The court must be satisfied that such opinion has a logical basis, in other words that the expert has considered comparative risks and benefits and has reached 'a defensible conclusion' (at 241G-242B). If a body of professional opinion overlooks an obvious risk which could have been guarded against it will not be reasonable, even if almost universally held (at 242H).

A defendant can properly be held liable, despite the support of a body of professional opinion sanctioning the conduct in issue, if that body of opinion is not capable of withstanding logical analysis and is therefore not reasonable. However, it will very seldom be right to conclude that views genuinely held by a competent expert are unreasonable. The assessment of medical risks and benefits is a matter of clinical judgment which the court would not normally be able to make without expert evidence and it would be wrong to decide by a simple preference where there are conflicting views on either side, both capable of logical support. Only where expert opinion cannot be logically supported at all will it fail to provide 'the benchmark by reference to which the defendant's conduct falls to be assessed' (at 243A-E).

...

This essential difference between the scientific and the judicial measure of proof was aptly highlighted by the House of Lords in the Scottish case of *Dingley v The Chief Constable, Strathclyde Police* 2000 SC (HL) 77 and the warning given at 89D-E that:

'[O]ne cannot entirely discount the risk that by immersing himself in every detail and by looking deeply into the minds of experts, a judge may be seduced into a position where he applies to the expert evidence the standards which the expert himself will apply to the question whether a particular thesis has been proved or disproved – instead of assessing as a judge must do, where the balance of probabilities lies on a review of the whole of the evidence.'

The CTG tracings.

[243] There has been a lot of evidence about the plaintiff having been on a running CTG for the most part of the labour. However, and I might add, regrettably, those CTG tracings have allegedly faded. There are also allegations that they may even have been tampered with deliberately in some places. I do not think that this Court should comment much on the tampering issue, especially an attempt to apportion blame for the alleged tampering, if indeed it actually happened without any form of evidence. It did not help that no document expert was called to testify so that the court would be able to ascertain that the CTGs were not just damaged or had not just faded. They had, in those parts, been deliberately tampered with. I simply do not think that this Court should be expected to answer that question nor was it asked to make a finding on it. It would be gravely concerning though were it to be true as it is laced with pure criminality and an attempt to hide the truth by the obliteration of evidence. However, it appears that the CTG tracings from about 16:00 to about 25 minutes or so thereafter were not affected by the alleged tampering. They seem to have only been affected by fading. It was never suggested by any of the parties that anything else beyond fading might have happened to that portion of the tracings. Consequently, I intend to deal with those CTG tracings which were the only ones about which some of the experts gave evidence.

[244] The defendant contends that professor Anthony's evidence based on the enhancement of the photos of the CTGs which were taken from the original CTGs by Dr Murray is not established on facts and is therefore speculative even to the extent that anything could be made out of them. The defendant argues that even where clear and legible CTGs which appear abnormal are available, CTGs do lend themselves to different interpretations by clinicians of the same level of seniority and are therefore an unreliable tool. The defendant rejected professor Anthony's enhancement which it called a drawing in circumstances in which even a document examiner was unable to enhance the faint or faded original CTGs which the document examiner had been requested to do. Therefore, the defendant rejected any opinion based on professor Anthony's enhancement. The defendant, in a nutshell, argued, even on the basis of those CTGs that the plaintiff has failed to prove that its employees acted negligently when attending to the delivery of A by the plaintiff. It further argued that the plaintiff also failed to establish, on probabilities, that any negligence as the court may find, caused the injury in A.

[245] The issue of the CTGs' illegibility and the evidence of the plaintiff's experts both Dr Murray and professor Anthony and indeed that of the defendant's obstetrician, Dr Koll and Dr van Rensburg, the defendant's paediatric neurologist has made them one of the key cogs in the assessment of the evidence in this matter. Perhaps it is important to state the obvious fact that parties are poles apart about the admissibility of the faded CTGs and their enhancement. Therefore, the relevance of the evidence that was given by the plaintiff's expert witnesses in seeking to rely on them to the extent that the plaintiff contends, they prove the existence of foetal distress and syntocinon infusion at the relevant time is disputed. The defendant disavows their relevance and in fact contends that there are no CTGs to speak of as the CTGs were unreadable due to fading. It is common cause that during the hearing of this matter in the open court in Bhisho on 28 November 2019 the original maternity case records were available in court. Dr Murray was given access to them and saw that the original CTGs had faded. However, she felt that some areas in them were readable especially the CTG tracings from about 16:00. She could read the FHR and contraction patterns on them. She decided to take some pictures using her cellphone so that she could expand the pictures for better legibility. It is common cause that photocopies of those pictures were made part of the court bundles and were included in Dr Murray's addendum report and were dealt with in her joint minute with Dr Koll.

