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| Reportable: NO  Circulate to Judges: NO  Circulate to Magistrates: NO  Circulate to Regional Magistrates: NO |

**IN THE HIGH COURT OF SOUTH AFRICA**

**NORTH WEST PROVINCIAL DIVISION, MAHIKENG**

**Case No.: 726/2016**

**In the matter between:**

**BKM obo NB Plaintiff**

**and**

**MEC FOR HEALTH, NORTH WEST PROVINCE**

**GOVERNMENT Defendant**

**JUDGEMENT**

**O Y DIBETSO-BODIBE AJ**

**INTRODUCTION**

[1] The Plaintiff is Ms BKM who claims damages in her personal and representative capacity on behalf of her minor child, NB, against the MEC for Health. The claim emanated from the medical negligence alleged to have been committed by the employees of the Defendant during the scope and course of their work as health professionals as a result of which NB suffered Cerebral Palsy.

**MERITS AND QUANTUM SEPARATED**

[2] At the commencement of trial, as agreed between the Parties during a pre-trial conference and by order of this Court in terms of Rule 33(4) of the Uniform Rules of Court, the merits (liability) were separated from quantum. The matter only proceeded on the issue of liability, which this court is called upon to adjudicate.

**THE ISSUES**

[3] The issues for determination are whether the Defendant’s employees (the nursing staff and/or medical doctors) who attended to BKM during labour were negligent in the management and monitoring of the Plaintiff’s labour and subsequent delivery of NB on any of the grounds pleaded on behalf of the Plaintiff in the amended Particulars of Claim dated 05 July 2021. The issue further is whether or not such negligence caused NB to develop Cerebral Palsy as a consequence of a hypoxic-ischaemic event.

[4] For the Plaintiff to succeed and hold the Defendant liable for damages, she must prove on a balance of probabilities, causal connection between the Defendant’s alleged negligent acts or omission relied upon and the harm suffered.

**COMMON CAUSE FACTS**

[5] The Plaintiff, a 26-year-old female presented to the De La Rey Hospital (the Hospital) at 09h05 on 15 July 2013.

[6] On examination, the cervix was 5cm dilated, thin and well applied[[1]](#footnote-1) with intact membranes. No maternal-foetal or labour risk factors were identified. The assessment was made of the active phase of labour and the plan was made to monitor the maternal and foetal condition and to allow labour to progress.

[7] The Partogram[[2]](#footnote-2) was commenced and well completed at this time, other than the fact that the times do not fully correspond to the blocks in the Partogram as each block should represent one hour whereas between 09h05 and 11h05 there are three blocks instead of two.

[8] A Cardiotocograph (CTG)[[3]](#footnote-3) was not done as the CTG machine was out of order.

[9] The gestation[[4]](#footnote-4) was ‘term’ meaning that labour occurred at the right time when the neonatal risks would be expected to be low. There were no concerning features regarding presentation in labour.

[10] The foetal heart was normal on arrival meaning that the foetus entered labour in a satisfactory condition.

[11] The Plaintiff delivered a female baby by normal vaginal delivery (NVD) at 14h09 on 15 July 2013.

[12] The baby was admitted to hospital until 02 August 2013.

[13] NB developed Hypoxic Ischaemic Encephalopathy otherwise known as Neonatal Encephalopathy[[5]](#footnote-5).

[14] As a result of the brain injury NB suffers from spastic quadriplegic cerebral palsy[[6]](#footnote-6), mental retardation and developmental delay.

[15] Two experts, Prof Smith (Neonatologist / Paediatrician) and Dr Murray (Obstetrician / Gynaecologist) adduced evidence on behalf of the Plaintiff.

[16] The Defendant closed its case without calling any witnesses.

[17] The joint minutes of Dr Alheit and Dr Mweli (Radiologists) and of Prof Smith and Dr Ballot (Neonatologists / Paediatricians) were submitted by consent.

[18] The expert summaries were also submitted by consent including the medical hospital records in respect of the labour process of NB

**THE EVIDENCE OF DR MURRAY (THE OBSTETRICIAN / GYNAECOLOGIST) ON BEHALF OF THE PLAINTIFF**

[19] The evidence of Dr Murray was briefly that the Plaintiff presented at the Hospital at 09h05 on 15 July 2013. According to the Clinical Notes she was noted as being a 26-year-old woman who came in self-referred and brought by Emergency Medical Services (“EMS”) complaining of labour pains that started at 03h00. She gave no history of raptured membranes.

[20] On vaginal examination, the cervix was 5cm dilated, thin and well applied with intact membranes. Station[[7]](#footnote-7) and Caput[[8]](#footnote-8) or Moulding[[9]](#footnote-9) was present. The foetal heart rate[[10]](#footnote-10) (FHR) was 135 to 145 beats per minute (bpm). No maternal-foetal or labour risk factors were identified. The assessment was made of the active phase of labour and the plan was made to monitor the maternal and foetal condition and to allow labour to progress.

[21] Pregnancy blood tests were performed and were all within normal limits in that the Plaintiff tested negative for syphilis and HIV, was blood group O+ and her haemoglobin[[11]](#footnote-11) was 12.5g/dL.

[22] The Plaintiff presented in the active phase of the first stage of labour[[12]](#footnote-12) at a term gestation meaning labour occurred at the right time when neonatal risks would be expected to be low. No concerning features regarding presentation in labour were noted.

[23] According to the Guidelines for Maternity Care in South Africa (2007) (the Guidelines), during active phase of labour, the following should be done by the midwives and/or medical doctors:

[23.1] The maternal blood pressure and heart rate should be monitored hourly, the temperature should be monitored 4-hourly and the urine volume should be measured and tested 2-hourly.

[23.2] The foetal heart rate should be monitored half-hourly, listening before, during and after a contraction.

[23.3] The colour and odour of the liquor or amniotic fluid[[13]](#footnote-13) should be observed every two hours to check if membranes have raptured.

[23.4] The frequency and strength of contractions should be monitored hourly.

[23.5] The cervical dilation, level of the presenting part and presence of caput and moulding must be assessed two-hourly

[24] Dr Murray testified that given the fact that the Plaintiff was a low-risk patient, monitoring of the CTG was not necessary and that intermittent auscultation[[14]](#footnote-14) was appropriate. The foetal heart was normal on arrival and the documented range implies that the foetal heart was not auscultated as a once-off, but over a period of some time, meaning the foetus entered labour in a satisfactory condition.

[25] The next assessment was made at 11h05 on 15 July 2013. Although “Assessment No. 2” is untimed, it correlates with the timed entry at 11h05 made on the Partogram. Progress of labour was noted as being good. Moderate contractions were palpated[[15]](#footnote-15). The cervix was 8cm dilated, thin and well applied, and the presenting part was at the level of the ischial spines (station 0). The foetal heart was 158 beats per minute (BPM). Further information from the Partogram indicate that membranes were still intact, and the head was 0/5 palpable above the pubic symphysis[[16]](#footnote-16). Three strong contractions in 10 minutes were present. The plan was to “monitor maternal and foetal condition and allow labour to progress”.

[26] Dr Murray formed an opinion that there was failure on the part of the midwives to auscultate the foetal heart every 30 minutes as required by the Guidelines, in that the foetal heart had not been intermittently checked for the preceding two hours. She opined that this failure to monitor the foetal heart as per the Guidelines constitutes substandard monitoring of the foetal condition during the course of labour and means that had the foetal condition changed in response to the potential hypoxic stress associated with uterine contractions[[17]](#footnote-17), then this change would not have been evident to the midwives and no steps would therefore have been taken to either improve the foetal condition by performing intra-uterine resuscitation[[18]](#footnote-18) or expediting delivery if the foetal condition was seen to be poor and delivery was not imminent.

[27] Regarding the presence of maternal pyrexia (raised body temperature or fever) at this stage of “No.2 Assessment”, Dr Murray stated that this was concerning especially that the Plaintiff was only administered Amoxil and Flaggyll (antibiotics) without first checking why the Plaintiff’s body temperature presented at 38.4˚C. To this effect she stated that because of the possible injurious effects of maternal fever, careful monitoring of the foetus should take place by way of CTG, where possible. If this is not possible, then the foetal condition should be meticulously monitored for signs of foetal infection.

[28] According to the Partogram, the Plaintiff was next to reviewed at 13h05 on 15 July 2013. Three strong contractions in 10 minutes were present. The Plaintiff’s cervix was fully dilated, and the presenting part was at station+1[[19]](#footnote-19) with 1+caput[[20]](#footnote-20) and 1+moulding[[21]](#footnote-21). The liquor[[22]](#footnote-22) was not as being meconium stained[[23]](#footnote-23). Nothing regarding the foetal heart was noted on the corresponding part of the Partogram for the foetal condition.

[29] The Partogram shows that the Plaintiff had continued to make good progress during labour though no reference to the foetal heart rate was made for the preceding two hours, nor is there any indication that the foetus was intermittently assessed from 11h05 up until delivery at 14h09, some three hours later.

