NOT REPORTABLE

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

CASE NO. 413/2017

In the matter between:

NKM (obo BM) Plaintiff

and

THE MEMBER OF THE EXECUTIVE COUNCIL
FOR HEALTH: EASTERN CAPE Defendant

(in re the negligence of the staff at the Livingstone and Dora Nginza Hospitals, Gqeberha)

JUDGMENT IN RESPECT OF MERITS

HARTLE J

Introduction:

[1] The plaintiff claims damages in her representative capacity on behalf of her minor son, BM, who was born at the Dora Nginza Hospital in Gqeberha ("*DNH*") on 19 August 2007.

[2] BM, presently 16 years of age, suffers from asymmetrical cerebral palsy with one side more affected than the other. Although this is not the typical spastic or dyskinetic cerebral palsy, it nevertheless renders him permanently disabled and is a significant injury.

[3] The plaintiff alleges negligent intrapartum care on the part of the staff who attended to her during her hospitalization, latterly at the Dora Nginza Hospital where BM was delivered, and in the few days preceding his birth, at the Livingstone Hospital.

[4] In essence the plaintiff's case is premised on BM having suffered a hypoxic ischaemic brain injury during a prolonged and stressful labour that was due to the claimed negligent care. The injury is validated by an MRI brain scan showing a picture consistent with an acute intrapartum event compatible with the allegations of substandard management during this interlude.

[5] The plaintiff exhaustively pleaded a history of relevant events relating to her care (or alleged lack of it) at each hospital and several grounds of negligence.[1]

[1] Although the particulars of claim are drawn on the basis of 3 separate distinct claims, at the commencement of the trial plaintiff's counsel indicated the intention to pursue only plaintiff's claim founded in delict, described as claim B in the particulars of claim.

Both aspects of the history relied upon by her, and the claimed negligence, were denied by the defendant in her plea.

[6] Apart from denying the essential elements of the plaintiff's claim, the defendant differed with the plaintiff about dates, her supposed presentation at each hospital, the nature of the care administered to her (or not), the maternal and foetal well-being (or not) of her and her then unborn foetus, the nature of and whether her labour was prolonged (or not) and, significantly, whether BM was born in a poor state of health and exhibited signs of neonatal encephalopathy ("*NE*") at birth, which is one of the accepted early markers for cerebral palsy sustained intrapartum according to a professional Consensus Statement.[2]

[7] Although on the pleadings BM's condition of cerebral palsy was not acknowledged, the defendant yet denied that she was responsible for his "*condition*". In giving a context to her denial of causal negligence, the opinion formed by the experts relied upon by her to assist the court in getting to the bottom of the unfortunate outcome, is that BM probably suffered the brain injury which he did as a result of a meningitis infection, thus contending for a non-negligent cause for his condition.

Background:

[8] The plaintiff was 25 years old when she delivered BM who according to antenatal records was expected to be born on a date in September 2007. This was her first pregnancy.

[2] Neonatal Encephalopathy and Neurological Outcome, 2nd Edition, Report on the American College of Obstetricians and Gynaecologists Task Force on Neonatal Encephalopathy (2014).

[9] She is HIV positive, a factor that was confirmed by a blood test undertaken in the second month of her pregnancy at the Marselle Clinic in Bushman's River where she initially booked to receive antenatal care. The plaintiff in her testimony clarified that her HIV status (which she has lived with since 2002) was clearly endorsed on her "*green passbook*" (clinic card) that accompanied her after her initial visit at the Marselle Clinic on 15 January 2007 once she relocated to Gqeberha, from whence she continued to receive antenatal care at the "*Dwesi Clinic*".[3]

[10] By all accounts her antenatal care was unremarkable and she was relatively healthy despite her HIV status.

[11] On 13 August 2007 she was collected by ambulance and taken to DNH at her request because she was in abdominal pain and had passed vaginal mucous. She recalls that this happened on the same day as her last routine visit at the "*Dwesi*" clinic that morning. She was assessed and told that she was not yet due to deliver and was thus sent home. On 14 August 2007 she was still in continuous pain and reported again to the DNH via ambulance.[4] She was examined vaginally and similarly returned home on the basis that her baby was not yet coming. On 16 August 2007, with worsening pain, she hired private transport to the Livingstone Hospital where she was admitted *inter alia* with high blood pressure. On the afternoon of the 17 August 2007 she was transferred to DNH[5] whose staff took over her obstetrical management. Although the reason for the transfer was flagged as being due *inter alia* to her raised blood pressure, she was only given medication for her gestational hypertension at the DNH from 18 August 2007.

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[3] Probably Kwadesi in Gqeberha.

[4] No records were available in respect of these visits.

[5] It is common cause that DNH is a tertiary hospital that *inter alia* deals with complicated obstetric cases. According to the applicable Guidelines for Maternity Care in South Africa issued by the Department of Health, 3rd Edition, 2007, ("*Maternity Care Guidelines*") it would have been recognized as a Level 3 hospital at the time.

[12] By 16h00 on 18 August 2007 she was diagnosed as being in prolonged labour (latent phase). Despite this no intervention was undertaken to birth BM by caesarean section despite her also being preeclamptic and HIV positive. Her labour was instead augmented with Syntocinon with trial of labour continuing even though there were indications of foetal distress.

[13] BM was ultimately born at 15h35 on 19 August 2007 by a difficult forceps delivery after the delivery of his head was delayed in the plaintiff's birth canal.[6]

[14] By all accounts he was born in a "*significantly compromised neurological condition*".[7]

The plaintiff's testimony:

[15] The plaintiff related her own experience of the delivery and focused in her testimony on the key events leading up to it and how BM presented at birth. It is necessary to advert to aspects of her testimony (both relating to the management of her care and BM's condition) to give a context to the opinions offered by the experts and to close some of the gaps left wanting in the records of DNH that were self-evidently deficient and mostly unreliable.

[16] The fact of the plaintiff being HIV positive was a known entity. She was first diagnosed in 2002 but despite this was healthy and not taking any medication for her condition. She was advised upon her first visit to the Marselle Clinic that

[6] This is evidenced by a note in the DNH Maternity Case Record ("*MCR*") to the effect that the person making the entry "*struggled to apply forceps*".

[7] Dr. Kara who testified on behalf of the plaintiff described BM's condition in his manner. Prof. Bolton who testified on behalf of the defendant agreed with this assessment adding that: "*I could not have said it better myself*".

it would not be necessary to take any medication during her pregnancy for the disease but the anticipation was that she would receive Nevirapine at the time of her baby's birth.

[17] She learnt upon her admission at the Livingstone Hospital that she had high blood pressure although it was the abdominal pain that had driven her to DNH twice before to seek care. She could not recall how they treated her for this but remembers being especially informed as to such a diagnosis. The hospital staff put a red sticker on her file and told her that she was a high risk, hence the need to transfer her to DNH. She acknowledged that the staff at Livingstone Hospital had put a strap around her stomach to check her baby's heart rate[8] which they did "*all day*". They were however more focused on her raised blood pressure.

[18] Her recall, upon being transferred to DNH and after being initially examined, is of being dizzy but told to ambulate with about eight other expectant mothers. She was not assigned to a bed, but fellow patients recognized that she should be lying down and made a place for her on one of the three beds available in this ward, which she could at best describe as a waiting room although not an admission waiting room.[9]

[19] She slept until the Saturday morning when she was again examined. This was around 9 – 10am. During this assessment some water ran out on her legs which she thought was urine, but the examining doctor told her that her water was breaking. She says that was transferred to the labour ward at 6pm.

[8] It was assumed that this was monitoring by cardiotocograph ("*CTG*"), a technique used to monitor foetal heartbeat and uterine contractions during pregnancy and labour.
[9] This was possibly a triage ward.

[20] Until her transfer to the labour ward she waited in a chair. According to her neither she nor her then foetus were checked at all and her move to the labour ward was coincidentally contemporaneous with another patient's whose water was said to have broken, a nursing sister complaining at the time: "*Yoh it is the two now and I must take the other one and go to the labour ward*". Here for the first time she was given a bed in a ward with the other patient. They were also asked individually about their status. She informed the nursing sister that she was HIV positive.

[21] Her next recollection was of waking up the following morning on Sunday 19 August 2007. A nursing sister was shouting at her saying: "*Do not sleep, you must wake up and walk because the head of your child…must come out. If you are lying down like that your child is not coming out*".[10] She attempted to reason that she was dizzy yet was urged to "*wake up and walk*". She tried to walk. Her next recall is of the nurse telling her conversely not to push because her baby's head was coming out. At this time she claims that she started to fit. This was around 4pm. She described the sensation as being conscious but hearing voices from afar.

[22] She was assured that she should not panic. An oxygen mask was put over her mouth and a doctor was called to help her. Three doctors came to her bedside. One of them pushed on her stomach just below her sternum.[11] Another arranged her legs in stirrups and the third one put something cold in her vagina. BM was delivered. He was momentarily given to her but was instantly spirited away to the nursery. She heard them say as if their voices were far away: *"Nursery, Nursery, Nursey"*. She gleaned that her baby was a boy. In the brief moment of holding

[10] The plaintiff expressed herself quite quaintly in English which is not her first language.
[11] This action suggests the use of fundal pressure which is controversial. It was one the grounds of negligence relied upon by the plaintiff in her particulars of claim but its significance (or not) was not a focus in the expert testimony.

him she noticed that he was not crying. She was brusquely informed after a while that the nurse would be stitching her because the doctor is "*leave it like that*" which is when she learnt that they had made a cut in her vagina to get her baby's head out.[12]

[23] Asked how she knew she had fitted during labour she explained that a nurse had told her afterwards that she had fitted when the head of her baby was coming down.

[24] Later that afternoon in the ward she fitted again whilst coming from the toilet and was similarly informed by the nursing sisters that she had had a fit. On this occasion she had felt weak and dizzy and was cautioned to rather remain in bed.

[25] She only saw her baby again on the ensuing Monday, 20 August 2007. In the nursery he was in an incubator. It was then that the nurse told her that she had had a fit during the delivery and added that it was her fault that BM had also fitted.

[26] BM had a tube coming out of both his nostrils. She could only hold him for brief moments out of the incubator. He could not latch on when she tried to feed him at her breast. He was otherwise fed by tube.

[27] She related that she had seen a special doctor at BM's cot who told her that he was not breastfeeding, or at least could not suck, because of something inside his mouth on the tongue that had to first be cut.[13]

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[12] It is common cause that an episiotomy was performed.

[13] No clinical justification emerged from this explanation given to her, leaving the impression that the full extent of her baby's condition was probably withheld from her.

[28] BM remained in the hospital nursery for ten days. He was discharged from the nursery together with her. She remembered that this was on the same day that she received a blood transfusion.[14]

[29] She noticed soon after his birth that BM was different than other children and not meeting the regular milestones in growing up. He was for example two years and nine months old before he started to walk and that was with the dedicated assistance of a Dr. Baker at a hospice in Hamburg who administered physiotherapy to him to get him mobile.