[246] When she testified, her cellphone pictures were shown in court during trial by being projected on a screen in court when she testified about them. At that stage there was no objection to her evidence as far as the admissibility of the photographs was concerned or her evidence in that regard. It was never contended that she could not give evidence based on her pictures on the basis that her pictures were inadmissible. What has always been the defendant's contention was that the original CTGs had faded and were consequently unreadable and nothing could be made of them. It was never contended that she could not have enhanced their visibility and testify on the basis of what could be gleaned from them or that her cellphone enhancement was inadmissible.

[247] In essence the plaintiff contends that the evidence of both Dr Murray and professor Anthony should be assessed and weighed together with the rest of the evidence in this matter. This would include their evidence on the enhanced CTG tracings and professor Anthony's own enhancements which he described how he did it in his evidence. His evidence was that he used Dr Murray's photocopy, and with a felt pen, made that copy more readable. He also used a good light and a magnifying glass so that he would be able to be properly guided as he did his enhancement with a felt pan. The real issue is, in my view, less about professor Anthony's enhancement or enhanced CTG copy but more about Dr Murray's photographs of the CTG about which she testified in court and was in fact cross-examined with no objection. During professor Anthony's evidence when he was cross-examined, it was never suggested that his drawing was in anyway at variance with the photograph taken by Dr Murray and on which she led evidence in court.

[248] With regard to all this evidence about the CTGs from 16:00 or there about to the end and the photographs that Dr Murray made which found their way into the court bundle in terms of the Rules of Court, the plaintiff relies on *Protea Assurance*¹¹. On that authority the plaintiff submitted that Dr Murray's evidence on her photographs of the CTG tracings and professor Anthony's enhancement thereof is admissible evidence from which the court is entitled to draw certain inferences and conclusions in the normal course of its evaluation of the entire evidence. The court said in *Protea Assurance*:

"The remarks to be found in the cases to which I have been referred and in which the concept of a document has been considered, were not intended to be exhaustive expositions of what the word comprehends and they certainly do not positively support the notion that a photograph cannot be regarded as a document. See *Seccombe and Others v Attorney – General and Others* 1919 TPD 270 at 277 and *Sneech v Hill Kaplan Scott and Partners* 1981 (3) SA 332 (A) at 338. Nor is it important, for present purposes, to distinguish between a photograph which amounts to 'reele getuienis', as Schmid calls it in *Bewysreg* 3rd ed at 341, and one which is taken to prove the existence of that

¹¹ *Protea Assurance Co Ltd v Waverly Agency CC & Others* 1994 (3) SA 241 (CPD) at 250 B-G.

which has been photographed. Rule 36 (4) shows, I think, that a photograph was regarded as a document by the framers of the Rules for reference is made in that subrule to 'medical reports, hospital records, X-ray photographs or other documentary information of a like nature' (my emphasis). The use of the word 'other' after the inclusion of X-ray photographs indicates, to my mind, that photographs were regarded by the framers of the Rules as documentary information. Furthermore, as was pointed out during argument, if photographs are not covered by the use of the word 'documents' in Rule 35 (1), no provision would have been made in the Rules for discovery in the ordinary course of photographs. It seems most unlikely that photographs were intended to be excluded. The fact that specific provision regarding photographs in some of the other Rules does not point to a contrary conclusion, for those provisions relate only to those photographs which a litigant proposes to use at the trial, and would have remained privileged but for the litigant's desire to use them at the trial. Such provisions do not cater, as Rule 35 (1) does, for the possibility that the litigant's adversary might wish to make use of a relevant, but unprivileged, photograph in the former's possession. I conclude, therefore, that the photographs in question are documents within the meaning of Rule 35(12) and that respondents are entitled to inspect and copy them."