[30] Her opinion was, therefore, that in view of the inadequate foetal monitoring and failure to record the foetal response (the foetal heart rate and pattern) to the “stress” of uterine contractions, the Partogram is a substandard reflection of the foetal condition during advanced labour. This means that the foetal condition during advanced labour was unknown, which increased the risk that the foetus would come to harm and that this would go unnoticed by the midwives.

[31] This is so, she stated, because labour poses hypoxic stress to any foetus. During contractions, less oxygen rich maternal blood is available in the intervillous space[[24]](#footnote-24) for delivery of the foetus. This means that during each contraction the foetus suffers brief hypoxic bouts. These are normally well tolerated by healthy foetuses and do not lead to progressive change in the foetal condition in the form of hypoxaemia[[25]](#footnote-25) or acidosis[[26]](#footnote-26). However, in cases where the foetus has poor reserves with which to cope with, the stressors of labour (as may be the case, for example, when a foetus is growth restricted or the pregnancy has progressed post term), or when the contractions are excessively strong or frequent, or the labour is prolonged, the foetus may be exposed to more hypoxic stress than she can deal with without incurring progressive oxygen debt. Although a healthy foetus will undergo various cardiovascular adaptations aimed at directing blood and oxygen delivery to vital organs such as the brain, if the oxygen debt is ongoing, eventually even a previously healthy foetus will run out of reserves and will progressively deteriorate and decompensate, becoming profoundly hypoxic.

[32] Failure, therefore, to monitor the foetus adequately means that the foetal condition would remain unknown, any change in the foetal condition could not be diagnosed, and no steps would be taken aimed at improving foetal oxygenation and expediting delivery.

[33] Dr Murray stated that although the presence of meconium in the amniotic fluid can be a normal finding indicative of gut maturity, it has also been attributed to stress secondary to hypoxia[[27]](#footnote-27). Thick (usually fresh) meconium has been associated with poorer neonatal outcome. Passage of meconium in the absence of other signs of foetal distress is not a sign of hypoxia, but the appearance of abnormal heart tracings in the presence of thick meconium is a strong indicator of foetal distress, above and beyond that seen with abnormal foetal heart rate patterns alone. In other words, the presence of thick meconium makes careful monitoring of the foetal condition by way of CTG mandatory to evaluate the foetal condition, and any change in the foetal condition should be timeously diagnosed and action taken. Her conclusion was that the fact that thick meconium was present at the delivery of NB increases the likelihood that foetal heart rate abnormalities were also present.

[34] The Clinical Notes written after birth revealed that the cord was noted as being “around the arm and the neck x 2 and tightly applied” as against what is stated in the Summary Labour-Form that the cord was “around the neck twice and tightly applied” and there was a compound presentation[[28]](#footnote-28). According to her the majority of compound presentation consist of a foetal hand or arm presenting with the head. A compound presentation may be diagnosed during labour by feeling a foot or hand next to the foetal head during labour or may be apparent at the time of birth. Generally, if labour is progressing well, then labour is allowed to progress, and no specific management is necessary. If there is second stage rest and a compound presentation is diagnosed, one would usually gently push the hand or foot up to complete delivery. Her view is that it is inconceivable that a hand or foot pulled up near the head could bring a loop of cord with it and cause some entanglement, as is described in the records.

[35] Tight nuchal cord[[29]](#footnote-29) may cause the foetus to become acutely hypoxic during delivery itself. However, this is usually very short-lived and self limiting, and most foetuses would tolerate this episode of hypoxia well and respond well to resuscitation on delivery. However, a foetus who may have been in a compromised, or already decompensating state, before the onset of the second stage of labour, may have no further reserve with which to “buffer” the sudden hypoxia associated with delivery in the face of tied cord loops and may become profoundly hypoxemic and acidotic.

[36] Dr Murray formed an opinion that in the setting of an umbilical cord that is wound multiple times around the neck and arm, as reported in the present matter, it is unlikely that there would be no signs at all of cord compression[[30]](#footnote-30) during the labour before delivery. In other words, if it is accepted that the tight cord was problematic for the foetus, there would probably have been warning signs of this during the course of labour and intervention could have resulted in expedited delivery, in the form of a caesarean section, thereby not exposing the foetus to further risks of hypoxia during the second stage of labour.

[37] The second stage of labour (the period from full cervical dilation [10cm] to delivery) was 64 minutes in length, which is within the normal range. However, there is again no reference to the foetal condition being monitored in contravention of the Guidelines that the foetal heart should be auscultated every 5 minutes or with every second contraction in the second stage of labour. This is because the second stage of labour is the most stressful time for any foetus with contractions being the strongest and occurring most frequently. Dr Murray concluded that monitoring during the second stage is therefore of utmost importance as the foetus who is decompensating will need rapid and expedited delivery to prevent further worsening of hypoxaemia.

[38] According to the “Assessment of the Newborn-form”, the Plaintiff delivered a female baby by normal vaginal delivery (NVD) at 14h09 on 15 July 2013. The baby’s Apgar scores[[31]](#footnote-31) were recorded as 5/10 and 9/10 at 1 and 5 minutes of life respectively. However according to the Hospital Admission form, the Apgar scores were recorded as 0/10 and 3/10 at 1 and 5 minutes of life respectively. The baby was noted to have needed resuscitation in the form of “oxygen”, “mask” and “intubation”.

[39] Dr Murray stated that the discrepancy between the Apgar scores were concerning and that the doctor’s rewritten Apgar scores were considered as being more accurate as they are in keeping with the baby born in a critical condition and requiring resuscitation in the form of intubation as is recorded. Further that the Hospital form which recorded the Apgar score as 0/10 and 3/10 shows that the Apgar scores were very low meaning that the baby was born dead with no signs of life, a sign of how dire the in-utero-environment was.

**THE EVIDENCE OF PROF SMITH (PAEDIATRICIAN / NEONATOLOGIST) ON BEHALF OF THE PLAINTIFF**

[40] Prof Smith quoted the following excerpts from the clinical records upon which he based his opinion regarding the monitoring of the maternal and foetal condition and whether the monitoring under the circumstances passed the muster of the requirements as outlined in the Maternity Guidelines.

[41] The “Clinical Notes” from the birth file revealed that the gestation was 9 months [9/12] by dates (i.e according to the date of the Plaintiff’s last menstrual period) and 36 (36/40) by palpation, the heart rate was ticked off as normal. The station was -1, and the Plaintiff was diagnosed as being in the active phase of labour, and labour was allowed to progress.

[42] “Assessment No. 1-Form” from the birth file recorded at 09h05 revealed that the membranes were intact, moderate contractions were palpable, the foetal heart rate [FHR] was recorded as 133 to 145 beats per minute [BPM], and it was noted that the CTG was out of order.

[43] “Assessment No. 2-Form” from the birth file revealed that labour was good, there was moderate contractions, the station was “0” (i.e, it was at the level of the ischial spines, the cervix was 8cm dilated, the foetal heart rate was 158 beats per minute [BPM]), the Plaintiff was assessed to be in the active phase of labour and the plan was to monitor the maternal and the foetal condition and to allow labour to progress.

[44] “Summary of Labour Form” from the birth file revealed that the Plaintiff’s cervix was fully dilated at (10cm) at 13h05, the time of delivery was 14h09, the birth was normal vertex delivery or normal vaginal delivery [NVD], it was noted that there was a “cord around the neck x 2”. Tightly applied and that there was meconium liquor grade III with compound presentation, resuscitation was done and that it included suctioning, O² chest compression.

[45] “Clinical Notes” from the birth file (no time specified) revealed that a 26-year-old G2PO delivered an alive female infant weighing 3.1kg at 14h09 with Apgar 5/10 – 9/10. Resuscitation[[32]](#footnote-32) immediately commenced by mechanical suctioning, O² by bag, cardiac massage and chest compressions, and the ward doctor notified and responded quickly, and that initially the baby was flat, bluish in colour, but later responded to the resuscitation. During delivery it was a compound presentation with cord around the neck x 2 and tightly applied, and there was also meconium grade III.

[46] “Assessment of the Newborn Form” from the birth file recorded the Apgar scores at 1 minute – 5/10 (scores awarded for heart rate [1], respiration / breathing [1], muscle tone [1], response to stimulation [1] and colour [1]) and at 5 minutes – 9/10.

[47] Prof Smith dealt with the discrepancies in the Apgar scores and followed a similar conclusion as Dr Murray that the 9/10 score was not physiologically plausible if one considers the baby’s condition at birth as well as the level of resuscitation that was required. The said Apgar scores in the “Newborn Form” differs from those in the “Admission Form Neonatal / KMC” written by Dr De Wet where the Apgar score was recorded as 0/10 at 1 minute of life and as 3/10 at 5 minutes of life and the 10 minute Apgar score was not recorded.

[48] “First Examination of Neonate Form” from the birth file revealed that the baby was blue, chest movement were shallow, the respiratory rate was slow, the moro[[33]](#footnote-33) and grasp reflexes were absent and the baby did not cry.

[49] “Discharge Summary: Neonates / KMC Form” revealed that the diagnosis of the baby was Low Apgar + hypoxic ischaemic encephalopathy. The baby was discharged on 02 August 2013.