[30] It is opportune to state here that although counsel for the defendant in closing submissions contended that the plaintiff was a poor witness, this was certainly not my impression. She was exhaustively cross examined in English (which is not her primary language) and consistently maintained a narrative that she had earlier repeated to the specialist experts who she had consulted with in the course of litigating in the same minute detail. Although certain minor aspects escaped her recall, not surprisingly given BM's delivery fourteen years before the trial, her version of the material events was also coincidentally corroborated by notes in the DNH Maternity Case Record ("*MCR*") and other medical records some of which were only made available to her legal team after she had testified. Moreover her account is neither improbable nor implausible and the unique vignette, for example, of her having "*fitted*" (which detail was not written in DNH's MCR) is entirely consistent with her having been diagnosed with preeclampsia at least in circumstances where she was not pre-emptively given any medication to avoid seizures after her blood pressure became seriously elevated. A random note alluded to by Dr. Chimusoro, specialist obstetrician and gynaecologist who testified on her behalf, also co-incidentally referenced an

[14] The records reveal that this must have happened on 28 August 2007.

isolated entry dated 17 February 2017 in BM's Marselle Clinic card (made before the issue of summons) in which it is deduced, after a history taken contemporaneously from the plaintiff, that "*Mom had eclamptic seizures at delivery*". I mention finally that the plaintiff's insistence of this minutiae is rendered credible by her amplification that subsequently she was advised by a nurse that her fitting had caused the injury to BM, a disclosure which made her very emotional at the telling when she gave her testimony.

The medical records:

[31] It is necessary to give a brief exposition of the medical records that were discovered by the defendant and to correlate these with the plaintiff's evidence. The records, such as they are, also constitute the foundation for the expert opinions tendered in this matter.[15]

Antenatal care:

[32] Firstly the records of the Marselle Clinic carried forward to the "*Dwesi*" Clinic reflect that regular antenatal visits were maintained by the plaintiff and that nothing abnormal was noted to suggest any problems with her pregnancy. The uterine growth evolved as it should have and there was no suggestion of any earlier hypertension. The plaintiff's last menstrual period was loosely reflected as "*11/06*" but her expected delivery date was anticipated towards the end of September 2007. This is obvious from the indication recorded at her last routine visit that she should come again ("*TCA*") on "*27/09/7*".

[15] The records were admitted into evidence on the customary basis that they are what they purport to be.

[33] Despite the wrong indication aforesaid, the staff at DNH did not believe that the plaintiff's baby was premature when she presented to deliver him.[16]

Livingstone Hospital (16 – 17 August 2007):

[34] The single page of clinical notes from the LH (which reflect a "*PTO*" at their foot but with nothing following) confirms the plaintiff's admission there on 16 August 2007 at "*34 weeks*" (*sic*).[17]

[35] They note that the plaintiff had a "*history*" of lower backache and abdominal pains since 16h00 that day and that she had been treated "*at DNH for the same problem with oral antibiotics. Course finished*". (This coincides with the plaintiff's testimony that she had presented herself at the DNH with

[16] Dr. Janowski, paediatrician, who gave evidence on behalf of the defendant, believed that the plaintiff was in fact "*post-dates,*" that is beyond her expected date of delivery upon admission to the LH. Dr. Kara, specialist paediatrician who testified on behalf of the plaintiff, assumed a delivery date of 18 August 2007 and opined that the plaintiff was post term. By all accounts the expectation that the plaintiff should come back on 27 September 2007 to birth her baby was unrealistic. The gestational age was fortunately responsibly reassessed by a member of staff at the DNH upon the plaintiff's transfer there to be approximately 38 weeks based on the fundal height of the foetus. An ultrasound scan during her pregnancy would have put the uncertainty of her delivery date beyond the pale but unfortunately no scan was undertaken. It was not suggested that the plaintiff ought to have had a scan during her labour, but the criticism all round was that the gestational age assessed at the antenatal clinic was inaccurate. This may have been the reason why she was turned back by DNH twice before when she presented with abdominal pain on the basis that her baby was not yet due to be born.

[17] See footnote 16. The last entry in the antenatal records on the same date record a "*HOF*" of 34cm. This could perhaps have been misread as the gestational age by the person making the opening entry at the Livingstone Hospital. It is coincidentally evident from the same entry in the records of the Marselle Clinic dated 17 February 2017 referenced in paragraph 30 above that the plaintiff herself believed that BM had been born "premature" consistent no doubt with what she had been made to believe by the "*Dwesi*" clinic was her expected date of delivery.

abdominal pain twice before her admission to LH.)[18]

[36] They record an estimated gestational age of "*34 weeks*" (sic),[19] foetal movement felt, vaginal discharge and cervical os closed and uneffaced on the night of the 16th, but by 13h00 on 17th August 2007 allowing the tip of a finger in (without any contractions though), a single recording of a reactive CTG on the 13th, an indication of trace protein in the plaintiff's urine, and most significantly, a blood pressure that was rising (from 152/83 on admission to 159/94 upon her transfer to DNH the following day). The last entry recording the plaintiff's leaving for DNH by official transport at 16h30 indicates the reasons for this as being "*slow progress and raised blood pressure*".

DNH (17 August 2007):

[37] A single page of clinical notes appears in the DNH MCR for 17 August 2007. They acknowledge the plaintiff's referral for the said reasons at 17h20 with an opening blood pressure reading of 146/94 recorded and an indication that a medical officer is to see her.[20] They further note the first assessment of her by a Dr. Lamprecht at 18h00. The doctor puts the gestational age at 38 weeks (with

[18] The notes of this treatment did not surface during the trial. The plaintiff in giving the exposition of the dates may have been confused. She says that her first visit to DNH was on the same day as her last routine visit but on the antenatal records this date was cut off in the photocopying so could not be verified. (The original antenatal records were not provided). There was also a suggestion that between her second visit to DNH and her reporting to LH a week had passed. This too could not be checked with reference to formal records which ought to exist but were not made available by the defendant. The earlier DNH records were certainly relevant given the plaintiff's claim (at least in the particulars of claim) that the hospital staff were negligent in failing to diagnose that she was post term and suffering with hypertension when she presented there with lower abdominal pain not once but twice before going to LH. The fact that she took private transport to get to LH after having been treated dismissively by the staff at DNH who informed her that it was not yet her time to deliver indicates the level of the plaintiff's concern that something was wrong with her pregnancy as well as her determination to have done something about it. It was thus an important aspect of the history for the hospitals treating her to have taken into consideration. (Dr. Janowski coincidentally believed that the comment about the plaintiff's prior treatment related to the two occasions during her pregnancy when she was treated with antibiotics by the "*Dwesi*" clinic.)

[19] See footnote 16.

[20] Dr. Chimusoro clarified that a normal blood pressure would be 140/90 and that anything outside of this range would make the pregnancy a complicated one.

reference to fundal height because the plaintiff was unsure of dates). She is assessed to be in "*early latent labour*" (mild contractions are palpable) with gestational hypertension ("*GPH*"). The plan indicates that she is to have bloods taken and is to be put on CTG with a review after 6 – 8 hours. A random note at the foot of the page indicates that she is transferred *"to the side wards*" at 17h00.[21]

DNH (18 August 2007):

[38] As testified to by the plaintiff, there is no assessment of her again until the next morning on 18 August 2007.[22] At 07h00 there is a question mark indicated in the notes over whether she is in early latent labour, but she is found to be 1cm dilated with her membranes intact. Blood results are interpreted for example haemoglobin of 7.5 G/dl (low – anaemic), white cell count was 12 (normal) and platelets 290 (normal). The urea and electrolytes were normal. Uric acid was 0.19 (low). Liver enzymes were not suggestive of HELLP syndrome.[23] The plan is to repeat CTG and to start anti-hypertensive medication (evidently only if her blood pressure remains on the increase). (Notably there is no indication of what the blood pressure is at this stage.) There is no rupture of her membranes yet at this point.

[21] The nature of this ward or relevance of the transfer was not given any context. If it was for special management one would imagine that the notes would confirm as much but since according to the plaintiff's testimony she was not given any treatment there overnight it follows logically that she was not sent there for any meaningful objective. Dr. Chimusoro formed the view that it appeared that she had been cast to one side. Further and in any event she was only scheduled for a review in 6-8 hours which was an unacceptable wait given her raised blood pressure and the suggestion recorded by LH that she was slow to progress in her labour.

[22] Her alarm expressed at the fact that she had received no treatment overnight after her transfer from LH with a specific concern having been noted that she was hypertensive was not misplaced. The examining doctor upon her arrival had also indicated that her case should only be reviewed within 6 – 8 hours. As it turned out 13 hours lapsed before she was checked again.

[23] These results were gratefully expounded upon by the experts. The tests obviously implicate the battery of tests that would monitor the concern of the plaintiff's raised blood pressure. HELLP syndrome is a complication of high blood pressure during pregnancy. The acronym stands for haemolysis, elevated liver enzymes, low platelet count.

[39] At 16h00 she is assessed by the same Dr. Lamprecht again who examined her upon her admission. She is still found to be 1cm dilated "*since 16/08/07*" and complaining of labour pains. The gestational hypertension is again recognized and there is a comment that there are "*contractions on CTG*". There is a random note that ward "*BG 3 will not take patient*" which gives credence to the plaintiff's testimony that she was not in a ward but left in a chair until that evening.[24] A diagnosis of "*prolonged latent phase*" is made by the doctor in addition to the hypertension. The plan is to monitor her by CTG, to give her 50mg Pethidine (notes endorsed to say "*given*"),[25] and to transfer her to the labour ward. Augmentation is proposed (possibly with "*Miso*")[26] and the doctor acknowledges a discussion with a consultant, Dr. Blignaut, in this regard. The concluding remark is that the plaintiff is having regular (painful) contractions.

[40] At the foot of the page, but with no time indicated, Dr. Blignaut ostensibly has recorded his/her review of the plaintiff. She is noted to be asymptomatic re "*IE*" (imminent eclampsia). Blood pressure is recorded as 178/105.[27] The cervix is 4cm dilated. [28] Fully effaced. The presenting part is at 3/5 (above brim). Her membranes are noted to be intact. The plan is to sedate her intravenously and see her in two hours. CTG is to be repeated. 75mg of Pethidine is to be given immediately.

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[24] It is worrying that a ward would not take the plaintiff and one is left to surmise that this might have been because of her HIV positive status.

[25] The medication chart in the MCR confirms that Pethidine was dispensed on the 18th but no time is indicated.

[26] This is explained as a reference to Misoprostol which Dr. Chimusoro said was being used off licence for induction or augmentation and was not recommended by its manufacturer for use in pregnancies with viable foetuses. Dr. Janowski, specialist obstetrician and gynaecologist, who testified on behalf of the defendant pointed out that the DNH MCR does not support any suggestion that the pharmacy ever dispensed it.

[27] The blood pressure reading here coincides with the entry made at 20h10 so probably times the review entry by Dr. Blignaut to have been made at this juncture.

[28] According to the 2007 Maternity Care Guidelines this would have heralded the start of the active phase of labour for the plaintiff.

[41] To return to the period in-between the plaintiff's morning and late afternoon assessments, there are three entries made.

[42] The first at 08h53 records a blood pressure reading of 155/105, an assessment of no ROM (rupture of membranes) and a 1cm dilation. The plan (with regard to the diagnosis of GPH) is that she is to be admitted into ward BG 3 (which ward did not take her and with no explanation forthcoming from the defendant as to why that was the case), to be given Aldomet 500mgs,[29] to do CTG and, *"if reactive*[30] *to administer Pethidine 75mg stat*". A urine dip stick is also indicated in the plan.