[249] The defendant did not make any countervailing argument on the basis of which it was sought to suggest the basis on which Dr Murray's photographs of the original CTGs should not be accepted as real evidence. I was not referred to any authority on the basis of which Dr Murray's photographs should be deemed inadmissible or that her evidence on them should not be assessed together with the rest of the evidence. As I said before, no suggestion was made that there were any differences between what the defendant referred to as professor Anthony's drawing and Dr Murray's photographs in a way that sought to suggest that professor Anthony might have either erred or been inaccurate or came up with a document or drawing that was, in even a minute way, at variance with Dr Murray's photograph or photocopy. What the defendant sought to do was to adopt an obstructionist approach to that evidence and to professor Anthony's enhancement and indeed refused to engage

with the CTGs on the basis that they were illegible or unreadable and in fact the defendant submitted that there were no CTGs to speak of.

[250] Even Dr Koll's refusal to engage with those CTGs on the basis that they are unreadable was difficult to understand. He vacillated between saying that the entire CTGs were unreadable to saying that only the cardio portion was unreadable but the tocograph portion was readable. That is how he propagated his evidence on a mother pushing uncontrollably. In other words, he would not engage on those CTGs but where engaging on them suited his views he was prepared to give evidence on them. His refusal to engage with Dr Murray's photographs, his refusal to engage with the enhanced version or drawing by professor Anthony and in fact his selective refusal to have anything to do with the faded CTGs to the extent of not dealing with Dr Murray's photographs on the basis that they were made from faded original CTGs is troubling for an expert witness. One would have expected him to, at the very least, directly deal with them even if conditionally upon them being an accurate reflection of the actual CTG tracings. He never made any of the attempts that were made by his colleagues and counterparts and fail to see what they could see. After all, it is for the court to determine the admissibility of any evidence. A witness cannot choose not to engage with any evidence based on her or his views of that evidence.

[251] This approach by an expert witness is concerning as it seems to be at variance with the duties of an expert witness in court proceedings which were reaffirmed recently in *MEC for Health, Limpopo*¹² by Molemela JA (as she then was) as follows: "The functions of an expert witness were explained by this Court as follows in *McGregor and Another v MEC for Health, Western Cape*:

'... The functions of an expert witness are three fold. First, where they have themselves observed relevant facts that evidence will be evidence of fact and [be] admissible as such. Second, they provide the court with abstract or general knowledge concerning their discipline that is necessary to enable the court to understand the issues arising in the litigation. This includes evidence of the current state of knowledge and generally accepted practice in the field

¹² MEC for health, Limpopo v LWM obo D M (502/2021) [2022] ZASCA 146 (27 October 2022) para 17.

in question. Although such evidence can only be given by an expert qualified in the relevant field, it remains, at the end of the day, essentially evidence of fact on which the court will have to make factual findings. It is necessary to enable the court to assess the validity of opinions that they express. Third, they give evidence concerning their own inferences and opinions on the issues in the case and the grounds for drawing those inferences and expressing those conclusions.”

[252] Dr Koll did not perform the functions of expert witnesses as explained above when it comes to the CTG tracings. His approach was to evasively and in some ways obstructively refuse to engage with that evidence altogether even if subject to any conditions and reservations as he could have. This unfortunately gave the impression of an expert witness who refused to say something that was not aligned with the defendant’s postulations or defence as if he was in court as a defendant’s witness as against an expert called by the defendant. I think that there is a significant difference between the two. I accept the photograph of the CTG tracings taken by Dr Murray and I accept the enhancement thereof by professor Anthony being satisfied as to how he went about doing the enhancement to make the picture more legible which he explained in his evidence. Therefore, the evidence of Dr Murray and that of professor Anthony on the CTG tracings from about 16:00 will be considered as will the rest of the evidence to assist the court in determining what could reasonably be concluded about the foetal condition during that final hour before the birth of A. That evidence will also be factored in in determining both the issue of negligence and the issue of causation and be considered together with the entire body evidence in this matter.

The syntocinon infusion.