[50] The Magnetic Resonance Imaging (MRI)[[34]](#footnote-34) of the child’s brain was performed on 01 October 2015 and reported by Dr Alheit on 02 June 2021.

[51] Prof Smith agrees with the conclusion that the MRI study is diagnostic of a Perirolandic Paracentral lobule, Basal Ganglia and thalamus (PPBGT) / central Hypoxic Ischaemic injury of the brain, as seen from 35-36 weeks’ gestation onwards, now visualized in the chronic stage of evolution on the MIR scan performed at the age of 2 years and 3 months.

[52] Based on the said medical records Prof Smith formed an opinion that when the Plaintiff was admitted to the Hospital in active labour at 09h05, the foetal condition was probably reassuring and labour was allowed to progress.

[53] The fact that the CTG was out of order puts an onerous burden on the midwives to manually auscultate the foetal heart and/or to use a Doppler device to monitor the foetal condition in accordance with the Guidelines which requires that during active labour (i.e from when the cervix is 4cm or more dilated) there should have been monitoring of the maternal condition – Blood Pressure and heart rate should be monitored hourly, this was not done. The foetal heart rate should be monitored half-hourly, before, during and after contractions using a hand-held Doppler instrument, this was not done and only checked on 22% of the required occasions. The frequency and strength of uterine contractions should be monitored hourly, this was inadequately monitored as it was done only 44% of the required occasions. The observations of the first stage of labour should continue, this was not done. Listen to the foetal heart rate between every second contraction, this was never checked or recorded.

[54] Prof Smith formed an opinion that based on the said standard of care, monitoring of the foetal condition and the foetal response to the stress of uterine contractions was clearly inadequate and substandard throughout the active phase of labour as well as during the most dangerous period of labour, i.e, the second stage of labour. No foetal distress would have been detected with the inadequate foetal monitoring as a result of which a decision to expedite labour was not a possibility or even considered.

[55] Probable foetal distress developed but due to inadequate monitoring or no monitoring at all, foetal distress was completely overlooked during the late active phase and second stage of labour.

[56] Considering that there was the presence of multiple nuchal cord entanglement, there probably was detectable foetal heart rate abnormalities and foetal distress during labour (i.e during the intrapartum period).

[57] The baby was born in a severely compromised condition as described in the clinical notes. She was initially flat and bluish in colour, the Apgar scores was contradictorily recorded as 9/10 and 5/10 whilst the clinical events (the degree and the level of resuscitation administered) are more in keeping with a score of 0/10 at 1 minute, and the extent of the delivery room resuscitation immediately after birth which included cardiac massage and tracheal intubation and manual ventilation (“Bagged”).

[58] Prof Smith formed an opinion that the said foetal condition indicates that the baby was born lifeless or in a state of secondary apnoea (i.e, with a bradycardia[[35]](#footnote-35) and the absence of breathing). There was grade III (thick) meconium-stained liquor at birth and this is left for Obstetricians to deal with.

[59] Prof Smith opined that the bradycardia must have occurred before birth and was probably present for 20 or more minutes in order to have resulted in permanent brain injury. Relying on Baxter he stated that term born infants can withstand 20 minutes of foetal bradycardia without acute or chronic brain damage and, therefore, that durations (in terms of timing brain injury) in humans are not the same as in animal models. Further that a persistent bradycardia is likely to have been caused by a preceding period of hypoxia and that tissue eschaemia due to reduced cardiac output probably coincides with bradycardia. After the onset of a persistent bradycardia with or without sentinel events in 125 term born infants, normal outcomes were reported in 46 out of 60 (77%) of those delivered before 20 minutes. Normal outcomes were reported in two after more than 25 minutes. Abnormal outcomes were reported in two by 10 minutes, and in 55 out of 65 (85%) of those delivered after 20 minutes. Survival with impairment was possible after more than 45 minutes[[36]](#footnote-36).

[60] Prof Smith concluded that detectable foetal distress was more likely present than not, and that it would have been detected, had proper monitoring occurred.

[61] He further opined that since the baby was born in a lifeless condition and since she developed an early onset moderate or moderate-severe degree of neonatal encephalopathy, the probability that encephalopathy occurs, given that foetal distress is present, is higher than the probability that neonatal encephalopathy occurs in the absence of foetal distress.

[62] There was no recording of a sentinel event (abruption placenta[[37]](#footnote-37), placenta praevia[[38]](#footnote-38), cord prolapse[[39]](#footnote-39), uterine rapture[[40]](#footnote-40) or tear or maternal collapse) having occurred, which could explain the birth of a compromised baby in the present matter. In the absence of such a catastrophic sudden event, suboptimal/substandard intrapartum obstetric practice emerges as the likely (probable) causal factor which leads to the development of undiagnosed hypoxic ischaemia which insulted and injured the foetal brain, explaining the compromised condition at birth, as well as the subsequent outcome of permanent neurological disability.

[63] Prof Smith’s view is that even if a tight nuchal cord is hypothesized as having caused significant foetal compromise or is considered as a ‘sentinel event’, which it is not according to ACOG (reaffirmed 2019), the likelihood of a tight nuchal cord being associated with concurrent detectable abnormal foetal heart rate and pattern abnormalities is high, and is more probable than it being silent.

[64] Prof Smith relied on Yamada[[41]](#footnote-41) that “healthy foetuses according to foetal heart rate (FHR) tracing at the onset of labour pains can develop neonatal encephalopathy (NE) and subsequently develop Cerebral Palsy (CP). Well known causative factors for CP due to hypoxic conditions include placental abruption, uterine rupture, cord prolapse, feto-maternal hemorrhage[[42]](#footnote-42), or placenta praevia. Some infants however, develop NE leading to CP (NE-CP) in the absence of such risk factors. Given that suboptimal intrapartum care is commonly seen with NE, especially in neonates with metabolic acidemia in Sweden, and given that NE proceeds to CP in some cases suboptimal intrapartum care may be a risk factor for NE-CP. The authors reported a significantly high incidence of suboptimal care[[43]](#footnote-43) (57%) in their cases with NE-CP compared to controls.

[65] The conclusion by Yamada is that suboptimal care is prevalent worldwide among neonates with NE and is a major intrapartum risk factor for NE and/or NE-CP after excluding well-known risk factors.

[66] Prof Smith further relied on De Knijf and Pattison[[44]](#footnote-44) a study based on South African research that confirms the role of suboptimal intrapartum obstetric care as likely (probable) causal factor explaining the development of neonatal encephalopathy, especially hypoxic ischaemic encephalopathy (HIE). The authors studies revealed avoidable factors contributing to neonatal morbidity and mortality due to “birth asphyxia” and to ascertain if hypoxic ischaemic encephalopathy (HIE) alone could act as a good marker for avoidable factors for birth asphyxia were refusal of medical treatment, inadequate facilities, no detection of, or reaction to foetal distress, and incorrect management of the second stage of labour.

[67] Based on the said studies, Prof Smith concludes that in the present matter, the CTG device of the unit was out of order in consequence whereof no electronic foetal monitoring occurred. The midwives and doctors of the unit were therefore put on alert in that they had to rely either on manual auscultation and/or Doppler (handheld sonar) devices to monitor the foetus during labour. There is no evidence to show that adequate foetal monitoring occurred, using any method.

[68] The second stage of labour 13h00 / 13h05 and 14h09 was probably unassisted and/or unattended since no foetal monitoring occurred during that period. This omission would be in keeping with gross substandard obstetric intrapartum care and management.

[69] Relying on the study of Clapp JF, et al[[45]](#footnote-45), Prof Smith stated that nuchal cords occur with increased frequency as gestation increase and appear to be normal part of intra-uterine life that is rarely associated with perinatal[[46]](#footnote-46) morbidity and mortality. A NC does not usually produce clinical evidence of acute foetal compromise before labour.

[70] The loops may be loose, which can be easily slipped over the foetal head, or tight requiring clamping before untwining during delivery.

[71] A cord around the neck may cause asphyxia through obstruction before delivery. However, most patients are likely to have uneventful labour as cord compression is transient and most foetuses are able to compensate for reducing umbilical blood flow and deliver uneventfully.

[72] The conclusion by the authors[[47]](#footnote-47) on the study of entanglement of umbilical cord around the foetal neck and adverse perinatal outcome is that entanglement of umbilical cord around the foetal neck or NC is not related with adverse perinatal outcomes such as acidosis and low Apgar scores. Prof Smith’s conclusion based on this study is that when faced with a nuchal cord, there is a higher likelihood of foetal distress related to the number of coils and therefore delivery by caesarean section, but without adverse outcomes, compared to labour in the absence of a nuchal cord, becomes necessary. This implies that with adequate foetal monitoring, foetal distress can be timeously detected and delivery can be expedited, avoiding adverse neonatal outcome and, therefore, probably the development of cerebral palsy.

[73] Badawi[[48]](#footnote-48) evaluated risk factors for newborn encephalopathy, present in the intrapartum period and adjusted for factors before birth and antepartum, and found no association between the presence of a nuchal cord and the development of a neonatal encephalopathy. Based on this study, Prof Smith concluded that since neonatal encephalopathy is regarded as a doorway to the likelihood of development of cerebral palsy, linking intrapartum asphyxia to cerebral palsy, one may exclude nuchal cord as an independent causal factor.