[43] A note at 10h55 records that the plaintiff is "*attached to CTG FHR is 150 bpm. Cervix 2cm dilated* (written over an obvious "4"). *Cervix thick. Membranes ruptured at 10.10*" (the ten minutes written over a figure "*30*").[31]

[44] The next consequential entry made at 13h38 (before the plaintiff is reviewed again by Dr. Lamprecht two hours and twenty-two minutes later) surprisingly records that she is 1cm dilated again. Again a diagnosis of GPH is

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[29] The expert evidence suggested that this is standard treatment for gestational hypertension, well at least for moderate cases and that the medication is slow acting. Dr. Chimusoro explicated that Adalat was more rapid acting. He thought it more appropriate that the plaintiff be treated as if her blood pressure was veering toward the severe range. He noted that the highest he has seen was 150/105 which was still in the "*upper moderate range*" but was concerned by the "*unconfirmed issue*" in the clinical notes that she had fitted. The medication chart in the MCR coincidentally references a script for Adalat, also dated 17 August 2007, if a certain (illegible) contingency materializes, but it does not appear that this medication was in fact given to the plaintiff.

[30] The evidence established that a CTG referred to as reactive is one that suggests no compromise to the foetus after a contraction. It is reassuring in its nature.

[31] This entry was noted by Dr. Chimusoro to be inconsistent with the next entry. Also confusing is the indication that the plaintiff's membranes were ruptured because elsewhere it is noted that the artificial rupture occurred only on the 19th. The plaintiff did suggest in her testimony that around this time some waters leaked out which she was informed meant that her water had broken. Dr. Chimusoro explained that this may have been hind waters but the timing was important because there was a risk to her foetus by any kind of rupture which would have meant that it was no longer in a sterile sac. Indeed it would by the breach of its safe environment from that point have been rendered vulnerable to ascending infections in the plaintiff's vagina, and to the risk of transmission of the HIV virus. He proposed that two steps were not taken that would have made a difference. The first is that the plaintiff should have been treated with antibiotics (assuming a pre-screening for infection(s)) and the second is that counting from that moment, augmentation should have commenced to get the baby out or put another way, the decision to deliver ought to have been contemplated right then and there.

reflected (but with no reference to gestational proteinuria hypertension) and a plan is framed to admit her to the ward that would not take her.[32]

[45] The plaintiff was ostensibly admitted to the labour ward at 20h10 according to the notes, denoted by an appropriate entry that indicates that she is having strong and regular contractions and is attached to CTG with FHR of 120-160 bpm. Dr. Chimusoro especially noted his concern regarding the fact that the plaintiff's blood pressure at this point was severely elevated and needed consideration for "*MGS04*" to prevent seizures.[33] Her pulse was 109. (It is fair comment that nothing is written to indicate what steps, if any, were taken contemporaneously with this elevated BP reading.)

[46] Co-incidentally the remark that the plaintiff is having strong and regular contractions may have been made at 23h45. If it was intended to concern events just before midnight (and that this is possibly the time when she moved to the first stage of labour), the person making the entry has written it over the 20h10 entry which is when the plaintiff moved to the labour ward with the markedly elevated blood pressure reading. (It is disconcerting that a reader cannot really fathom when exactly the plaintiff's first stage of labour commenced.)

DNH (19 August 2007):

[47] At 01h45 an entry is made that "*CTG reactive (Signed). Pethidine 75mg given intramuscularly as ordered.*"

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[32] There are two conflicting times indicated in respect of this entry. The second time written opposite the same entry is *"23:45"*. The handwriting is different but what is written also encroaches on to the entry of Dr. Lamprecht and overwrites his/her signature suggesting that it may have been made after the fact and was squeezed into a gap between entries. (Prof. Bolton does not reference these entries in his report which may suggest that his copy of the MCR did not have these two entries on the page at all.)

[33] This is a reference to Magnesium Sulphate (commonly known as Epsom Salts) which Dr. Chimusoro explained is the standard international medication used to prevent fits in pregnant women when they are in labour or when they get hypertension. He emphasized the point that it is given preventatively and not when the patient is fitting already.

[48] At 03h45, randomly written at the foot of a blank "*Forceps Delivery/Vacuum Extraction*" page, is "*Rpt CTG – FHR 120 – 140 Strong contractions"* and *"(P) RPT. GPH bld*."

[49] At 5h10, written on the same page ostensibly by the same hand is: "*PV CX fully effaced. Os 4cm dilated. BP 129/106, membranes intact, AROM done - clear liquor."* The plan indicated is to do *"5U of Syntocinon infusion stat and a CTG at 6h00".*[34]

[50] At 6h00 an entry is recorded that the plaintiff is "*attached to CTG machine*" and that the FHR is "*ranging between 135 – 129 bpm*".

[51] At 6h15 it is acknowledged that she is given 75mg of Pethidine intramuscularly for pain.

[52] At 8h10 there is a substantial entry to the effect that the plaintiff *"is sleeping, no contractions, (having short contractions on augmentation) PV – fully effaced, still 4 -5 cm dilated, caput +, clear liquor draining, CTG reactive."* The plan indicated is to do GPH Bloods, short/10 U duration of augmentation, review in two hours – if no progress then for caesarean section ("CS"). Prepare for CS in the meantime. Discussed (handover) with Dr. Blignaut – For CS. The Blood results are interpreted and then follows a review by Dr. Sipuka with a plan to do a short trial of augmentation.[35] CTG is noted to be reactive. A review in 2 hours is expected to happen.

[34] Dr. Chimusoro opined that whilst on the Syntocinon infusion the plaintiff ought to have been on *continuous* CTG.
[35] Dr. Chimusoro remarked that the Syntocinon infusion had been running from 5h10 already.

[53] A further entry appears at 8h45 recording that the dilation has progressed to 6cm. There is a caput++, with no moulding. CTG is said to be reactive. The plan is to give Pitocin 10 U[36] (as a bolus or as an infusion in the drip),[37] to empty bladder, and review in 2 hours.

[54] At 8h50 a note indicates that the plaintiff's bladder has emptied (100ml) and that she has felt foetal movements.

[55] At 09h30 she is "*attached to the CTG. Heart ranges between 100 – 160 bpm. Foetal movements felt by mother*". The next annotation is "*nursed left lateral position 02 per mask*".[38] No implication is provided for this significant event.

[56] At 10h50 a review is recorded to the effect that the plaintiff's cervix is fully effaced and 8cm dilated. A caput of ++ is noted. Clear liquor. CTG reactive. The plan is to continue augmentation. Repeat CTG and review in an hour.

[57] At 11h36 the plaintiff is noted to be attached to CTG with foetal heart ranges between 100 – 160 bpm.[39]

[58] At 12h30 it is noted that the plaintiff's cervix is 9cm dilated, and well effaced. A caput of ++ is observed. The plan is that the patient is to "*sit up*" and to review her case again at 13h30. Nothing is said about the foetal condition despite the drop in range mentioned in the last note an hour before.

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[36] Dr. Chimusoro suggested that Pitocin and Oxytocin were one and the same stimulant.
[37] In his view this was not the correct way to administer the stimulant as something squirted into the plaintiff's vein. Instead it is supposed to be a slow infusion.
[38] The evidence revealed that this CTG and the one referenced in paragraph 57 above were non reassuring. Dr. Chimusoro's opinion was that the event recorded at 09h30, taken together with the drop in the foetal heart rate range between "*100 – 160*", should have raised concerns that there was foetal distress at this juncture.
[39] See footnote 36. This is the second indication according to Dr. Chimusoro's opinion that the foetal condition was non reassuring.

[59] The next entry records a review only at 15h00. The plaintiff is by now noted to be fully dilated, having a caput ++, no moulding, and the head is said to be at the "*spines*". CTG is said to be reactive. Bladder is empty. The reviewing clinician questions whether there is a malposition but affirms that the pelvis feels adequate. The plan is to continue augmentation if undelivered in 30 minutes for CS.

[60] At 15h20 an entry is made to the effect that an episiotomy is performed and that there was a struggle to apply forceps. The note goes on to say that Dr. Sipuka was called and at 15h40 a live male is recorded as having been delivered. APGAR scores of 5/10 and 8/10 are written down.[40]

[61] A separate entry is made at 15h35 on a different page regarding delivery. It confirms the forceps delivery of a live male infant however with APGAR scores of 1/10 and 3/10 and seen by Dr. Titus. Dr. Sipuka appears to have signed "*pp*" on the latter's behalf.

[62] At 16h05, a retrospective note is made (evidently by Dr. Lamprecht) regarding the plaintiff's labour and the condition of BM as follows:

> "Called to labour ward. "Flat baby"
> Apgar's 1/ 10, 3/ 10.
> Forceps delivery (with) delay + difficulty of delivering head.
> Analgesia (Pethidine) given in morning only.
> Initial assessment
> Apgar 6/ 10
> - good pulse, HR < than 100
> - no spontaneous respiration

[40] Dr. Janowski was the only specialist to suggest that these scores might be authentic whereas everyone else agreed that they were unrealistic and inconsistent not only with what the nurses had recorded but also with the fact that the baby was said to have been flat and only resuscitated after about 15 minutes. In my view a discussion of them is unnecessary. If the defendant wished to make out a case that the doctor's inconsistent entry should have any cogency she should have adduced this person's testimony.

- some flexion
- peripherally pink
- Baby bagged. 0.4mg Naloxone given IM[41]

Baby responded slowly; Nasopharynx suctioned = clear fluid
Spontaneous respiration only after +- 15 minutes
O/E:
+++Caput
pink – (no signs) of respiratory distress
No obvious (signs) of dysmorphism
Baby cold to touch"

Postnatal records:

[63] It is unnecessary to go into minute detail regarding BM's condition after birth given the parties' agreement that there were classic indicators for a diagnosis of NE. It is relevant to mention however that Dr. Lamprecht immediately recorded an assessment after delivery of birth asphyxia and *"? HIE"*.[42] The doctor further references cycling and lip smacking as noted by the sisters and that BM is feeding via nasogastric ("*NGT*") tube. On examination the baby is said to be lethargic, pink, and comfortable in the room air. There are no signs of respiratory distress.[43] The assessment at this point is of a Grade II HIE and he is to start on "*Phenobarb*" (probably Phenobarbital).[44]

[64] It is also noted in the clinical notes that BM had convulsions on the 20th, after which a cervical spinal fluid ("*CSF*") sample was taken.

[41] Evidence suggested that this medication would be given to reverse the opioid effect of the Pethidine given. It is hard to make sense of the entries regarding when Pethidine was dispensed and how much exactly. The medication chart in the MCR indicates a single dose of 75mg stat given on the 18th and nothing else. It was supposed to have been given 8 hourly. Dr. Chimusoro proffered his view that the plaintiff was probably not sleeping on the morning of the 19th but that she was "*woozy"* from Pethidine because by then she was already in the active stage of her latent phase of labour and 4-5 cm dilated so would hardly have been naturally sleepy or inclined to want to sleep at this juncture.

[42] This assessment was repeated on the 20th.