[253] This brings me to the similarly contentions issue of syntocinon infusion. The evidence of sister Mbada was that when she assessed the plaintiff at 14:00 she found her to be still 7cm dilated. This meant that she had remained at 7cm for two hours from 12:00 which called for action. She informed the doctor who prescribed syntocinon which was to be administered if the CTG was reactive. She accepted that there was no note at 14:00 which showed what the CTG tracings reflected at

that time. She insisted that although she could not remember the events of that day she did not administer syntocinon. If it were to be accepted that the doctor's instruction for syntocinon infusion was given at 14:00, it would have to be accepted that in line with her evidence that she would need 30 minutes to observe the CTG which would take her to 14:30. There was no note at 14:30 in the same way that there was none at 14:00 indicating what the CTG tracings reflected. Therefore, there is no way of knowing what the CTG showed between 12:00 and 14:30 for two and a half hour. There is another troubling feature about the defendant's case in this regard. Dr Linde distanced herself in her evidence from having ordered or given an instruction for syntocinon at 14:00. In fact she said that such entry in the notes was falsified. This says a lot about sister Mbada as the maternity case records, where she is concerned, are punctuated by many things from inconsistencies, gaps and writing over to changing some of the original entries that she had made. When she would have made those changes is difficult to tell. This is over and above the alleged tampering with certain portions of the CTGs for which nobody has taken responsibility.

[254] If the CTG was reactive at 14:30 with the 30 minutes CTG observation time having lapsed she should start syntocinon infusion. There is no note indicating that it was started or that it was not and the reason therefor. If she started it at 14:30, this would explain the improvement in cervical dilation at or about 15:00. Her evidence was that the reason she would not have done the infusion would not be because the CTG was not reactive. There was a possibility that from 14:00 and 16:00 contractions would have changed making it unnecessary to administer it. Her evidence was that contractions were stronger even though their frequency did not improve. She testified that between 14:30 and 15:00 the doctor arrived. It is common cause that Dr Linde made a note at 15:00 indicating that the plaintiff was still 7cm dilated. This means that for two hours from 12:00 to 14:00 she remained 7cm dilated.

[255] However, and inexplicably there was no note of what the CTG showed and there was no partogram entry by Dr Linde reflecting the foetal well-being. If prescription was actually made at 15:00, it was done without any indication of the foetal well-being in circumstances in which the CTG was said to be running. Her

evidence that she could not have prescribed syntocinon without looking at the CTGs is neither based on fact nor what she could remember in circumstances in which she reposed a lot of confidence on sister Mbada. It is common cause that Ringers Lactate was put up in preparation for syntocinon infusion and the catheter was inserted. There was basically no reason for not administering syntocinon except of course if the CTG was non-reactive but the partogram is silent about the CTG readings at that time. The proposition that despite syntocinon having been prescribed and all the necessary preparations for its infusion having been made, the sudden cervical dilation improvement from 7cm to 10cm in one hour after the prescription was done was a mere coincidence is difficult to understand. So is the sudden phenomenon of a natural oxytocin which was not there at any time before the prescription or at least did not have this huge and remarkable effect on contractions and dilation. This would mean that the dilation that was not happening at 1 cm per hour for 7 hours or so, suddenly made a quantum leap of 3cm in one hour on its own. This defies logic.

[256] The only reason given both by sister Mbada and Dr Linde for the idea that syntocinon was not infused despite all its indications being met on their evidence and preparations having been made is the fact that they did not make notes or ticks indicating that syntocinon was infused. This is bewildering from both of them as they both failed to make crucial notes about the foetal well-being at crucial times and in fact ignored the guidelines. They both failed to make the necessary partogram entries as required by the guidelines. In other words, they did not at any stage show themselves as being diligent in note keeping or making crucial entries in the maternity case records. In fact Dr Linde herself gave an instruction for syntocinon infusion without as much as having seen the patient and assessed her condition and that of the foetus at 14:00, if the evidence of sister Mbada is to be accepted in that regard. Looking at their evidence and that of the plaintiff's expert witnesses, Dr Murray, professor van Toorn, professor Anthony and even that of Dr Koll in which inexplicably he sought to align himself with the defendant's factual witnesses even on this issue, none of this argument about syntocinon not having been administered is sensible let alone probable. On probabilities syntocinon was administered grossly negligently which resulted in the picture that is depicted in the evidence of both Dr Murray and professor Anthony about the pathological CTG tracings from about

16:00. The evidence, considered as a whole, clearly suggests that syntocinon infusion was prescribed negligently. It was also negligently administered with no doctor being present at the time of its administration. This even though Dr Linde was aware about the patient's severely delayed labour progress which caused her to prescribe syntocinon in the first place. After that, instead of remaining with the patient to personally monitor her, she disappeared from the scene inexplicably. A new doctor, Dr Philips came into the picture who had no previous background about the foetal condition. He would have to rely on whatever he would make of the incomplete and often contradictory hospital notes which it is common cause, were deficient in some material respect or whatever briefing he would get from the hapless sister Mbada.