[74] The doorway through which an intrapartum hypoxic ischaemic insult leads to permanent brain injury (cerebral palsy) is through the early development of a neonatal encephalopathy and Prof Smith concludes that the available evidence confirms that this occurred in the present case.

[75] The overall conclusion of Prof Smith in this case is that the final outcome of cerebral palsy is directly ascribable to substandard / suboptimal intrapartum obstetric practice, and a complete failure to properly and adequately monitor a patient in labour, thereby missing both the diagnosis or probable foetal distress and the window of opportunity to expedite delivery.

**JOINT MINUTES OF PROF SMITH (PAEDIATRICIAN / NEONATOLOGIST) FOR THE PLAINTIFF AND DR BALLOT (PAEDIATRICIAN / NEONATOLOGIST) FOR THE DEFENDANT**

[76] The gestation was “term”

[77] The 26-year-old female presented to the De La Rey Hospital at 09h05 on 15 July 2013. She experienced lower abdominal pains. Her cervix was 5cm dilated and after her assessment labour was allowed to progress.

[78] The implication is that the foetal condition must therefore have been reassuring upon admission.

[79] The last foetal heart rate recording was at 11h05 on 15 July 2013, three (03) hours before the birth of NB at 14h09.

[80] There is no evidence that the foetus was monitored at all during the second stage of labour (13h05 – 14h09)

[81] The baby, NB, was born as “NVD” (normal vertex / vaginal delivery) at 14h09 on 15 July 2013.

[82] Her growth parameters rule out long standing intra-uterine hypoxia related to placental insufficiency.

[83] The placental fundings rule out a placental abruption as a possible cause for the asphyxia.

[84] It was noted that there was a “cord around the neck x II tightly applied. Meconium-stained liquor grade III. Compound presentation” The issue of “Compound presentation was referred to the Obstetric experts for their opinion.

[85] Excerpts from the “Clinical notes” (printed page 23 of the birth file) that were recorded at unspecified time on 15 July 2013 regarding the birth of the baby revealed the following:

[85.1] “A 26-year-old G2 PO delivered an alive female infant weighing 3.1kg at 14h09 with Apgar 3/10 5/10 – 9/10. Resuscitation immediately commenced by mechanical suctioning, O² by bag, cardiac massage and chest compressions. Ward doctor notified and responded quickly.

[85.2] Initially the baby was flat bluish in colour, but later responded to the resuscitation. During delivery it was a compound presentation with cord around the arm and neck x 2 and tightly applied. There was also meconium grade III.”

[86] The Apgar scores as recorded on the “Assessment of the Newborn” – form (5/10 and 9/10 at 1-and-5-minites of life, respectively) differ from what the doctor (Dr E de Wet) recorded on the document headed “Thusong/Gen De La Ray Hospital Complex – Admission Form: Neonatal/KMC” where the Apgar scores were recorded at 0/10 at 1 minute of life and as 3/10 at 5 minutes of life. The 10 minute Apgar score was not recorded.

[87] The Apgar scores recorded by Dr de Wet were more in keeping with the clinical descriptions of a baby who was born flat and blue, and who required extensive resuscitation as noted above, than the additional recorded scores of 3/10, 5/10 and 910 (as per paragraph 9.10.1 above)

[88] The above indicates that the baby was either born lifeless or in a state of secondary aponoea (i.e., with a bradycardia and the absence of breathing).

[89] NB’s condition at birth therefore satisfied the very basic, but imprecise, **WHO** (World Health Organisation) definition of ‘birth asphyxia’ i.e., when a baby, “Fails to initiate or maintain regular breathing at birth[[49]](#footnote-49).

[90] NB developed an early onset moderate-severe neonatal encephalopathy (NE), alternatively hypoxic ischaemic encephalopathy (HIE).

[91] The diagnosis of an early onset NE confirmed the essential component (“doorway”) linking intrapartum asphyxia to subsequent cerebral palsy in a causal pathway (MosterD, MarkestadT. The potential use of Apgar Score and neonatal encephalopathy in registry-based studies. Norsk Epidemiology 2007, 17(2): 181-184, Neonatal Encephalopathy and Neurologic Outcome, Second Edition Ostel Gyneco1 2014; 123(4); 896-901)

[92] NB was not offered therapeutic hypothermia.

[93] The MRI (Magnetic Resonance Image) of NB’s brain revealed a basal ganglia-thalamic (BGT) hypoxic ischaemic pattern injury. The following was reported:

[93.1] Dr T Mweli (Radiologist for the Defendant) – Bilateral near symmetrical hyperintensities in the thalami, basal ganglia (posterior putamen), parahypocampal, and perirolandic cortex. The topographic locations are consistent with the chronic stage of previous acute profound “acute near severe asphyxia”. Dr Mweli also suggested as possible causes, “Hypoxic ischaemic encephalopathy +/- sentinel event”.

[93.2] Dr B Alheit (Radiologist for the Plaintiff) – the MRI study is diagnostic of a PPBGT (Periroloandic, Paracentral lobule, Basil Ganglia and Thalamus) / central hypoxic ischaemic injury of the brain, as seen from 35-36 weeks’ gestation onwards, non visualized in the chronic stage of evolution.

[94] The respective radiologists described the exact similar brain injury pattern, using different descriptive words.

[95] The type of structural brain damage described in NB is also described as:

[95.1] The BGT Hypoxic ischaemic pattern injury – Dr Vries L, Groenendaal F[[50]](#footnote-50). These authors described the pattern of injury as most often seen following an acute sentinel event, for instance a ruptured uterus, placental abruption or a prolapsed cord, and is also referred to as a pattern following ‘acute near total asphyxia’,

[95.2] Volpe – refers to the structural lesions as described in this matter as the “cerebral-cortex-deep nuclear injury”, occurring because of a moderate insult of prolonged duration[[51]](#footnote-51),

[95.3] ACOG– refers to the lesion as the “Cerebral-deep nuclear neuronal injury” which occurs after a severe partial insult of prolonged duration or a combined partial with profound terminal insult[[52]](#footnote-52),

[95.4] Wisnowski– likewise to Volpe, describe the structural injury as the Central/BGT (also known as “Cerebrocortical-deep nuclear”) pattern. This pattern is characterized by injury to the BGT and may extend to the Cerebral cortex, usually localized to the perirolandic region. On neuropathology, it is often referred to as the cerebrocortical-deep nuclear pattern”. The BGT pattern is typically bilateral and symmetric. The authors also stated that the cerebrocortical-deep nuclear pattern occurs after sentinel events, or severe partial asphyxia with prolonged duration, or a combination of partial and near-total asphyxia[[53]](#footnote-53),

[95.5] Nakao– acknowledged that the BGT injuries were common after acute and total insults, but also indicated that when cortical involvement (as occurred in this matter) accompanied BGT damage, the insult was gradual and partial. Of the intrapartum asphyxia – CP group (-37%), there was gradual developing hypoxia (> 1 hour) in 17%, or an initial reassuring foetal heartrate pattern, followed by abrupt (< 1 hour) severe bradycardia in 20%[[54]](#footnote-54).

[96] NB has mixed type cerebral palsy (predominantly dystonic). Her co-morbidities include profound intellectual disability, microcephaly, wasting and nutritional concerns, early contractures uncontrolled epilepsy, and severe developmental delay[[55]](#footnote-55).

[97] NB’s clinical outcome is in keeping with the structural brain injury.

[98] The experts agreed with ACOG analysis that in the present matter there was no identifiable perinatal sentinel event.

[99] In the present matter there was no identifiable perinatal sentinel event.

[100] NB was born in a lifeless condition. His one-minute Apgar Score of ‘0’ (zero) implies that a preceding bradycardia must have been present before it terminated in asystole[[56]](#footnote-56) (flatline) during the latter stage of the second stage of labour.

[101] The probable terminal, bradycardia – asystole situation could have been the consequence of one of three processes, namely;

[101.1] There was severe partial asphyxia with prolonged duration (process 1), or

[101.2] A combination of partial and near-total asphyxia (process 2), or

[101.3] There was a sudden onset terminal bradycardia, without forewarning (process 3).

[102] Process 3 may rarely occur as a de novo (sudden onset) bradycardia. In this matter the cervix had been fully dilated since 13h00. If sudden onset, terminal bradycardia occurred during the last 45 minutes before birth, foetal monitoring would have detected same and birth could have been expedited before a brain-injurious threshold of 20 minutes was reached.

[103] A sudden onset is a rare phenomenon, certainly in cases where there was no perinatal sentinel event (PSE) or no prior foetal abnormalities.

[104] A tight nuchal cord could have been involved in all three “processes” referred to in paragraph 101 above as would its probable accompanying abnormalities would have been readily detectable with foetal heart rate monitoring and intervention could follow.

[105] There is no evidence of any foetal monitoring between 11h05 and the delivery of the baby at 14h09. Any, and all, foetal distress and foetal heart rate abnormalities during this period would therefore have been missed.