[43] Dr. Kara testified that it was hugely unlikely that there were no signs of respiratory distress if there was also at the same time "*subcostal recession*" recorded by the nurses upon BM's admission to the ward. See par [66].

[44] This is a drug used for anti-seizure management.

[65] On 22 August 2007 a provisional diagnosis of meningitis was made which was repeated over the next few days and pursuant to which BM was treated with an antibiotic for 9 days.[45] I deal with this below.

[66] It is also worth repeating the initial nursing note made at 16h40 after BM's admission to the ward, which is as follows:

> *"A live baby boy born by forceps delivery. Flat. Apgar 1/10, 3/ 10. Admitted in for observations. On admission baby …colour pale Caput +++ cold and clammy to touch. Subcostal recession but not in distress. Lethargic with some poor muscle tone- HGT 7.9 G/dl. Put into a closed incubator attached to the... and SA O2 .94- 99% on room air. HR 133 beats/ minute RR 58 beats per minute. Stable awaits review by doctor. Oxygen not administered baby stable."*

Miscellaneous other entries in the DNH MCR:

[67] Other indications appear in the Newborn Care Record which confirm that Dr. Sipuka delivered BM at 15h35 by forceps. The birth weight is 3.80kg, length is 53cm, head circumference 36cm. The APGAR here is also reflected as 1/10 at one minute and 3/10 at ten minutes. For the first time the MCR notes the plaintiff's positive HIV status but other vital information about the labour and resuscitation, distress indicators and neonatal treatment of the baby is lacking on this form. The part especially indicated for APGAR scoring has been left blank and unsigned.[46] Under the discharge part "*check and plan*" of the same page (with no problems noted and no plan indicated) the indication is given, contrary to the plaintiff's evidence and inconsistent with what has become known about BM's condition after the fact, that the baby is "*breastfed*" and "*feeding well*".

[45] By all accounts this was for a shorter than usual period of 14 days applicable at the time.
[46] See page 51 of the medical records, Bundle B1.

[68] The Summary of Labour form has some details recorded, some of which I have already related above. Only two features of the APGAR rating criteria have been scored but indicate a total of 1/10 at one minute and 3/10 at five minutes.

[69] The first stage of labour is said to have commenced on 19 August 2007 at 00h03 for a duration of 14h25, second stage on 19 August at 14h30 (for 1 hour 5 minutes) and the third stage on the same date at 15h35 – 15h45 (for ten minutes). The total duration of labour is given as 15h40. These periods are self-evidently incorrect (and inconsistent with other clinical notes) and Dr. Chimusoro went so far as to suggest (not unfairly in my view) that the person recording them was probably trying to gloss over the prolonged labour.

[70] The forceps delivery report (elsewhere indicated as being a difficult procedure) is completely blank and leaving one to surmise what about it caused the concern for the staff involved and how they dealt with whatever challenges they were faced with.

[71] The partogram is similarly incomplete but reflects supposedly normal foetal heart rates at every half hour from 8h10 to 15h40 on 19 August 2007 (the only period chartered),[47] the last one and time coinciding with the comment "*delivered*" as if the writer (all in the same handwriting) knew when he/she started recording the data that the birth would happen within half an hour of the penultimate check. The contractions shaded in on the partogram are designated as having been moderate in nature contrary to clinical notes appearing elsewhere suggesting strong contractions.

[72] Dr. Chimusoro ventured his view (again not implausibly) that it looked like a person had in a single sitting sat and wrote all the times in on the partogram at

[47] There are 16 entries recorded.

once. He pointed out that the stroking of the purported contractions (the shading-in referred to above) contradict what the notes say as do some of the supposed heart rates recorded on the graph. One looks in vain for example to find on the partogram the two so called non reassuring CTG reports referenced in the clinical notes at 9h30 and 11h36 in that interlude.

[73] There is indeed in my view nothing in this vital document that a court is able to rely upon that gives comfort that it is authentic and responsibly made.

The lack in the medical records and the perspective offered by DNH:

[74] The clinical records kept by the DNH were appallingly deficient and haphazard. No original documentation was produced in evidence. The copies passed off as true copies of the originals bear annotations or comments written all over them. As I have already remarked, the records of the plaintiff's assessments at DNH before she presented herself at LH were not made available at the trial and some notes only came to the party after the trial had commenced.[48] Several critical templates were not completed at all. Some reflected bare entries here and there. Clinical notes were scribbled indiscriminately in places where one would not look, foiling the objective of note keeping which is to maintain a continuous record of critical events both to appreciate what has gone before and to plan going forward on the premise of a reliable consequential thread especially at handovers. Some entries were not timed. In the one instance I have already referred to above there was evidently an overwriting of the extent of the plaintiff's cervical dilation so obviously out of sync with the foregoing and subsequent entries. If not careless the only inference to be drawn from this is that the writer was deliberately obfuscating the reality or seeking to paint it in a better light after the fact.

[48] The two pages of notes of the 18th, for example.

[75] Another critical discrepancy or confusion which arises from the notes is whether there was a rupture of the plaintiff's membranes as early as 10h10 on 18 August 2007 already given the inconsistency with a later note as to the supposed defining moment of AROM indicated. This detail is important because of the plaintiff's HIV status and danger of ascending infections and transmission of the virus to the baby. (According to Dr. Chimusoro's evidence the standard operating procedure at the time would have required the delivery to have been within 4 hours of the rupture because of the plaintiff's positive HIV status even if the early entry related to hind-waters only coming down.)

[76] I am further satisfied that the information on the partogram was probably reconstructed as a reality after the fact. This was ostensibly to give a picture of what supposedly pertained during the plaintiff's labour and to create an impression of compliant step-taking and recordkeeping. This can in my view be plausibly inferred because the on-the-half-hour times and foetal heart rates written down do not align with separate entries written down in the clinical notes as to foetal heart rate checks or CTG interpretations. The strength of the plaintiff's contractions are also as I have said above inconsistent with what is written in the clinical notes.

[77] Odd CTG tracings were made available but the full picture was left to everyone's surmise. Intermittent notes appear regarding fetal heart rate monitoring in a highly complicated labour in circumstances where the foetus was supposed to be subjected to continuous surveillance by cardiotocography. The trend of her blood pressure readings was evidently not captured on a graph anywhere.

[78] Not only are the records such as were made available tainted with suspicion, but the staff fell horribly short of their legal obligation to keep a proper maternity case record and/ or to have ostensibly taken every measure they were supposed to take in the management of the plaintiff's labour at the requisite intervals, which steps were consequently required to be reported on in the official maternity case record.

[79] If I accept the plaintiff's evidence that she fitted, for example (as I do since it appears plausible and consistent with all the features of her labour taken wholistically), this vital information was not recorded in her MCR. There is further an absence of her vital statistics taken at each regulated interval such as her blood pressure, this despite her gestational hypertension being one of the most serious risk factors that threatened her health and life as well as the well-being of her foetus going in to labour.

[80] Despite every indication being that those who were involved in the oversight of the plaintiff's labour and who could have filled in the obvious gaps were available to testify, the defendant chose to present her impression of what went down in this critical period solely with reference to the testimony of the experts who reviewed the inadequate or slanted MCR on her behalf. How these notes could have been defended by Dr. Janowski as reliable in showing a supposedly properly managed labour is frankly concerning.

[81] Mr. Kincaid who appeared on behalf of the plaintiff appropriately drew my attention to the dictum of Lord Brooke in the matter of *Ratcliffe v Plymouth & Torbay Health Authority*,[49] cited with approval and applied in *M obo M v MEC*

[49] [1998] EWCA Civ 2000 (11 Feb 1998) at para 48

Health and Social Development; Gauteng Provincial Department,[50] in which the following realism is expressed:

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

[82] I am grateful to all the experts who assisted in making sense of the entries that were made in the plaintiff's hospital records put at the court's disposal.

Issues for determination:

[83] As is the expectation in matters such as these involving a review by specialist experts of the medical records and data giving flesh to the relevant clinical setting, some of the points of difference raised in the pleadings were resolved by the time the trial commenced. Certain basic premises were accepted which were recorded in joint minutes of the radiologists, obstetricians, and paediatricians filed in preparation for and during the trial. I deal with these below.

[84] I also made an order at the onset of the matter separating quantum from merits and the trial proceeded on the issue of liability only.

[85] Although the parties seemed to have accepted in the Joint Practice Note filed pursuant to the case management processes that the obstetric management of the plaintiff was sub-standard leaving only the issue of causal negligence to be

[50] 2018 ZAGPPH JHC 513 (1 October 2018) SAFLII.

determined, it became evident to me from the tenor of Dr. Janowski's evidence that only aspects of the management were conceded to have been below the professional standard. For example it was accepted that the failure to have treated the plaintiff's GHT before 18 August 2007 was substandard but the defendant resisted the implication that this could have led to the unfortunate outcome. It was also accepted that the record keeping was exceptionally poor but again any causal connection with BM's condition thereby was eschewed. As for the augmentation administered and the decision not to deliver BM by caesarean section, the contention was that the standard of management adopted was perfectly in line with the Maternity Healthcare Guidelines applicable at the time and beyond reproach. The defendant differed with the plaintiff that her labour was prolonged or that there was any cephalopelvic disproportion. Whatever challenges came in her obstetric management as from the 18th, so the defendant's case went, these were met properly and professionally. Negligence in her treatment, such as there may have been, was in any event abjured on the basis that BM's condition more probably arose along a different pathway than intrapartum NE or, conversely put, the supposed hypoxia and ischemia were not the unique initiating causal mechanism for the neurological outcome in contention here.

[86] I set out below the allegations of negligence relied upon by the plaintiff in her particulars of claim to understand her starting point.

<u>The alleged negligent intrapartum care:</u>

[87] In summary the claimed basis for the defendant's negligence is premised on the failure on the part of the hospital staff to have pre-empted that BM would be especially at risk from suffering asphyxia and HIE during labour, a hypoxic-

centric event all on its own,[51] because of the high risk factors that pertained to her situation.

[88] Firstly, she was a *prima gravida* (cephalopelvic disproportion always being a concern with a first pregnancy) and is HIV positive.

[89] Additionally she ostensibly developed gestational hypertension which was not diagnosed by DNH at her earlier presentations when she reported complaining of abdominal pain.

[90] Although her hypertension was recognized at LH, she was not given any anti-hypertension treatment for her condition before being transferred to DNH.

[91] Even once transferred to DNH, which is a tertiary institution geared to deal with complicated cases, however, treatment for the added on risk factor was delayed.

[92] The failure to bring her blood pressure in check put the plaintiff at risk of suffering from fits.

[93] The hospital staff failed to take steps to deliver BM by caesarean section despite the indications for this step as a necessity being recognized by the staff because of the plaintiff's delayed labour.

[94] Instead her labour was augmented whereas the plaintiff was having strong, normal and regular contractions.

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[51] Each of the experts testified to the hypoxic strain placed on a foetus in utero during contractions. See further par [169] explaining the unique features and risks that pertain during labour especially.

[95] The hospital staff also failed to take note of documented signs of foetopelvic disproportion.[52]

[96] They failed to promote intrapartum resuscitation by causing the plaintiff to lie on her side rather than instructing her to sit up which would further have conduced to a hypoxic environment rather than ameliorating the challenge for BM's delayed delivery.