[257] It turned out that he could not do vacuum extraction and had to call Dr Cilliers to come and assist. This was for a patient to whom time was of the essence and these medical personnel were rather lackadaisical about what was clearly a critical situation of a risky labour. This negligence which was the theme of their handling of the plaintiff's labour in this matter cannot be wished away. Even this idea that it made a difference that the contraction pattern of about as much as 8 or 9 contractions in 10 minutes was caused naturally by the mother uncontrollably pushing, a postulation of sister Mbada, Dr Cilliers and Dr Koll, as against syntocinon infusion is bewildering. The fact of the matter is that the plaintiff was encouraged to push by sister Mbada who was clearly out of her depth, with little or no proper consideration for the foetal well-being. She was left on her own devices in circumstances in which she was clearly neither qualified nor experienced enough to understand the gravity of the situation.

Conclusion.

[258] The result of all of this is that the plaintiff has, on a balance of probabilities, proved negligence and causation as the evidence clearly indicates. The approach by Dr Christianson to pursue the genetic testing hypothesis by ignoring crucial evidence misses one very important point. That is that the plaintiff is not required to establish as a scientific fact that it was not the genetic predisposition that led to the outcome and birth defects of A. She was required to prove on a balance of

probabilities that more than anything else it was the negligent conduct of the defendant's employees which caused the birth defects of A.

[259] In essence professor Christianson appeared to have set out to do a scientific exploration of the endless possibilities in the hope that he will achieve the scientific certainty that he pursued with zeal in his quest to answer the question relating to the unfortunate family history of the four children born of the plaintiff allegedly from the same partner. This is because all of them had some or other misfortune. This ignores the fact that the first child was born at home with no form of medical assistance whatsoever where nothing could have been done to prevent or even monitor foetal distress. Speculation about the first child possibly having some form of a genetic disorder has no factual basis. The same applies with the third child even though he was born in a hospital environment. The fact is that there is no factual basis for a conclusion that that child had any form of genetic malformation. The fourth child had epilepsy on the second day of life after what was described as very good apgar scores. The facts around her are not very clear and it would be improper if not dangerous to make conclusions about anything relating to her in circumstances in which in the fullness of time evidence might suggest something else. The second child A, the child in this case was a victim of many things. This is from incompetence of the nursing staff as clearly demonstrated by an inability to plot a partogram meaningfully and to make meaningful notes that are not only informative but also complement that which is plotted on the partogram. In fact, that partogram was largely ignored as was the foetal well-being of the child. The doctors themselves did not give an impression of medical practitioners that gave themselves time to ensure that foetal well-being was not compromised or that neonatally nothing was amiss.

[260] The fact that the date was 31 December 2015, a new year's eve did not help as there was skeleton staff. I need not repeat what was appears to have happened with regard to the evolution of the neurological situation of this child. What is clear is that from being born in a compromised state, her precarious situation was not appreciated which led to it not being adequately addressed. In fact, this baby appears to have been treated neonatally no differently from the intrapartum obstetric handling. Her delivery was handled even less than that of a normal as against risky

pregnancy. The failure to appreciate the deterioration that started from birth until the speech therapist came into the picture and looked into her seriously speaks volumes. As for Dr Mans, he appears to have been lulled into a false sense of confidence about the status of this baby by how Dr Cilliers and Dr Philips who were involved in the actual delivery would have handed her over as a normal healthy “vigorously crying baby” which is improbable and farfetched. It was a proverbial comedy of errors, to make light of a very serious matter. The genetic investigation cannot be used to blindly embark on endless scientific research of genetic possibilities at the expense of the facts of the shocking handling of this delivery which led to the outcome. I must say that professor Christianson’s attempt to brush everything aside in pursuit of this genetic testing theory whose end point he could not cogently articulate cannot be countenanced. I also had some difficulties with Dr van Rensburg’s evidence at least in one respect, still on the genetic testing issue. Dr Gericke incomprehensively and shockingly allowed notes or documents of two other patients he was working on to get mixed up with the records in this matter, if his explanation is anything to go by. It was beyond carelessness of him to allow that to happen.