[106] In considering the question as to when the insult turned into injury, Prof Smith is of the opinion that if it is accepted that the foetal condition was reassuring until 11h05, and considering “process 1 and 2” (i.e, there was severe partial asphyxia with prolonged duration or a combination of partial and near-total asphyxia respectively), it then follows that a hypoxic or hypoxic insults occurred thereafter, and that the hypoxic insults turned into injury at some point in time. Reference is made to Flesher and associates who showed that when initial foetal heartrate tracings were normal (as in this case until probably 11h05), a relative acidosis-free interval could be demonstrated during the first 90 to 100 minutes. Following this period rapid cumulative threshold acidosis was seen, which varied according to the foetal heartrate pattern observed. The threshold acidosis values for various patterns were late decelerations, 115 minutes variable decelerations, 145 minutes. Applying their findings to the present matter takes one to approximately 13h00 if there were late foetal heartrate decelerations after 11h05, and approximately 13h30 if there was repeated variable foetal heartrate decelerations after 11h05 following development of threshold acidosis, another 10-20 minutes is required to significantly injure the brain.

[107] Prof Smith therefore concludes that for “processes 1 and 2” an abnormal foetal condition probably existed between approximately 12h50 and 13h35 in the present matter and Dr Ballot said “It is difficult to comment on the time of the asphyxia episode in the absence of any fetal heartrate monitoring between 11h05 and 14h09”.

**THE EVIDENCE**

[108] No evidence was adduced on behalf of the Defendant. Two witnesses testified on behalf of the Plaintiff, Dr Murray (Obstetrician / Gynaecologist) and Prof Smith (Neonatologist / Paediatrician). The joint minutes of Dr Alheit and Dr Mweli, (Radiologists) and Prof Smith and Dr De Ballot, (Neonatologists / Paediatricians) were accepted as part of the evidence. So too were the hospital records and expert summary reports.

**THE VALUE OF EXPERT OPINION**

[109] At the center of this matter are agreements between experts and evidence adduced by experts on behalf of the Plaintiff. The Supreme Court of appeal in **Glenn Marc Bee v The Road Accident Fund (093/2017) [2018] ZASCA52 (29 March 2018)** dealt with the opinions of expert witnesses and their evidence as follows:

“[22] It is that an expert witness is required to assist the court and not to usurp the function of the court. Expert witnesses are required to lay a factual basis for their conclusions and explain their reasoning to the court. This court must satisfy itself as to the correctness of the expert reasoning… An expert opinion must be underpinned by proper reasoning in order for the court to assess the cogency of that opinion. Absent any reasoning the opinion is inadmissible... ‘courts are not bound by the view of any expert. They make the ultimate decision on issues on which experts provide an opinion’.

[23] The facts on which the expert witness expresses an opinion must be capable of being reconciled with all other evidence in the case. For an opinion to be underpinned by proper reasoning, it must be based on correct facts. Incorrect facts militate against proper reasoning and the correct analysis of the facts is paramount for proper reasoning, failing which the court will not be able to properly assess the cogency of that opinion. An expert opinion which lacks proper reasoning is not helpful to the court.

[28] … Expert witnesses are witnesses who are allowed to speak to their opinion, but they are not the judges of the fact in relation to which they express an opinion, the court… is the judge of the fact…

[30] … if an expert witness cannot convince the court of the reliability of the opinion and his report, the opinion will not be admitted. The joint report of experts is a document which encapsulates the opinions of the experts and it does not lose the characteristics of expert opinion. The joint report must therefore be treated as expert opinion. The fact that it is signed by two or more experts does not alter its characteristic of expert opinion. The principles applicable to expert evidence or reports are also applicable to a joint report. The joint report before the court is consequently part of evidential material which the court must consider in order to arrive at a just decision. The court, in such an instance, will be entitled to test the reliability of the joint opinion, and if the court finds the joint opinion to be unreliable, the court will be entitled to reject the joint opinion. The court is entitled to reject the joint report or agreed opinion if the court is of the view that the joint report or opinion is based on incorrect facts, incorrect assumptions or is unconvincing.”

**NEGLIGENCE**

[110] Both Counsel cited helpful legal authorities in relation to negligence and factual causation relevant to medical negligence to which I am indebted. It is trite that the negligent conduct of a medical professional is to be assessed against the standards prevailing in the medical profession at a particular time period, in order to determine whether reasonable steps were taken or not. In the oft-quoted leading case on negligence, **Kruger v Coetzee**[[57]](#footnote-57), Holmes JA stated as follows:

“For purposes of liability culpa arises if –

(a) A diligent paterfamilias in the position of the defendant –

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

(ii) Would take reasonable steps to guard against such occurrence, and

(b) The defendant failed to take such steps.”

[111] In **Sea Harvest Corporation (Pty) Ltd and Another v Duncan Dock Cold Storage (Pty) Ltd and Another**[[58]](#footnote-58) the Supreme Court of Appeal stated that:

“[21]… In the ultimate analysis the true criterion for determining negligence is whether in particular circumstances conduct complained of falls short of the standard of the reasonable person. Dividing the inquiry into various stages, however useful, is no more than an aid or guideline for resolving this issue.

[22] It is probably so that there can be no universally applicable formula which will prove to be appropriate in every case. As Lord Oliver observed in **Caparo Industries PLC v Dickman and Others [1990] UKHL2, [1990] 2 AC 605 (HL) at 633 F-G**; “the attempt to state some general principle which will determine liability in an infinite variety of circumstances serves not to clarify the law but merely to bedevil its development in a way which corresponds with practicality and common sense.”

[112] “71. In simple terms, negligence refers to the blameworthy conduct of a person who has acted unlawfully. In respect of medical negligence, the question is how a medical practitioner in the position of the defendant would have acted in the particular circumstances”[[59]](#footnote-59)

[113] “72… What is or is not reasonably foreseeable in any particular case is a fact bound enquiry… Where questions that fall to be answered are fact bound there is seldom any assistance to be had from other cases that do not share all the same facts”[[60]](#footnote-60)

[114] It is trite that the negligent conduct of the medical practitioners is to be assessed against the standards prevailing in the medical profession at a particular time period, in order to determine whether reasonable steps were taken or not. This is so because of the rapid developments in the medical field influenced by innovative technologies. In this case, the applicable standards are those contained in the Department of Health Guidelines for Maternity Care in South Africa 3rd Edition 2007 (the Guidelines).

[115] Health professionals such as doctors and nurses are required to dispense reasonable care by adhering to the level of skill and diligence exercised by members of their profession failing which they would be negligent. In the circumstances of this case, the hospital staff who attended to the Plaintiff will be found to have been negligent if, in dispensing medical care to the Plaintiff, they failed to foresee this possibility and would have taken steps to prevent it.

[116] The facts of the case are substantially undisputed. Joint minutes were submitted by consent. Two expert witnesses adduced evidence on behalf of the Plaintiff. No evidence was led on behalf of the Defendant. The Plaintiff presented to the Hospital at 09h05 on 15 July 2013 complaining of labour pains. On vaginal examination, the cervix was 5cm dilated. The foetal heart rate (FHR) was 135 to 145 beats per minute. No maternal-foetal or labour risk factors were identified. The Plaintiff presented in the active phase of labour (5cm cervical dilation) at a term gestation, meaning labour occurred at the right time when neonatal risks would be expected to be low and labour was allowed to progress. There were no concerning features regarding presentation during labour.

**FOETAL MONITORING**

[117] It is not disputed that there were discrepancies in the two sets of clinical notes wherein the Apgar scores were recorded as 5/10 and 9/10 at 1 and 5 minutes of life respectively and those which were recorded after birth as 0/10 and 3/10 at 1 and 5 minutes of life respectively. The conclusion of experts that the Apgar scores recorded after birth (0/10 and 3/10) are more accurate as in keeping up with the condition of the baby is in my view more plausible. The fact that the baby was born flat and bluish in colour, did not cry on delivery and had to be resuscitated supports the Apgar scores which revealed that the baby was born lifeless.

[118] It is common cause that the CTG device was out of order at the time of delivery of NB and therefore no evidence of electronic tracing of the foetal heart monitoring as well as the maternal contractions were presented. Dr Murray opined that given the fact that the Plaintiff was a low-risk patient, monitoring of the foetal heart by way of CTG was not necessary as auscultation of the foetal heart was appropriate. She stated that there was nothing wrong about the Plaintiff as a first-timer that required the CTG to be performed but that the hospital is required to have a CTG machine should the condition of the mother change. This view by Dr Murray was taken at the initial stage of “Assessment No. 1”.

[119] The presence of pyrexia and of thick meconium were detected during “Assessment No. 2”, when the labour process had advanced and the maternal and foetal condition required careful monitoring. Dr Murray stated that the use of CTG device had become necessary for symptoms to be detected timeously. Dr Murray, in my view, did not proffer a blanket exemption from the use of the CTG, nor did she justify the conduct of midwives for failure to use the CTG in contravention of the Maternity Guidelines, as suggested by Counsel for the Defendant.