[97] They failed to take regular CTG recordings prior to delivery, missing a valuable opportunity to have detected foetal distress and to have acted upon it.

[98] They were ill-prepared, in the sense that there was no doctor on hand when her labour went awry warranting an ultimate delivery by forceps, and also failed in any event, when the plaintiff's foetus' condition deteriorated, to diagnose and react properly to meet this emergency.

[99] In general the contention is that the hospital staff failed to take reasonable precautions and to exercise the requisite level of skill as professionals to ensure BM did not suffer from birth asphyxia or HIE.

BM's condition:

[100] It is opportune to begin with what BM's condition is as this is not in dispute, and to reflect on the agreement between the parties concerning what the neuroimaging reveals about the injury sustained by him.

[52] This would have been with reference to the baby's size and weight and the fact that the plaintiff was probably beyond her date for delivery.

[101] According to the clinical assessment, he has gross motor function classification system (GMFCS) level 1 (*the grading looks at movements such as sitting, walking and use of mobility devices*); manual ability classification systems (MACS) level 1 (*the grading looks at handling objects*); and communication function classification system (CFCS) level 2 [*the grading looks at everyday communication*].

[102] The joint minute of the radiologists, which embodies the accepted premise for how BM's brain scan image (taken 10 years 11 months after his birth) presents, or ought to be interpreted, indicates as follows:

> *"1. This joint minute has been prepared between Dr D Alheit (**BA**) and Dr Z Zikalala (**ZZ**). This joint agreement is presented as a constructive attempt to present to the Court the imaging features of the MRI brain scan and to advance a diagnosis for the described pattern.*
>
> *2. BA refers to the body and comment of ZZ's report.*
>
> *3. **BA** agrees with **ZZ** that the MR study displays features **of hypoxic ischaemic injury** of the **brain**.*
>
> *4. **BA** submits that the MR findings make the diagnosis, in the appropriate clinical context, the peri-partum ***PBGT/Central hypoxic ischaemic injury of the brain** highly probable.*[53]
>
> *5. The experts agree that the findings of the MRI study suggests that genetic disorders as a cause of the child's brain are unlikely but not excluded in the light of the signal changes of the Dentate nuclei and posterior Pons. Further, clinical, genetic and metabolic assessment is advised.*
>
> *6. The **experts agree** that there is no evidence of current or previous infective or inflammatory disease on the various MRI sequences and agree that inflammatory or infective conditions are unlikely as causes of the child's brain damage.*

[53] The acronym means Peri Rolandic, Basal Ganglia & Thalamus.

> *7. The **experts agree** that a review of the clinical and obstetrical records by appropriate specialists in the field of Neonatology and Obstetrics to be essential in determining the cause and probable timing of this hypoxic ischemic injury."*

[103] The parties agreed that the reports of the expert radiologists could be admitted into evidence.[54]

[104] The diagnosis of "*PBGT*" as indicated above is what the radiologist previously used to refer to as an "*acute profound (central) hypoxic ischaemic injury of the brain*".

[105] In the field of radiology this pattern of injury would be consistent with a history of an intrapartum sentinel event.

[106] It is common cause that no obstetric emergency sentinel event is in contention here, but Dr. Alheit qualified in his report that the injury pattern seen on the MRI image should be interpreted in the context of an article by Smith *et al*.[55]

[107] This article postulates that more recent scientific evidence indicates that short or relatively short incremental hypoxic insults play a significant role in the eventual outcome of an injury pattern.[56]

[108] In this context, a "*PBGT*" injury, if not one that develops over a short period of time during an obstetric emergency (read classical sentinel event), can

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[54] Mr. Kincaid placed on record before closing the plaintiff's case that there was *"no controversy*" in respect of the joint minute of the radiologists and that their respective reports and recorded agreement could go in as a reflection of the evidence that they would have testified to at the trial.

[55] "*Intrapartum Basal Ganglia – Thalamic Pattern Injury and Radiologically Termed "Acute Profound Hypoxic-Ischemic Brain Injury" Are Not Synonymous*" Johan Smith *et al,* PubMed (Dec 2020), also accepted for publication in the American Journal of Perinatology.

[56] Dr. Kara explained that an "*insult*" is the event that occurs that can lead to an injury, but that it may not.

also result from final circulatory collapse in a neonate exposed to subacute or subthreshold hypoxia over a period of time.[57] This type of injury in the nature of an terminal insult is juxtaposed with a "*prolonged partial injury*" that develops over a period allowing compensatory redistribution of blood flow to occur, resulting in a different pattern of injury.

[109] The kind of injury pattern advocated for here (where there are documented warning signs in the form of a non-reassuring foetal status - in some instances even hours before delivery) are preventable as opposed to classical sentinel events that are not, for obvious reasons.[58]

[110] An image shown on the scan typically reflects the structural pattern description and severity, rather than it implies a causative mechanism of the brain injury. It can for example be accepted that if the clinical context or history supports the premise of a non-reassuring foetal status developing during the labour in question and is prolonged, a BGT pattern may result in the absence of a perinatal sentinel event.

[111] I add that the 2019 ACOG update referenced in the report of Dr. Alheit states that *"Deep gray nuclear injury commonly occurs (25 – 75% of cases) following severe partial insult of prolonged duration or combined partial with profound terminal insult".*

[112] But this is where the assistance to this court ends from the radiological perspective. The timing and pathogenic mechanism of the injury falls outside the field of radiological imaging according to the experts and must be informed by

[57] This is in my view exactly the kind of scenario in contention here.
[58] See Smith *et al*, *Supra*, where the following is stated: *"This paper supports the notion that with appropriate intrapartum care and timeous reaction to FHR abnormalities and action in the form of intrapartum resuscitation and expedited delivery, in the majority of cases adverse BGT pattern injury would have been prevented."*

obstetricians and paediatric experts, hence the concluding remark in paragraph 7 of the joint minute above in which the radiologists fairly defer these issues to the other experts.

[113] Regarding the reservation expressed in paragraph 5 of the joint minute above, although Dr. Alheit pointed out that the differential diagnosis suggested by the increased signal and some loss of volume in the dentate nuclei and posterior pons seen on BM's scan included the likelihood of congenital/genetic/hereditary and metabolic conditions, this was definitively ruled out by Prof. Denis Viljoen, a specialist in medical genetics.[59]

[114] Prof. Viljoen did not testify, but his opinion was accepted that:

> *"From both the negative family trees and clinical histories of this child's birth and subsequent obstetric/neonatal complications, (supported by MRI brain scans), hypoxic ischaemic cerebral palsy is the most probable diagnosis.* ***Genetic causes for his clinical findings are very unlikely****. Similarly, syndromic or metabolic cause for the epilepsy, development delays and cerebral palsy are also unlikely."* (Emphasis added.)

[115] Notwithstanding the common opinion expressed in paragraph 6 of the joint minute that there is no evidence on the MRI picture of previous infective or inflammatory disease and that such conditions are unlikely as causes of BM's brain damage (in effect putting paid to the defendant's hypothesis of neonatal meningitis being the cause of BM's brain injury), the professional view held by

[59] Mr. Kincaid at a point before closing the plaintiff's case placed on record that he was awaiting the defendant's instructions in respect of Dr. Viljoen's report (to which there had been no corresponding report from the defendant). When the matter resumed after a long break I was informed from the bar that Dr. Viljoen would not be testifying so I assumed that the contents of his report was no longer in contention. With hindsight though I did not enquire from Mr. Ngadlela, who appeared for the defendant, as to the status of the report in the evidentiary context. There was however no countervailing evidence adduced by the defendant that the causal potentiate for BM's brain injury was generic or hereditary or congenital.

the defendant's paediatric neurologist expert, Dr. Yavini Reddy, who was initially expected to testify on the defendant's behalf and who put up a summary foreshadowing such opinion, [60] is that BM's confirmed neonatal encephalopathy post-delivery was "*most likely due to neonatal meningitis*". To give a context to the picture seen on the MRI scan, Dr. Reddy stood poised to state that neonatal meningitis can cause neonatal encephalopathy and bilateral basal ganglia or thalamic infarcts on MRI.[61] (The defendant in the end led the evidence of Prof. Bolton, paediatric neurologist, who also sought to present such a hypothesis.)

[116] With this agreed premise of the expert radiologists in mind, the issue arising is whether the originating cause for BM's cerebral palsy in this case can, on a balance of probabilities, be ascribed to intrapartum birth events.

[117] If the answer to this question is in the affirmative, a causal connection between the alleged negligence (if established) and BM's injury can then in my view plausibly be inferred.

<u>The timing of the damage causing event:</u>

[118] It is often repeated in our courts by specialists in the fields of neonatal and obstetrics in matters such as these (as was the case here), that the timing of the HIE injury, at least from a clinical perspective, is hard to gauge unless some sentinel event in the classical sense of an obstetric emergency has occurred. In all other cases and in the absence of direct evidence on the obstetric management or other indications that there was fetal compromise at a specific point in time, or a noticeable "*bradycardia moment*" in this instance as Mr. Kincaid who appeared

[60] Prof. Bolton stepped into the breach.

[61] I clarify that the parties agreed in the pre-trial processes that the reports of the experts who could not avail themselves to testify for varying reasons would not amount to evidence, but the joint minute already finalised as between the paediatric specialists by then obviously bound the parties to their agreement.

for the plaintiff put it, this is often left to inferential reasoning based on the medical records indicating the clinical setting and objective expert opinion concerning the probabilities one way or the other. The condition of the baby at birth is also vital to this enquiry.

[119] Since HIE suffered during labour is a recognized pathway in itself to cerebral palsy (as discussed below) it is already in my view suggested as a cause for such a condition or having a natural relationship with intrapartum birth events.

[120] Before discussing the criteria the specialists in the field commonly advert to in order to determine the cause and probable timing of a hypoxic ischaemic injury, and more especially BM's in this instance, it is necessary firstly to outline certain basic concepts and premises referenced by the experts in this matter that are applicable to the issues at hand.[62]

[121] Firstly neonatal encephalopathy is a clinically defined syndrome of disturbed neurological function in the earliest days of life in the term infant, manifested by difficulty with initiating and maintaining respiration, depression of tone and reflexes, sub normal level of consciousness and often seizures, usually affecting the full term infant.

[122] HIE, in turn, is a subgroup of neonatal encephalopathy. To consider hypoxic ischaemic encephalopathy to have occurred in the intrapartum period, there has to be evidence of neonatal encephalopathy. This needs to be confirmed to be a moderately severe encephalopathy (grade 2), and to last at least 7 days.

[62] The ensuing detail is derived from the expert reports of the corresponding obstetricians which in turn reference the summary of the ACOG Consensus Statement and the standard textbook on the neurology of the newborn by Volpe.