[261] However, he explained his mistake which is still difficult to fully understand. He then conceded that he was accepting that A was GMFCS 1 and not 4 as he had previously testified. On the basis of the reference to A being GMFCS 4 by Dr Gericke, Dr van Rensburg insisted on the idea that A had a progressive as against static cerebral palsy. This ignored the evidence that in fact A’s cerebral palsy was static as a matter of fact. She clung on this progressive cerebral palsy idea to also push for genetic testing. I could not understand that even as I accept her discomfort which was well placed, with Dr Gericke’s process of examination of his patients and how he then goes about compiling his reports. However, none of this should be allowed to remove focus from the established facts and the logical process of legal reasoning as against scientific reasoning leading to legal as against scientific proof.

[262] In dealing with the factual witnesses’ evidence and the conflicting opinions of experts in this matter, I was guided by our case law some of which is referred above

including the recent case of *J.A. obo D.M.A*¹³. In that matter Van Zyl DJP had this to say:

“Conceptually there are several types of conflicts in expert evidence that may present itself in any given case. The first is a conflict with regard to the assumed facts. By reason of its very nature, expert opinion must have a factual basis. The facts upon which an expert’s opinion is based must be proved by admissible evidence. An expert opinion based entirely on inadmissible evidence is itself inadmissible. The facts may be established by asking the expert witness in examination-in-chief what those facts are. “An expert’s opinion represents his reasoned conclusion based on certain facts or data, which are either common cause, or established by his own evidence or that of some other competent witness. Except possibly where it is not controverted, an expert’s bold statement of his opinion is not of any real assistance.” How those facts are proven is determined by the principles of evidence and the usual methods used for judicial fact-finding and rational decision-making. Where the expert him or herself observed relevant facts, that evidence will be evidence of fact and admissible as such. Where the opinion seeks to take issue on the facts with the version of direct eyewitness evidence, credible eyewitness evidence conforms to the probabilities, will generally take preference to the opinion of an expert of what the facts are. In the final result, the decision of what the facts are must be founded on an assessment of the evidence as a whole and the probabilities as they appear therefrom.

The inferences drawn from the facts must be sound. The internal logic of the opinion must be consistent, and the reasoning adopted in arriving at the conclusion in question must accord with what the accepted standards of methodology are in the relevant discipline. The reasoning will be illogical or irrational and consequently unreliable, if (i) it is based on a misinterpretation of facts; (ii) it is speculative, or internally contradictory or inconsistent to be unreliable; (iii) if the opinion is based on a standard of conduct that is higher

¹³ *J.A obo D.M.A. v Member of Executive Council for Health, Eastern Cape* 2022 (3) SA 473 (ECB); [2022] 2 All SA 112 (ECB) paras 11 & 12.

or lower than what has been found to be the acceptable standard; (iv) if the methodology employed by the expert witness is flawed. What flows from this is that the mere fact that an expert opinion is unchallenged does not necessarily mean that it must be accepted. However, if that evidence is based on sound grounds and is supported by the facts, there exists no reason not to accept it.”

[263] In the final analysis the plaintiff has, on a preponderance of probabilities, proved that the birth defects of A were caused by the negligence of the defendant’s employees. Professor Andronikou’s MRI scan report made the following finding concerning the injury pattern of A:

“Bilateral, symmetrical involvement of the para-sagittal, peri-Rolandic and peri-trigonal deep/periventricular white matter regions of the brain in combination with the involvement of the thalami and caudate nuclei **may** be due to a hypoxic ischemic injury of a combined acute-profound and partial-prolonged nature. However, the pattern of injury in this patient can also be seen with toxic metabolic and post infectious causes. Possible metabolic conditions that can have this appearance include Canavan disease, crabbie disease and Wilsons disease. The patient requires evaluation by a paediatric neurologist and may have to undergo testing for metabolic disorders to distinguish these potential causes.”

[264] I am of the view that to a fairly reasonable extent, all the other potential causes of the injury pattern in A have been reasonably excluded. Furthermore, I take the view that despite professor Andronikou having, in his report, used the word “**may**”, his primary finding was that the birth defects of A were due to a hypoxic ischemic injury of a combined acute-profound and partial prolonged nature.