[120] Perhaps a more clearer view is one posed by Prof Smith that the fact that the CTG was out of order puts an onerous burden on the midwives to manually auscultate the foetal heart and/or to use a Doppler device to monitor the foetal condition in accordance with the Guidelines. I agree. The Guidelines poses as a standard against which the conduct of the medical profession are measured without which it will be impossible, to detect any abnormalities and to expedite delivery timeously.

[121] Prof Smith did not agree with Dr Murray, given authorities at hand that there was probability that the foetus may have suffered infection as a result of pyrexia, stating that temperatures around 38.4˚C are normal for women in labour process thus ruling out the possibility of foetal injurious infection as a result of pyrexia in this case.

[122] There was fresh thick meconium at the time of delivery. Dr Murray stated that this finding was abnormal as meconium has been associated with poorer neonatal outcome and a strong indicator of foetal distress. This was not detected as a result of lack of monitoring during active labour.

[123] The presence of the nuchal cord around the neck or around the neck and arm tightly applied was ruled out by Dr Murray as a possible risk factor stating that it is inconceivable that a hand or foot pulled up near the head could bring a loop of cord with it and cause some entanglement of the cord as is described in the record. Prof Smith on the other hand stated that there probably was detectable foetal heart rate abnormalities and foetal distress during the intrapartum period. This was not diagnosed because there was no foetal monitoring during this period.

[124] The clinical notes also revealed that there was a cord compression and Dr Murray opined that if the cord was wound multiple times around the neck, it is unlikely that there would be no signs at all of cord compression during labour before delivery, and that if it is accepted that the tight cord was problematic for the foetus, there would probably have been warnings of this during the course of labour and intervention could have resulted in expedited delivery, thereby not exposing the foetus to further risks of hypoxia. This was not detected as there was no monitoring during this critical period of labour.

[125] NB developed an early moderate or moderate-severe degree of neonatal encephalopathy. The joint minutes of the Neonatologists shows that both experts are in agreement except where Dr Ballot expressed a disqualification as to when the insult or injury occurred. During cross-examination, Prof Smith responded that Dr Ballot did not disagree when he said “It is difficult to comment on the time of the asphyxia episode in the absence of any fetal heart rate monitoring between 11h05 and 14h09”, he simply expressed difficulty as to time. It was contended on behalf of the Defendant that the fact that an expert opinion is unchallenged, does not necessarily mean that it must be accepted. I disagree with this line of reasoning. The experts are not in disagreement. The response of Dr Ballot is simply that if it was not for lack of monitoring of foetal heart rate between 11h05 an 14h09, I would have been able to opine on the timing.

[126] The Neonatologists agreed that in the present matter there was no identifiable sentinel event as per ACOG. The probable ‘terminal bradycardia-asystole’ situation could have been the consequences of one of the three processes, namely ‘Process 1 – There was severe partial asphyxia with prolonged duration, or Process 2 – a combination of partial and near-total asphyxia, or Process 3 – There was sudden onset terminal bradycardia, without forewarning’.

[127] In ruling out the probability of Process 3, the experts agreed that ‘Process 3’ may rarely occur as a de novo (sudden onset) bradycardia. In this matter the cervix had been fully dilated since 13h00. If a sudden onset terminal bradycardia occurred during the last 45 minutes before birth, foetal monitoring would have detected same and birth could have been expedited before a brain-injurious threshold of 20 minutes was reached.

[128] There was also agreement that ‘a sudden onset foetal bradycardia is a rare phenomenon certainly where there were no perinatal sentinel event (PSE) or no prior foetal heart rate abnormalities’ and that ‘a tight nuchal cord could have been involved in all three ‘processes’ as would its probable accompanying abnormalities of the foetal heart rate. These abnormalities would have been readily detectable with foetal heart rate monitoring and intervention could follow. There is no evidence of any foetal monitoring between 11h05 and delivery of the baby at 14h09. Any, and all foetal distress and foetal heart rate abnormalities during this period would therefore have been missed.

[129] All things considered, I find that the Plaintiff has established on a balance of probabilities that the Defendant’s employees were responsible for the care and treatment of the Plaintiff and her child and that they were negligent as expounded above.

**CAUSATION**

[130] NB’s brain injury pattern is according to the MRI report an extensive and severe RBGT. The Radiologists ruled out the presence of an obstetric sentinel event in this case. Prof Smith opined that if it is accepted that the foetal condition was reassuring until 11h05, then the abnormal foetal condition probably existed between approximately 12h50 and 13h35. Dr Murray’s evidence is that NB would not have suffered the brain injury, had the midwives expedited delivery by vacuum 20 minutes before delivery, or by forceps 10 minutes before delivery but missed this opportunity because of failure to monitor the foetal condition. There was no recording of a sentinel event (abruption placenta, placenta praevia, cord prolapse, uterine rapture or tear of maternal collapse) having occurred, which could explain the birth of a compromised baby in this matter.

[131] It is trite that causation has two elements, namely: (i) factual causation, determined by applying the ‘but for’ test, and (ii) legal causation which answers the question of whether the wrongful act is linked sufficiently closely to the harm suffered; if the harm is too remote, then there is no liability[[61]](#footnote-61).

[132] ‘The criterion applied by the court aquo for the determining of factual causation was the well known but-for test… What it essentially lays down is the enquiry – in the case of an omission – as to whether, but for the Defendant’s wrongful and negligent failure to take reasonable steps, the Plaintiff’s loss would not have ensued… the application of the “but-for test” is not based on mathematics, pure science or philosophy. It is a matter of common sense, based on the practical way in which the minds of ordinary people work, against the background of everyday-life experiences. In applying this common sense, practical test, a Plaintiff has to establish that it is more likely that, but for the Defendant’s wrongful and negligent conduct, his or her harm would not have ensued. The Plaintiff is not required to establish this causal link with certainty.’[[62]](#footnote-62)

[133] Applying the test set out above to the facts in this case, the crisp question is: Is it more likely than not that, but for the wrongful and negligent conduct of the Defendant’s employees, NB would not have suffered a brain injury during the birth process, as a result of hypoxic ischaemia?

[134] In her summary report Dr Murray relied on a case study of Smith et al[[63]](#footnote-63) where 10 medico-legal cases of neonatal encephalopathy – cerebral palsy survivors who sustained intrapartum hypoxic ischaemic (HI) basal ganglia thalamic (BGT) Pattern injury in the absence of an obstetric sentinel event. All 10 cases had evidence of pathological or suspicious CTG tracing prior to delivery and the median time interval between first pathological CTG and delivery of the infant was 179 minutes. The authors concluded that if a non-reassuring foetal status develops during labour and is prolonged, a BGT pattern HI injury may result, in the absence of a perinatal sentinel event. Intrapartum BGT pattern injury and radiologically termed “acute profound HI brain injury” are not necessarily synonymous. A visualized MRI scan pattern should therefore preferably solely reflect the pattern’s description and severity, rather than a causative mechanism of injury.

[135] In the 10 medico-legal cases and cases such as the present one, where there is no demonstrable sentinel event, the rendering of safe and appropriate intrapartum care, including regular foetal monitoring and reacting to signs of foetal distress, readily evident by foetal heart rate auscultation, would be expected to detect those foetuses not tolerating the stressors posed by labour before the hypoxia reaches a point where injury ensues.

[136] In other words, Smith et al concluded that in the absence of a perinatal sentinel event, subacute or subthreshold prolonged or intermittent intrapartum hypoxic ischaemia may cause BGT pattern brain injury but that warning signs in the form of non-reassuring foetal status, would be detectable by means of CTG or auscultation monitoring up to a few hours before delivery.

[137] The findings of the study by Smith et al was at the centre of the appeal in LWM. The MEC for Health Limpopo contended that “the High Court (GPPHC), erroneously accepted the validity of Smith et al even though the theory was in its developmental stage and thus unsupported, was not compelling and ran contrary to the ‘traditional view’ that a BGT pattern (grey matter injury) is associated with an acute profound hypoxic ischaemic event. The theory posited in that article had already been rejected in this court in AN obo EN v MEC for Health, Eastern Cape (AN v MEC), so it was argued. The appellant laid great emphasis on the fact that the generally accepted view supported by literature was that acute profound insults happen because of sentinel events and occur suddenly and without warning,and therefore could not be averted[[64]](#footnote-64).” “… The appellant contended that even if Prof Smith’s article were to be regarded as authoritative, there was no evidence showing that the foetus was in a compromised state for a prolonged time prior to delivery, which is a fact that is necessary for the application of Prof Smith’s theory”[[65]](#footnote-65)

[138] In determining whether on the facts set out in para [136] above, Prof Smith’s opinion was founded on a logical reasoning, the SCA stated that, “… This includes an assessment of whether the reasoning or methodology underlying his testimony is scientifically valid and whether that reasoning or methodology can be applied to the facts in issue. It is about the cogency of the underlying reasoning which lead the experts to their conflicting opinions. If the expert’s opinion is logical and can reasonably be held on those facts and his chain of reasoning, then the threshold will be satisfied even though his is not the only opinion that can be expressed on those facts”[[66]](#footnote-66)