[123] A Table of Grades of Encephalopathy appears below:

	Grade 1 Mild	**Grade II Moderate**	**Grade III Severe**
		Lethargy	Coma
Alertness	Hyperalert	Hypotonic	Flaccid
Muscle tone	Normal or increased	Frequent	Uncommon
Seizures	None	Small, reactive	Variable, fixed
Pupils	Dilated, reactive	Periodic	Apnoea
Respiration	Regular	2 – 14 Days	Weeks
Duration	<24 Hours		

[124] Further, before attributing intrapartum hypoxic injury to be the cause of neonatal encephalopathy, one has to consider the probability of other conditions that may cause an encephalopathy such as for example, meningitis, congenital brain abnormalities; vascular abnormalities; maternal infection or intrauterine infection or severe neonatal sepsis; metabolic or chromosomal disorder; obstetric cases that affect blood flow to the foetal brain (for example placental abruption; eclampsia, maternal hypotension, umbilical cord compression; prematurity; intrauterine growth retardation); maternal drug use; and severe neonatal jaundice.

[125] Then, to the question whether such an injury has occurred during labour, according to Volpe[63] certain factors need to be present before being able to make the diagnosis of an intrapartum insult being the cause of neonatal brain injury. Firstly, there should be evidence of foetal distress and/or foetal risk for hypoxia-ischaemia (FHR abnormalities, sentinel event, foetal acidaemia), secondly, there ought to be evidence that there was a need for resuscitation of the baby and/or low Apgar scores and, thirdly an overt neurological syndrome in the first hours and day of life should be indicated. If these criteria can be confirmed, this

[63] Neurology of the Newborn, 2017, Sixth Edition.

supports the probability of a relationship between intrapartum events and cerebral injury further strengthened by the injury picture shown on the MRI scan where applicable.

[126] The American Congress of Obstetrics and Gynaecology ("*ACOG*") stipulates its own criteria which all the experts who testified to in this matter subscribe to. In 2003 ACOG and the American Academy of Paediatrics ("*AAP*") published guidelines that had 4 essential criteria and 5 supplementary criteria to define an acute intrapartum event as sufficient to cause cerebral palsy. These guidelines were revised in April 2014 and "*essential criteria*" no longer exist. Presently, in order to determine the likelihood that an acute hypoxic ischaemic event that occurred in close proximity to labour and delivery contributed to neonatal encephalopathy, the Consensus Report instead emphasizes the weighting of various risk factors. It considers all potential contributing factors including maternal medical history, obstetric history, intrapartum factors (foetal heart rate and issues relating to delivery) and placental pathology. The more factors present, so it is suggested, the more likely it is that there was an intrapartum event that conduced to the NE.

[127] Indeed the second edition of the Consensus Statement recognizes that:

> *"There are multiple potential causal pathways that lead to cerebral palsy in term infants. A broader perspective is now necessary before attributing neonatal encephalopathy to an intrapartum event. It is now recommended that a comprehensive multidimensional assessment be performed of neonatal status and all potential contributing factors including maternal medical history, obstetric antecedents, intrapartum factors and placental pathology."* [64]

[64] This quote is referenced from Dr. Kara's expert report.

[128] Prof. Bolton who came late to the party in filing an expert summary applied the science through the filter of the abovementioned Consensus Report to determine if the features of the plaintiff's labour and the condition of BM immediately after birth and in the aftermath, culminating in him developing cerebral palsy, fitted the template. He thought not, or more specifically that neonatal meningitis was the more likely cause. The same scientific measure was applied by Dr. Kara, specialist paediatrician, who reached a different opinion, namely that the cause for BM's neurological outcome probably resided in an intrapartum birth event.

[129] As for the foetal heart rate monitoring patterns both Drs Kara and Chimusoro were satisfied that there were at least two foetal heart rate tracings at 09h30 and 11h36 on the morning preceding BM's birth that indicated a non-reassuring foetal status, coupled with the significant event that the plaintiff was nursed on her side and given oxygen by mask at 09h30. Both specialists opined that this was consistent with a concern for the wellbeing of the foetus at the time.

[130] That leads me to the type and timing of "*contributing factors*" that are consistent with an acute peri-partum or intrapartum event according to the Consensus Statement. Concerning the "*other significant factors*" that might steer one in the direction of concluding that an acute intrapartum event as the sole underlying pathogenesis of NE becomes "*much less likely*", the experts accepted that there were indeed proximal and distal factors in the offing, but Dr. Kara disagreed in this instance that the idea of infective causes could lead one away from concluding that the acute intrapartum event was the more probable causative agent for BM's cerebral palsy.

[131] The Consensus Statement conceptualizes certain pathways to cerebral palsy over various stages commencing with conception and ending with

childhood. In between these outer limits are the two stages of pregnancy and labour (ante and intrapartum respectively) followed by the neonatal stage.

[132] It recognizes that both distal and proximal risk factors straddling this five stage period can exert a pathogenic effect on foetal brain development. It is opportune to repeat these pathways envisaged by the Consensus Statement as it will give a context to Prof. Bolton's opinion that Pathway "*E*" pertains in the current scenario:

	A	B	C	D	E
Conception		+-DRF	+-DRF	+-DRF	+-DRF
		↓	↓	↓	↓
Antepartum		+-DRF	+-PRF	+-PRF	+-DRF
		↓	↓	↓	↓
Intrapartum	PRF	PRF	↓	PRF	DRF
	↓	↓	↓	↓	↓
	*	*	↓	*	*
	↓	↓	↓	↓	↓
Neonatal	NE	NE	+-NE	NE	PRF →*→NE
	↓	↓	↓	↓	↓
Childhood	CP	CP	CP	CP	CP

FIGURE 1

Prenatal and perinatal causal pathways to cerebral palsy in term infants. Distal risk factors exert a pathogenic effect on fetal brain development starting at a time that is remote from the onset of irreversible brain injury. Examples include genetic abnormalities, environmental and sociodemographic factors, and some placental abnormalities. Proximal risk factors exert pathogenic effects on fetal brain development at a time that closely predates or coincides with the onset of irreversible brain injury. Examples include abruption placentae, chorioamnionitis, and twin-twin transfusion. There are multiple potential causal pathways that lead to cerebral palsy in term infants, and the signs and symptoms of neonatal encephalopathy may range from mild to severe, depending on the nature and timing of the brain injury. A. Intrapartum brain injury that is due to a proximal risk factor may lead to neonatal encephalopathy and subsequent cerebral palsy. B. Intrapartum brain injury may be the result of both distal and proximal risk factors that predispose the fetus to brain injury and cerebral palsy. C. Brain injury or anomaly may occur in the antepartum period as a result of distal and proximal risk factors. D. Brain injury may occur at multiple points during gestation. E. Proximal risk factor and brain injury may occur in the neonatal period following predisposing distal risk factors. Abbreviations: DRF, distal risk factor; PRF, proximal risk factor."

[133] It is appropriate to point out that the concepts of "*distal*" and "*proximal*" are unique to the application of the professional Consensus Statement. Of course its criteria implicating intrapartum hypoxia in neonatal encephalopathy has utility from an obstetric and pediatric perspective and can and does assist the court in its determination of the proof of a causal link between a defendant's claimed actions or omissions (read negligent intrapartum care), on the one hand, and the

harm suffered by the plaintiff (cerebral palsy in this instance), on the other hand, but its application is not a substitute for the court's own legal causation enquiry that it must undertake. There is a clear difference between scientific and judicial measure which a court is constrained to keep in mind.

The legal requirements:

[134] It is a trite principle that a successful delictual claim entails the proof of a causal link between a defendant's claimed culpable actions or omissions, on the one hand, and the harm suffered by the plaintiff, on the other hand.[65] This is in accordance with the flexible "*but-for*" test.[66] As is indicated by the authorities, in order to apply this test one must make a hypothetical enquiry as to what probably would have happened but for the alleged wrongful conduct of the defendant. Sometimes however this enquiry involves the mental elimination of the claimed wrongful conduct and the substitution of a hypothetical course of lawful conduct and the posing of the question as to whether upon such hypothesis the plaintiff's loss would have ensued or not.[67]

[135] Legal causation is required to be established on a balance of probabilities.[68]

[136] Given the accepted premise underscored by the Consensus Statement that cerebral palsy has its pathogenesis in multifactorial pathways and is not necessarily the direct result of an adverse event during labour that could have been prevented, the significant question in this matter is therefore whether, as a

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[65] *International Shipping Co (Pty) Ltd v Bentley* 1990 (1) SA 680 (A) at 700F-I; *Siman & Co (Pty) Ltd v Barclays National Bank Ltd* 1984 (2) SA 888 (A) at 915B-H; Minister of Police v Skosana 1977 (1) SA 31 (A) at 35C-E; *Lee v Minister of Correctional Services* 2013 (2) SA 144 (CC) at para [37] – 58]; *Oppelt v Head: Health, Department of Health Provincial Administration: Western Cape* 2016 (1) SA 325 (CC) at [35].
[66] *Lee, supra at* para [37] – [58]
[67] *Mashongwa v PRASA* 2016 (3) SA 528 (CC) at [65]; *AN v MEC for Health, Eastern Cape* [2019] ZASCA 102 at [8].
[68] *Lee supra* at [39]

matter of probability, BM's condition would in any event have ensued even if the defendant's claimed negligent intrapartum care had not occurred.[69]

[137] Dr. Kara opined that all the indications in the multifactorial assessment supported the probability of BM having sustained the injury during the intrapartum period. Prof. Bolton conversely offered his primary view that the originating cause for BM's cerebral palsy is to be found in the differential diagnosis of meningitis made by the staff at DNH after his birth which he was comfortable elevating to a confirmed diagnosis as far as he was concerned.[70] Later however, as he was fleshing out his opinion and challenged by Mr. Kincaid as to why he was quite resolute about postnatal meningitis being causal of BM's neurological outcome, a new theory evolved that it was a "*foetal meningitis*" at play, caused by a viral infection that later emerged as a sepsis and inflammation of the placenta, in other words, a chorioamnionitis.

The case for the paediatricians:

[138] From the paediatric perspective certain features of BM's birth and neonatal status were confirmed in the joint minutes of Dr. Kara (paediatrician) and Dr. Reddy (paediatrician neurologist)[71] that set the tone for the way forward.

[139] It is firstly not in dispute that BM has hemiparetic cerebral palsy with global development delay and epilepsy. Despite this not being the typical spastic

[69] *Oppelt, supra*, at [35]; *Mashongwa, supra* at [65]
[70] This view was based solely on his interpretation of the blood report even though there was no culture of the organisms done that would have definitively clarified the issue. It is common cause that specimen extracted from BM's spine had traces of blood in it and that it was not adequate for its purposes.
[71] As indicated elsewhere Dr. Reddy was not called to testify and Prof. Bolton stepped into the breach. The agreement recorded by her with Dr. Kara however bound the parties and dictated the premise going forward.

or dyskinetic cerebral, it is nonetheless cerebral palsy. (He is also known with retroviral diseases on treatment.)[72]

[140] Also not in dispute is the fact that BM was actively resuscitated at birth and had spontaneous respiration only at 15 minutes. The admitting concerns were birth asphyxia and HIE. He had lip smacking at 23h00 on19 August and documented convulsions on 20 August 2007 at 09h00. He was commenced on Phenobarbitone. According to the experts there was no doubt that he had at least a moderate neonatal encephalopathy lasting several days after his delivery.

[141] There was certainly no concern over the foetal condition on admission in labour. There was poor progress in labour and augmentation of labour with Syntocinon. Despite the import of the notes in the MCR that suggested that the CTGs were generally reactive, there were concerns of foetal distress and intrapartum resuscitation was commenced at 09h30 on 19 August 2007, in his view exactly to ameliorate concerns of foetal compromise.