[265] The inadequate monitoring has, in this case, resulted in the foetal distress being missed which was substandard care. The lack of proper notes that would have easily enabled the various medical staff involved in the management of the delivery of A to appreciate the unfavourable environment the foetus was in largely also contributed to the foetal distress not being appreciated as it should have.

Perhaps even at the risk of repetition, it is not without significance that the plaintiff was already in the active phase of labour as early as 06:50. She remained at 5cm of dilation throughout until about 12:00 when she progressed to 7cm some five hours later. That was a severely delayed labour progress. There is no evidence of any thorough going investigation of the severe delay at any stage and an informed decision being made one way or the other about any necessary intervention. In fact the evidence is that despite the plaintiff being a risky labour, was only seen by a doctor at about 15:00 for the very first time. That was some six hours since she was seen by nurses at 09:00 who had in any event negligently wrongly plotted her in the latent phase of the partogram. There were numerous instances of substandard care which punctuated every step of A's delivery process with nobody making any serious effort, beyond a seemingly occasional glance at the CTG when it was running. The hospital staff's notes were so inadequate that they could easily confuse even the author thereof not to mention the doctors who seemed to have placed heavy reliance on the clearly underqualified and clearly inexperienced sister Mbada, if her notes are anything to go by. In fact she admitted that when A was born she had not yet done midwifery yet which she only finalised in about 2018. It is fair to say when she testified in this matter she was more experienced and qualified than in 2015 when she was allowed to single handedly monitor the delivery process of A and manage the plaintiff's labour when she was clearly not qualified to do so.

[266] The unguided syntocinon infusion simply made worse what was already a critical and potentially dangerous situation. It gets worse in that even if sister Mbada's and Dr Koll's evidence were to be accepted that the patient was pushing uncontrollably between 16:00 and the delivery, she did nothing to ensure that the pushing which she encouraged the plaintiff to do was being carefully monitored to ensure that the foetal well-being was not thereby compromised. It seems to me that from sister Mbada to Dr Linde who prescribed the syntocinon and disappeared thereafter, and indeed to Dr Cilliers, and Dr Mans, everybody seemed to rely on some hope for the best and not a well informed decision based on a careful observation of the facts and the critical tests like sugar test results being done. To make the point clear that there was lack of appreciation of the fact that the delivery of A was in troubled waters, an admittedly inexperienced Dr Philips was the one who had to deal with the actual ventouse delivery. He had to call a senior colleague, Dr

Cilliers for assistance. All of that added to the delay that was already severe and dangerous for the foetal well-being which was never, at any stage critically assessed. Dr Cilliers used a risky pregnancy to train a young and inexperienced doctor just to upskill him when she appears not have given herself time to critically evaluate the hospital notes and the partogram and make the required entries.

[267] In all these circumstances it is clear that A suffered intrapartum asphyxia which directly led her to suffer injuries that caused her the damage due to the failure of the hospital staff to adequately monitor her from the very beginning at 09:00 all the way to the delivery of the baby and thereafter until day 4 of her life. The attempt by to try to make a clear distinction between poor note keeping and substandard care is difficult to understand as they even ignored the obvious results of that poor note keeping. There was also substandard care even at the neonatal stage which manifested itself in a proper analysis of the neurological symptomatic lack of suck reflex, poor grasp and latch reflex and lethargy not being investigated. The crying was simply and lazily attributed to hunger by Dr Mans and a conclusion was made without properly analysing the situation. It was then concluded that the only reason the baby was not breastfeeding was the mother's inability to breastfeed hence the recommendation for breastfeeding counselling without any attempt at excluding encephalopathy. That too was substandard care as it contributed to the failure to place the baby under a higher level of care which, in any event, should have been done following a resuscitation and the obviously less than normal level of consciousness of the baby. In the result the plaintiff must succeed in her claim for damages.

The result.

[268] Therefore, the following order shall issue:

1. The defendant is held liable to compensate the plaintiff in her personal and representative capacities as the mother and natural guardian of A for the damages suffered as a result of the defendant's breaches of the agreement and/or legal duty to the plaintiff and A such amount as may be proved or agreed upon.

2. The defendant shall pay the plaintiff's costs of suit including the costs occasioned by the employment of two counsel as well as all costs of the plaintiff's expert witnesses.

M.S. JOLWANA
JUDGE OF THE HIGH COURT

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