[139] In addressing the criticism levelled against the article of Smith et al, the SCA concluded that, “… There is nothing illogical about Prof Smith’s opinion. It was not and could not be disputed that Prof Smith and his colleagues had identified 10 cases of patients with BGT pattern injuries (with no sentinel events and no fixed terminal bradycardia), where proper monitoring demonstrated that the babies had commenced displaying foetal distress at a median of about three hours before delivery. Thus, it was uncontroverted that such cases are possible and the only real remaining question on the merits is whether this probably was such a case.”[[67]](#footnote-67)

[140] The SCA held that “the opinion propounded by Prof Smith’s logical reasoning, survives scrutiny and is foursquare in accordance with the Linksfield principle”[[68]](#footnote-68)

[141] On the merits, the SCA stated that “Although the appellant criticized Prof Smith’s expert opinion regarding the mechanism of the insult and his reliance on the case-study involving 10 cases, the appellant could not point out to any contrary literature. None of the articles submitted by the appellant ruled out the theory that intermittent episodes of hypoxia can culminate in an injury of an acute profound type as propounded by Prof Smith”[[69]](#footnote-69)

[142] The SCA was satisfied that the High Court’s acceptance of Prof Smith evidence “that, a series of partial intermittent subacute / subthreshold hypoxic insults can result in this type of injury to the BGT deep nuclear structures including the perirolandic area was justified[[70]](#footnote-70)

[143] In determining the causation element the SCA stated that “the starting point is that the BGT brain injury pattern revealed by the MRI, i.e the injury to the central or deep grey matter of the brain, (the basal ganglia and/or thalami and/or sensorimotor cortex), could in principle have been caused either by an acute profound total or near-total hypoxic ischaemic insult or intermittent or prolonged episodes of subacute and subthreshold interruption of the supply of blood to the brain. Of cardinal importance in this regard is that in their joint minutes, Dr Murray and Prof Lombaard agreed that there was no evidence of a sentinel event, whereas the AN v MEC judgement recorded that the experts were agreed that there had been a sentinel event. This is one crucial aspect that distinguishes the case AN v MEC on the facts[[71]](#footnote-71).

[144] “It is a trite principle of our law that every case must be decided on its own facts and on the evidence adduced in that specific matter. Factual findings made in one case cannot be transferred to produce the same factual findings in another case with similar facts. AN v MEC was a judgement reached on the basis of expert evidence presented in that case and its conclusion was based on the facts of that case”[[72]](#footnote-72).

[145] In the present matter Counsel for the Defendant also relied on AN v MEC to refute the joint minute of the Neonatologists / Paediatricians that there was no sentinel event at the time of delivery of NB without providing any factual evidence or contrary study to that effect.

[146] Critical to the question whether based on evidence there was a causal link between the harm suffered and the negligent conduct of the midwives, the SCA held that “Both obstetric experts agreed that the exact time of which foetal distress occurred was impossible to determine due to the absence of clinical notes detailing the last 95 minutes of the respondent’s labour. Despite it having been the hospital staff’s obligation to monitor the foetal heart rate and to make the necessary clinical notes which it failed to do; the appellant tried to capitalize on the fact that the exact times at which the foetal heart rate was indicative of foetal distress could not be established. In my opinion, it is fallacious to posit that where a woman in labour has not been monitored by hospital personnel at all during the most critical stage of her labour, the MEC responsible for the relevant hospital should escape liability arising from negligence of its employees purely on the basis that the exact timing of the hypoxic injury of an acute profound nature cannot be ascertained. To do so would be to ignore the uncontested evidence that, on probabilities, shows a link between the negligence and the harm that ensued”[[73]](#footnote-73).

[147] The facts of LWM are similar to those in the present matter and the conclusions reached by the SCA based on the uncontested evidence of the experts. I referenced LWM substantially because what was stated in that case is what I would have stated without doubt.

[148] On probabilities, the brain injury sustained by NB would not have occurred had the Plaintiff and the foetus been properly monitored and managed by the hospital staff. I am satisfied that the evidence adduced on behalf of the Plaintiff, points to a causal connection between the substandard care the Plaintiff received from the hospital staff during labour and the acute profound hypoxic injury sustained by NB. In other words, there is a sufficiently close connection between the negligence of the nurses that attended to the Plaintiff and the damage suffered by NB. In the circumstances, causation has been established on a balance of probabilities.

**CONCLUSION**

[149] In an emergency situation the promptness of hospital staff is key and every minute counts. Adequate monitoring would have been able to detect when the change in the foetal heart rate occurred. There were indeed forewarnings in the form of foetal decelerations throughout the active phase of labour but these decelerations were not detected and recorded due to inadequate monitoring. The acute profound insult that occurred in the last 45 minutes of labour would probably have been averted if the provisions of the guidelines had been followed. In other words, the injury would probably not have resulted, but for the negligence of the hospital staff. By failing to properly monitor the Plaintiff’s labour in accordance with the provisions of the guidelines, the hospital staff created a situation that placed the foetus at a serious risk of HIE and its attendant sequelae. This risk precluded the timeous execution of any measures designed to avert injuries consequent upon suffering HIE.

[150] In the circumstances, the Defendant’s employees were negligent as their conduct failed to pass the standard of maternal and foetal monitoring as required in terms of the Maternity Guidelines. The hospital staff should have foreseen the reasonable possibility of their conduct causing harm to the foetus and should have taken steps to guard against such an occurrence. They failed to uphold this standard. The cause of NB’s injury resulted from the negligent conduct of the Defendant’s employees, acting in the cause and scope of their employment at De La Rey Hospital as aforesaid. In the premises, the Defendant is, in his nominal capacity, vicariously liable for the damages suffered by NB during delivery.

**ORDER**

[151] In the circumstances, the following order is made:

1. The Defendant is liable for payment of 100% (one hundred percent) of the proven or agreed damages of the Plaintiff’s minor daughter, NB.

2. The issue of quantum is postponed sine die.

3. The Defendant shall pay the Plaintiff’s taxed or agreed costs of suit on the High Court scale as between party and party up to finalisation of the issue of liability.

\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

**O.Y DIBETSO-BODIBE**

**ACTING JUDGE OF THE HIGH COURT**

**NORTH WEST DIVISION, MAHIKENG**

*Delivered: This judgment was prepared and authored by the Judge whose name is reflected and is handed down electronically by circulation to the Parties/their legal representatives by email*