[142] Dr. Reddy however deferred to expert obstetric opinion regarding the management of labour. Her observation was that there is no record *in the notes* regarding foetal distress at the time intrapartum resuscitation was commenced, hence one of the *indiciae* required in the Consensus Statement to confirm the presence of NE was lacking.[73]

[143] Ultimately the paediatricians differed (in the joint minute) regarding the more probable cause of BM's neonatal encephalopathy, the defendant's expert

[72] This diagnosis pertains to the plaintiff's separate claim for damages arising from the hospital staff's failure to have prevented the mother to child transmission of HIV (Claim C). This has no bearing for present purposes.
[73] It is common cause that there were only two CTG tracings leaning toward an indication of foetal compromise at 09h30 contemporaneously with the intrapartum resuscitation, and shortly afterwards at 11h36, but I have elsewhere stated that these recordings (at least according to the plaintiff's experts) support an inference that there was foetal distress at these moments.

contending that neonatal meningitis was a more likely cause, whereas on behalf of the plaintiff the theory promoted is that the injury shown in the neuroimaging fits in with an intrapartum hypoxic event which renders infective, metabolic and genetic aetiologies less likely for this appearance.

[144] Applying the criteria of the Consensus Statement, according to Dr. Kara. the confirmation of foetal compromise, the prolonged resuscitation at birth and the significant encephalopathy, together with the MRI scan features, the abnormal renal function suffered by BM, the respiratory distress and the events in labour make it probable that his cerebral palsy was due to an intrapartum hypoxic ischaemic injury.

[145] Contrariwise the opinion proffered by Dr. Reddy was that although BM had confirmed neonatal encephalopathy post-delivery, this was most likely due to neonatal meningitis. This view seems to be founded on her sub-view, if I can call it that, that neonatal meningitis can mimic the changes seen in hypoxic ischaemic encephalopathy on neuroimaging.

[146] Prof. Bolton when he testified on behalf of the defendant expounded upon Dr. Reddy's opinion that, in the absence of a sentinel event during labour and a supposed lack of evidence of negligence, neonatal meningitis was the most likely cause of BM's clinical condition.

[147] His approach initially was to resolutely adopt pathway "E" referred to in the Consus Statement which as I point out above differentiates from a clinical point of view between distal and proximal risk factors. He found distal factors at the conception stage to include the plaintiff's HIV status. Since HIV is a neurotrophic virus (that is likely to attack or affect the nervous system) he noted that the plaintiff's status increased the risk of infection within the amniotic cavity,

the umbilical cord, and the placenta - referred to as chorioamnionitis. Infection of the placenta results in inflammation and impairs the foetal oxygenation process. Chorioamnionitis is also associated with an inflammatory cytokine storm according to his testimony, which directly damages the brain.

[148] His conclusion was that the plaintiff's HIV infection in itself predisposed BM to cerebral palsy. The mechanism was probably placental inflammation, the impairment of foetal oxygenation during labour, and the production of inflammatory cytokines which he opined directly damaged the brain.

[149] He added that the plaintiff's hypertension would also have contributed to the impairment of maternal-foetal oxygen perfusion. In his view these two factors predisposed BM to the poor condition evidenced at birth and the subsequent development of NE.

[150] Under cross examination he conceded that a hypoxic ischemic injury may have occurred *during labour* but yet maintained that it was most likely caused by placental damage or insufficient placental perfusion as a result of the chorioamnionitis and that the possibility exists that the hypoxic ischemic injury could have been further compounded by the onset of neonatal meningitis.

[151] In essence his view is that the damage, manifested at the neonatal stage, probably had its origins in the risk factors which were in existence as far back as conception and which remained present throughout the ante and intrapartum development of the foetus.

[152] It goes without saying however that the known existing risk factors referred to by Prof. Bolton were common cause (this includes the plaintiff status as a primigravida) and impacted significantly on the labour management strategy

that had to be adopted as a very result. In this respect he deferred to the obstetricians.

[153] As Mr. Kincaid pointed out in his argument, if Prof. Bolton was deferring to Dr. Jankowski's assessment of the plaintiff's labour management, the implication thereby is that there was no fault whatsoever with the way in which the plaintiffs labour was managed, that there was no foetal distress recorded during the labour, and that accordingly BM's clinical condition at birth could not have had a hypoxic ischemic origin.

[154] However, as I intend to demonstrate below, this was a very tenuous ground on which to rest his theory as to the more probable cause of BM's NE.

[155] There further seems to be no basis to reject the accepted views of the radiologists that BM's brain injury seen on the MRI scan does not show any evidence of current or previous infective or inflammatory disease, hence such a causal agent for BM's damage is unlikely.

Obstetric management:

[156] As for the plaintiff's pregnancy and the management of her labour her antenatal care was described by the expert obstetricians generally as "*no abnormalities detected*". (Prof. Bolton introduced the possibility that the infections that the plaintiff had presented with during her pregnancy closer to her labour were "*relevant*", suggestive of chorioamnionitis, and at least constituted a distal factor in the whole scheme of things.)

[157] They agree that upon the plaintiff's earlier admission to the Livingstone Hospital (between 16 and 17 August 2007), despite her presenting with raised

blood pressure, she was not given treatment for her hypertension. It was also commonly accepted that no treatment for gestational hypertension was administered at DNH on the 17th. Treatment only commenced on the 18th at 07h00, after a recordal of a blood pressure reading of 155/105. Dr. Janowski readily conceded that up until the 18th this constituted substandard care, but with no causal outcome. In his view from the 18th everything was done in compliance with the Maternity Care Guidelines "*on the dot*", but was it? And, from a causal perspective, what about possible insults from before given his acknowledgement of the risks posed by the plaintiff's raised blood pressuring during contractions?

[158] The latent phase of labour commenced at the very latest at 7h00 on 18 August 2007 at the DNH, this after the plaintiff's transfer from LH. Dr. Chimusoro explicated that based on pain and cervical changes that the plaintiff suffered on the 17th already that this denoted that the latent phase of labour had commenced earlier but was prepared to accept that it was later as suggested by his counterpart. But by the standards set out in the Maternity Care Guidelines, the plaintiff's labour was way prolonged and Dr. Janowski's approach of letting nature take its course and adopting a wait-and-see approach with all that was going on with the plaintiff's pregnancy and the peculiar risk factors that pertained does not accord with the expectation of a reasonable clinician to be particularly concerned and more attentive to the risk of hypoxia developing. It certainly makes practical sense to have explored why after being more than forty weeks into her pregnancy the plaintiff's labour was progressing slowly by the hospital's own normative standards of how long it should reasonably have been taking.

[159] A CGT was commenced on the plaintiff's admission, but no comments appear from the MCR that on arrival these were moving to Category II (ACOG) or suspicious. (FIGO). (Dr. Kara coincidentally referenced this as a measure to determine that before labour the foetus' wellbeing was certainly not in contention.

This provided a further indication that the damage causing event must have been something that occurred during the plaintiff's labour, not before.)

[160] At 16h00 on the 17th the plaintiff was diagnosed as being in prolonged labour (latent phase) with recognized gestational hypertension. From this point on at least, according to Dr. Chimusoro, a different, focused, management strategy, again guided by the defendant's own normative guidelines, ought to have been put in place. Indeed after having identified the plaintiff as a code red patient who was especially required to be referred to a tertiary hospital to deal with her complications, it was counterintuitive to then have adopted a lackadaisical approach one she got to DNH.

[161] Digital foetal heart rate monitoring was purportedly in place and at least twice abnormalities were recorded (tracings interpreted by Dr. Chimusoro as non-reactive), even though other recordings (in-between and afterwards) were opportunistically latched on to by the defendant as being reassuring. I have elsewhere adverted to the poor standard of reporting and the criticism that those that had commented upon the foetal heart rate monitoring in the vaguest of terms fell short of the ordinary standard of reporting on the wellbeing of the foetus that is an imperative during the course of a patient's labour. But the fact is that the writer of these two cryptic reports indicated that the heartrate on those occasions came close to the bradycardia upper limit. I will assume that this is what was intended to be conveyed by the range indicated at these two moments. At least in respect of the 09h30 recording there is the further entry as to the plaintiff's intrapartum resuscitation which gives credence to a worrying concern for the foetus' wellbeing at this juncture.

[162] Despite the administering of anti-hypertension medication the plaintiff's blood pressure was raised even further. Even if asymptomatic (of imminent

eclampsia) as written by a doctor, I accept Dr. Chimusoro's reasonable suggestion that the management of it should have been preventative and that the possibility of the plaintiff fitting especially kept in mind.

[163] The success or not of the argumentation is open to interpretation because at the end of it the foetus became stuck in the birth canal and the plaintiff's contractions were no longer effective to push her baby out. I accept the plaintiff's evidence that she was also not conscious at critical moments when her effort was required to assist with the expulsion, an outcome that I should imagine was quite inevitable given what medication was being administered to her despite the fact that her contractions had been gathering a momentum on their own up to a point. (It is not clear exactly when the stimulant was introduced but it appears to have been after she had progressed to the active stage of labour by her own efforts.)

[164] The Apgar scoring was open to different interpretation but this to my mind is a mere red herring. I have dealt with this elsewhere.

[165] Clear liquor was in evidence at 5h10 on the 19th (although Dr. Kara disagreed that this was indicative of the fact that the foetus was not in a non-reassuring status). The defendant did not deal with the conflicting entry regarding the supposed rupture of the plaintiff's membranes at 10h10 on the 18th already. I accept that this event on its own had adverse consequences for placental perfusion given the plaintiff's HIV status.

[166] The caput ++ *with no moulding* (in relation to the possible existence of CPD against the background of an extremely protracted labour) is open to interpretation. Dr. Chimusoro's observation that it was strange to report "*no moulding*" given the obvious challenges to the foetus coming out and thereafter having to be extracted by forceps delivery commends itself to me.

[167] The fact that the plaintiff was given intrapartum resuscitation at 09h30 on the 19th is as I have said before entirely consistent with foetal distress. There is simply no reason offered by the defendant as to why a resuscitation would have been necessary at all if it was not significant in relation to the management of the plaintiff's labour at that point in time.

[168] Dr. Janowski was of the view that trial of labour was appropriate and that there was no indication for a caesarean section. The irony is though that those involved in the management of the plaintiff's labour recognized the necessity for such a procedure but dallied. On any reading of the situation the concerns that brought them to this conclusion were not kept in check afterwards as a reasonable practitioner ought to have done.

[169] The experts across the board all described the impact of labour on a foetus as hypoxic-centric. This is because when the womb contracts the muscles of the uterus compress the blood vessels and the blood supply to the placenta drops at the time when this is happening. After the contraction wanes, there is reperfusion and blood flows back into the placenta making sure that the foetus gets enough oxygen again. Dr. Chimusoro explained that in the latent phase very little harm is expected to occur because the contractions are mild and spaced out but even these stresses can conduce to sub hypoxia if the labour is protracted. This is because in the face of a continual constriction of the vessels the foetus will ultimately run out of oxygen but in the ordinary course a foetus has adequate compensatory mechanisms to bounce back. In the active phase of labour the mother's contractions are significantly ramped up in intensity, frequency and duration so the uterine environment becomes naturally more of an effort.

[170] The situation becomes uniquely challenging, so Dr. Chimusoro explicated, when the mother suffers from hypertension which can cause harm to both her and the foetus. In such a situation the blood flows into the uterus under very low pressure. The placenta has no real arteries. It is just a pool where the blood circulates slowly so when the mother's blood pressure goes up that flow is interfered with and the foetus stops getting enough oxygen perfusion. Other complications may also arise such as bleeding between the placenta and the uterus which can compromise the foetus even further. These factors need to be taken into consideration and acted upon by ameliorating the hypoxic risk to the foetus and ensuring that it is delivered in defined times, not as the staff chose to do on a provisional wait and see basis. For this reason, so Dr. Chimusoro noted, the caesarean threshold is lower for a patient who has hypertension.

[171] He added that challenges can also arise in the case of a patient who is HIV positive because once the protective membrane is ruptured and delivery does not ensue promptly, ascending infections from the mother's vagina can on their own cause damage to the placenta. He added his opinion that the risk of hypoxia is three times higher in a mother who is HIV positive, a notable risk factor in the plaintiff's pregnancy that was also confirmed by Dr. Kara's evidence.

[172] There would have been an imperative to have been aggressive in preventing the plaintiff from fitting, so Dr. Chimusoro observed, because this would also have involved a significant drop in perfusion for the foetus upon such an event. As it turns out the plaintiff in fact fitted and intrapartum resuscitation had to be implemented forthwith, plausibly both suggesting that the treatment of her hypertension had not been inadequate and that the foetus had been driven to the very point of distress that should have been reasonably contemplated.

[173] It is accepted that the plaintiff's labour (latent phase) was protracted or "*very prolonged*". Dr. Janowski was the only expert who thought it didn't matter and that one should let nature take its course. The Maternity Care Guidelines at the time suggested that the latent phase of labour for someone in their first pregnancy should not have been longer than 8 hours. In Dr. Chimusoro's view the national tolerance at the time was 12 hours.

[174] In the plaintiff's situation it seems that apart from suggesting that a caesarean section might be indicated there was no real investigation into the reasons for the delay and no alacrity was shown in moving the plaintiff's delivery along. Indeed no one seemed to be concerned that she was post term and had been suffering from raised blood pressure (with protein indicated in her blood 3 days before). The size of the foetus on its own should also have raised concerns in the context of the duration of the latent phase of labour, the other presenting challenges, and the known risk factors.

[175] It is not clear that the plaintiff's HIV status received appropriate recognition in the planning and management of her labour. There are simply no notes that give such an assurance and even Dr Janowski failed to factor it in as a consideration in his expert report.

[176] Regarding the issue of the plaintiff's hypertension there is no consistent recording of this and at one stage when bloods were interpreted there was a conflict in the numbers reading off the same blood report.[74] CTG monitoring was also inadequate especially closer to the delivery, which means that a valuable opportunity was lost to detect if the foetus was tolerating the labour and from what point it began to suffer compromises which it must have for the Grade II neonatal encephalopathy BM presented with at birth to have exhibited itself.

[74] The blood reports themselves were not provided at the trial.

[177] I accept for present purposes that there was no Misoprostol administered to the plaintiff, but the MCR is lacking in clarity concerning what medication was given and when. Dr. Janowski proffered the explanation that Pitocin would not have been dispensed from the pharmacy but from the midwife's stock as it were, which is all the more reason why detailed notes of its administration should have been in evidence to satisfy the court that the plaintiff's contractions (in effect being managed through the use of a stimulant and which at some point were said to have caused a tachysystole) was handled appropriately and professionally.

[178] Dr. Chimusoro found fault with inadequate monitoring according to standard operating procedure of all the necessary milestones and markers to confirm maternal and foetal wellbeing in the circumstances especially of the plaintiff's high blood pressure that continued in an upward trend including the absence of prompt treatment by medication *inter alia* implicating anti-seizure management; the absence of continuous CTG monitoring to make sure that there was no gap or window where the foetus was left unobserved; the absence of management by a senior specialist or a senior consultant or dedicated medical officer for an admittedly complicated case; the failure to have charted a proper partogram or to have taken any appropriate steps when on the supposed instrument the action line had in any event been crossed; the failure to have had a meaningful focussed strategy plan in place; the failure to have ruptured the plaintiff's membranes earlier to release the forewaters (by latest 17 Aug or to augment labour timed earlier in proximity to the rupture at 10h10 of the 18th); the failure in any event to have treated the recorded rupture of the plaintiff's waters on the 18th by way of an antibiotic course when delivery was not yet imminent; the giving of a bolus of Oxytocin (because flooding the plaintiff's veins would have had the opposite effect of stimulating and making the contractions more coordinated and might have caused receptors in the uterus that cause oxytocin to stop working to have been blocked off); the failure commensurate with the

recognition of foetal distress registered at 9h30 on the 19th to have taken steps to immediately get the foetus out of the hypoxic environment or the taking of other appropriate steps; the failure to have recognized the further indication of the slowing down of the foetal heart rate at 11h36 and thereupon to have redirected the plan regarding augmentation and again to have immediately removed the foetus from the harmful environment; and the failure of the staff to have recognized the hyper stimulation of the plaintiff's uterus by the augmentation.

[179] I have dealt elsewhere with the failure of the staff to have kept adequate notes.

[180] The opinion of Dr. Janowski by comparison is that the management of the plaintiff's labour was not substandard except before the 17th. He was not concerned by the number of hours added to the plaintiff's labour because in his view the condition of the baby was not compromised. Whilst acknowledging all the risks posed by the plaintiff being pre-eclamptic and whatever could go wrong, he yet defended the treatment as being adequate and according to the textbook.

[181] Regarding the augmentation, he was similarly alive to the reality that the plaintiff had to be strictly monitored, agreeing that more frequent contractions equals the risk of foetal distress and/or compromise.

[182] He acknowledged that the labour graph is incomplete and filled in incorrectly as well as the absence of CTGs to have given a picture after the fact of whether the monitoring ought to have raised any alarm bells. He appreciated that the value in these records is for the staff to have had a graphic recording to hand of foetal heart rates and uterine contractions exactly with a view to acting appropriately upon these results where necessary to promote the wellbeing of the foetus. Concerning the manner in which the staff reported on the CTGs he assumed that they were appropriately trained to say what they did (that is by only

reporting a curt conclusion) despite the reporting not conforming to the normative standard required for this.

[183] For the rest, and despite the reservation expressed by him in his summary at the outset that the incomplete records made it difficult for him to give a fair report he yet defended the actions of the hospital staff as having been beyond reproach, causing Mr. Kincaid to contend, not unfairly in my view, that he failed to meet the high standards required for an objective and independent review of the labour management.

[184] Not only that, but he also seemed prepared to put his head on a block that the plaintiff had as a fact not fitted because there was nothing in the records to this effect. Despite the standard of the recordkeeping which he noted in superlative terms to be poor, he went so far as to suggest that her evidence on his feature of her labour experience was contrived. When it became evident that this was a serious consideration that the court might accept, he failed to offer any opinion on the subject as if it were true.

[185] He was further adamant that the lower Apgar scores (as opposed to the lone recording by a doctor that purported to signal that BM was well at birth contrary to every other indication), fell to be rejected.

[186] This is certainly one of those instances in which the staff of the hospital should have been called to expound upon their treatment of the patient, rather than leaving it to an expert to make speculative submissions about whether they met the requisite standard of reasonable care predicated on a very tenuous basis.

[187] I am satisfied that both Drs Kara and Chimusoro's evidence meets the required logical reasoning standard[75] (the same cannot be said of Dr. Janowski's views) and that their views accord with generally accepted medical norms.

[188] I add that the alternative hypotheses put forward by Prof. Bolton as to the cause of BM's condition (at least the primary view that Pathway "E" pertained based on the hospital staff's differential diagnosis of meningitis) was also speculative and in fact contraindicated by the objective MRI brain injury pattern and the views of the expert neuro-radiologists expressed in this regard.

Conclusion:

[189] The failure of a professional person to adhere to the general level of skill and diligence possessed and exercised at the same time by the member of the branch of the profession to which he or she belongs would normally constitute negligence.[76]

[190] Concerning the approach to be adopted in determining the issue of negligence the court in *Goliath v MEC for Health*[77] noted with reference to Lord Justice Hobhouse's *dictum* in *Ratcliffe* that:

> "*At the end of 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 or probabilities that the defendant was negligent and that such negligence caused the plaintiff's injury. That is the long and short of it."* [78]

[75] *Michael and Another v Linksfield Park Clinic (Pty) Ltd and Another* 2001 (3) SA 1188 at [36]
[76] *Goliath v MEC for Health, Eastern Cape* 2015 (2) SA 97 SCA.
[77] *Supra* at para 8.
[78] *Supra* at par 18.

[191] The court noted further in this connection that:

> *"... it is important to bear in mind that in a civil case it is not necessary for a plaintiff to prove that the inference that she asked the court to draw is the only reasonable inference. It suffices for her to convince the court that the inference that she advocates is the most readily apparent and acceptable inference from a number of possible inferences (AA Onderlinge Assuransie Assosiasie Bpk v De Beer 1982 (2) SA 603 (A); see also Cooper & another NNO v Merchant Trade Finance Ltd 2003 SA 1009 SCA)".*[79]

[192] I am satisfied that BM's brain injury that predisposed him to cerebral palsy was caused by the negligent intrapartum care on the part of the hospital as contended for in the plaintiff's particulars of claim which had a deleterious effect on his foetal wellbeing, leading to the final acute insult that caused the damage.

[193] A plaintiff is not required to establish the causal link with certainty, but only to establish that the wrongful conduct was probably a cause of the loss.[80]

[194] When the factual premise is considered in its entirety, that is the plaintiff's protracted labour, the negligent monitoring, the failure to have properly assessed the evident risks, and the failure to have implemented the necessary and appropriate interventions in proper time as the exigencies dictated, all logically in my view contributed to and have a sufficiently close connection with the damage to BM that resulted.

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[79] *Supra* at par 19.
[80] *Minister of Safety and Security v Duivenboden* 2002 (6) SA 431 (SCA) at par [25].

Order:

[195] In the result I issue the following order:

1. The defendant is declared liable for all such damages as the plaintiff may prove on behalf of her minor child who was delivered at the Dora Nginza Hospital, Gqeberha, on 19 August 2007.

2. The defendant is liable to pay the plaintiff's costs of suit to date, which costs shall include the qualifying and related travelling and appearance fees of the expert witnesses retained by the plaintiff, as well as the costs of two counsel.

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B HARTLE
JUDGE OF THE HIGH COURT

DATES OF HEARING : 23-25 February,
1-2 August & 28-29 November 2022,
31 January-2 February &
12 April 2023
DATE OF JUDGMENT : 15 March 2024

Appearances:

For the plaintiff: Mr. J Kincaid together with Mr. X C Stemela, instructed by Gqeba Inc., East London (ref. Mr. Gqeba.).
For the defendant: Mr. N D Ngadlela instructed by the State Attorney, East London (ref. Mr M Maqambayi).