**APPEARANCES**

**DATE OF HEARING: 15, 16 and 24 August 2023**

**DATE OF JUDGEMNT: 03 January 2024**

**COUNSEL FOR THE APPLICANT: Adv M Coetzer**

**Instructed by: Wim Krynauw Attorneys**

**COUNSEL FOR THE DEFENDANT: Adv R Rathidili**

**Instructed by: The State Attorney , Mahikeng**

1. **Applied**: The application of pressure by the foetal head on the cervix that is caused by its downward movement. [↑](#footnote-ref-1)
2. **Partogram**: A graphic record of the course of labour that inter alia plots cervical dilation, foetus heart rate, duration of labour and vital signs of and provide for alert and action lines to prompt intervention if the curve deviates from the expected. [↑](#footnote-ref-2)
3. **Cardiotocograph (CTG)**: A tracing by means of electronic transducers which monitor the foetal heart (“cardio”) as well as the maternal contractions (“toco”) and this is seen as a continuous monitor on a screen and can be printed simultaneously on paper (“graph”) [↑](#footnote-ref-3)
4. **Gestation**: The period during which a fertilized egg cell develops into a baby that is ready to be delivered. [↑](#footnote-ref-4)
5. **Hypoxic Ischaemic Encephalopathy or Neonatal Encephalopathy**: A type of new-born brain damage caused by oxygen deprivation and limited blood flow. [↑](#footnote-ref-5)
6. **Cerebral Palsy**: Also known as CP is a group of disorders that affect a person’s ability to move and maintain balance and posture. CP is the most common motor disability in childhood. Ce.re.bral: of the brain – pal.sy: lack of muscle control – is caused by abnormal brain development or damage to the developing brain that affects a person’s ability to control his or her muscles. [↑](#footnote-ref-6)
7. **Station**: Is the descent of the foetal presenting part in the pelvis. An imaginary line is drawn between the two bones in the pelvis (known as the ischial spines) This is the “zero line”, and when the foetus reaches this line it is considered to be in the “zero station”. When the foetus is above this imaginary line it is in a “minus” station. When the foetus is below, it is in the “plus” station. [↑](#footnote-ref-7)
8. **Caput**: Is the temporary swelling of the soft parts of the head of a newly born infant that occurs during labour, due to the compression of the muscles of the cervix of the uterus. [↑](#footnote-ref-8)
9. **Moulding**: Is the changing of the shape of the bone of the skull, which is brought about by the pressures that it is subjected to when passing through the birth canal. [↑](#footnote-ref-9)
10. **Heart rate**: The normal heart rate ranges between 120 and 160 beats per minute (bpm) [↑](#footnote-ref-10)
11. **Haemoglobin**: An iron containing pigment of red blood cells that delivers oxygen from the lungs to the tissues of the body. [↑](#footnote-ref-11)
12. **Active labour**: Is that part of labour from 4cm cervical dilation until full dilation (10cm) [↑](#footnote-ref-12)
13. **Amniotic Fluid / Liquor**: Is a clear slightly yellowish liquid that surrounds the fetus (unborn baby) during pregnancy. It is contained in the amniotic sac. [↑](#footnote-ref-13)
14. **Intermittent auscultation**: Is the process of listening, usually with a stethoscope or a hand-held doppler device, to sounds produced by movement of agas or liquid within the body. [↑](#footnote-ref-14)
15. **Palpation**: Examination of the abdomen to discover how the baby is lying in the uterus. [↑](#footnote-ref-15)
16. **Pubic symphysis**: A joint sandwiched between the left pelvic bone and the right pelvic bone. [↑](#footnote-ref-16)
17. Potential hypoxic stress associated with uterine contractions is healthy during the active phase of labour. Hypoxic stress is initiated as contractions cyclically reduce blood and produce repeated hypoxic stress/ [↑](#footnote-ref-17)
18. Intra-uterine resuscitation consists of applying specific measures with the aim of increasing oxygen delivery to the placenta and umbilical blood flow in order to reverse hypoxia (prolonged reduction in oxygen supply to the brain) and acidosis (an excessively acid condition of the body fluids or tissues) [↑](#footnote-ref-18)
19. Station – See Footnote No. 7 [↑](#footnote-ref-19)
20. Caput – See Footnote No. 8 [↑](#footnote-ref-20)
21. Moulding – See Footnote No. 9 [↑](#footnote-ref-21)
22. Liquor / Amniotic Liquor – See Footnote No. 13 [↑](#footnote-ref-22)
23. **Meconium**: Is the first stools of a new-born baby that can also be passed in utero. Although meconium is normally retained in the infant’s bowel until after birth, it sometimes is expelled into the amniotic fluid (also called “amniotic liquor”) prior to birth or during labour and delivery, hence the reference to meconium-stained liquor (MSL) [↑](#footnote-ref-23)
24. Intervillous space of the placenta is a part of the fetal-maternal interface, where maternal blood enters to provide nutrients and gas exchange. [↑](#footnote-ref-24)
25. **Hypoxaemia**: An abnormally low concentration of oxygen in the blood. [↑](#footnote-ref-25)
26. **Acidosis**: An excessively acid condition of the body fluids or tissues. [↑](#footnote-ref-26)
27. **Hypoxia**: An absence of enough oxygen in the tissues to sustain bodily functions. [↑](#footnote-ref-27)
28. **Compound presentation**: A foetal presentation in which an extremity presents alongside the part of the foetus closest to the birth canal. [↑](#footnote-ref-28)
29. **Nuchal Cord (NC)**: NC is the umbilical cord being wrapped 360˚ around the foetal neck. It is estimated that the NC affects 23-33% of all pregnancies, i.e, it is quite common. A single loop is seen in 23-34% of pregnancies, two loops in 2.5-5%, and 3 loops are found in 0.2-0.5% of all pregnancies. More than three loops are rare. [↑](#footnote-ref-29)
30. Cord compression / umbilical cord compression happens as a result of pressure flattening or stretching the umbilical cord, affecting blood flow to the foetus. [↑](#footnote-ref-30)
31. **Apgar scores**: A method of rapidly assessing the general state of a baby immediately after birth. A maximum of two points is given for each of the following signs, usually measured at one minute and five minutes after delivery: type of breathing, heart rate, colour, muscle tone, and response to stimuli. [↑](#footnote-ref-31)
32. **Resuscitation**: Is an emergency medical intervention techniques employed immediately after childbirth to assist babies who are not able to breath independently after birth. [↑](#footnote-ref-32)
33. **Moro-reflex**: A reflex reaction of infants upon being startled that is characterized by extension of the arms and legs away from the body and then by drawing them together as if in an embrace. [↑](#footnote-ref-33)
34. Magnetic Resonance Imaging (MRI) is a medical imaging technique that uses a magnetic field and computer-generated radio waves to create detailed images of the organs and tissues in the body. In this case the MRI was used to scan the brain of the child, NB. After NB was diagnosed of hypoxic ischaemic encephalopathy, a brain damage pattern. An MRI scan provides an estimation of the extent of the brain damage and the possible long-term disabilities the child may face. [↑](#footnote-ref-34)
35. **Bradycardia**: Slowing of the heart rate. For infants, bradycardia is defined as a heart rate of less than 100 beats per minute (Normal is around 120 – 160 beats per minute) [↑](#footnote-ref-35)
36. Baxter P. Markers of perinatal hypoxia-ischaemia and neurological injury: assessing the impact of insult duration. Dev Med Child Neurol 2020; 62: 563 – 568. [↑](#footnote-ref-36)
37. **Placental abruption**: A condition known as abruption placentae, which is defined as bleeding from a normally situated placenta causing it complete or partial detachment from the uterine wall after the 24th week of gestation. [↑](#footnote-ref-37)
38. **Placenta praevia**: A condition in which the placenta partially or wholly blocks the neck of the uterus, so interfering with normal delivery of a baby. [↑](#footnote-ref-38)
39. **Umbilical cord prolapse**: The umbilical cord is pushed into the vigina ahead of the baby and becomes compressed, cutting off blood flow to the baby. [↑](#footnote-ref-39)
40. **Uterine rapture**: A tear in the uterus. [↑](#footnote-ref-40)
41. Yamada and co-workers (Intrapartum risk factors for neonatal encephalopathy leading to cerebral palsy in women without apparent sentinel events. J Obstet Gynaecol Res 2015). [↑](#footnote-ref-41)
42. **Feto-maternal hemorrhage**: The entry of fetal blood into the maternal circulation before or during delivery. Feto-maternal hemorrhage may have devastating consequences for the fetus such as neurological injury, stillbirth, or neonatal death. [↑](#footnote-ref-42)
43. **Suboptimal care**: Failure to seek and provide appropriate and timely interventions to risk patients. [↑](#footnote-ref-43)
44. De Knijf and Pattison (Confidential enquires into quality of care of women in labor using Hypoxic Ischaemic Encephalopathy as a marker. F, V&V IN OBGYN 2010) [↑](#footnote-ref-44)
45. Clapp JF, et al. (The natural history of antenatal nuchal cords. AM J Obstet Gynecol 2003) [↑](#footnote-ref-45)
46. **Perinatal period**: The perinatal period as defined by the World Health Organization, starts at 22 completed weeks of gestation (the time when birth weight is normally 500g) and ends 7 completed days after birth. [↑](#footnote-ref-46)
47. **Akkaya and co-workers (Nuchal cord)**: is it really the silent risk of pregnancy? J Matern Fetal Neonatal Med 2016) [↑](#footnote-ref-47)
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50. Patterns of neonatal hypoxic-ischaemic brain injury. Neuroradiology 2010, 52:555-566 [↑](#footnote-ref-50)
51. Page 201, Table 19.1 Volpe JJ editor. Hypoxic-ischaemic injury in the term infant: neuropathology, and pathophysiology. In Volpe’s neurology of the newborn. 6th ed. Philadelphia, PA Elsevier, 2018. Pp. 484-499, 500-583 [↑](#footnote-ref-51)
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53. Wisnowski JL, et al. Neuroimaging in the term newborn with neonatal encephalopathy. Seminars in Foetal and Neonatal Medicine 2021, 26(5) [↑](#footnote-ref-53)
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57. Kruger v Coetzee 1966 (2) SA 428 at 430 E - F [↑](#footnote-ref-57)
58. (12/97) [1999] ZASCA 87 (26 November 1999) [↑](#footnote-ref-58)
59. Oppelt v Head: Health, Department of Health, Provincial Administration, Western Cape (CCT/185/14) [2015] ZACC 33 (14 October 2015) (“Oppelt”) [↑](#footnote-ref-59)
60. Oppelt at para [10] [↑](#footnote-ref-60)
61. MEC for Health, Limpopo v LWM obo DM (502/2021) [2022] ZASCA 146 (27 October 2022) (“LWM”) at para [24] [↑](#footnote-ref-61)
62. LWM at para [24] [↑](#footnote-ref-62)
63. Smith J, Solomons R, Vollmer L, Langenegger E, Lotz J, Andronikou S, Aonthony A, Van Joorn R. Intrapartion Basal Ganglia – Thalamic Pattern Injury and Radiologically Termed “Acute Profound Hypoxic Ischaemic Brain Injury” Are not synonymous’ (2020) American Journal of Perinatology [↑](#footnote-ref-63)
64. LWM at para [26] [↑](#footnote-ref-64)
65. LWM at para [27] [↑](#footnote-ref-65)
66. LWM at para [28] [↑](#footnote-ref-66)
67. LWM at para [29] [↑](#footnote-ref-67)
68. LWM at para [31] [↑](#footnote-ref-68)
69. LWM at para [32] [↑](#footnote-ref-69)
70. LWM at para [38] [↑](#footnote-ref-70)
71. LWM at para [39] [↑](#footnote-ref-71)
72. LWM at para [40] [↑](#footnote-ref-72)
73. LWM at para [47] [↑](#footnote-ref-73)