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**IN THE HIGH COURT OF SOUTH AFRICA  
GAUTENG DIVISION, PRETORIA**

**CASE NO: 35801/19**

REPORTABLE: NO

OF INTEREST TO OTHER JUDGES: NO

NOT REVISED.

22 July 2022

In the matter between:

**N[....], S[....] L[....]**

**PLAINTIFF**

and

**THE MEMBER OF THE EXECUTIVE COUNCIL FOR HEALTH  
OF THE GAUTENG PROVINCIAL GOVERNMENT**

**DEFENDANT**

**JUDGMENT**

**FRANCIS-SUBBIAH, AJ:**

[1] Plaintiff sues the defendant for damages suffered by her minor child due to the alleged medical negligence of the defendant's employees. The child was born with an injury to the brain while under the care of the defendant in a public hospital. As a result of the brain injury the child suffers from spastic quadriplegic cerebral palsy, mental retardation and developmental delay.

[2] At the commencement of the trial, as agreed between the parties, the merits were separated from the quantum and only the issues relating to the defendant's liability is adjudicated upon. Quantum of damages, if arises, is postponed *sine die*. No special pleas by the defendant are pursued and the plaintiff does not proceed with the claim in her personal capacity.

[3] Plaintiff gave birth to the minor child on 25 January 2007 by vaginal delivery at the Charlotte Maxeke Johannesburg Academic Hospital. It is common cause that the child suffers from spastic quadriplegic cerebral palsy as a result of injury to his brain. The injury is described as hypoxic<sup>1</sup> ischemic encephalopathy (HIE, category II). This is a form of neurological dysfunction which is evident by a brain scan.

[4] This injury is alleged by the Plaintiff to have occurred in the intra-partum phase of labour due to the negligent care by the medical practitioners and nurses in the defendant's employ. They allegedly failed: -

- a) to properly monitor and assess the condition of the mother and unborn child and to administer appropriate medical treatment;
- b) to continuously monitor and assess the condition of the mother and unborn baby, under circumstances where it should have been done whilst the mother's labour was induced by the administration of Misoprostol;
- c) to cause the baby to be delivered expeditiously by means of caesarean section in the presence of foetal distress and a prolonged first stage of labour on 25 January 2007;
- d) to continuously monitor the foetal heart rate after CTG monitoring was non-reactive at 07h00 on 25 January 2007 to appropriately institute intrapartum resuscitation methods;

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<sup>1</sup>Hypoxia is the reduction of oxygen/ oxygen transfer resulting in tissues becoming damaged.

e) to properly and regularly monitor, assess and record the condition of the mother and unborn baby;

f) to recognize the risk of the baby developing brain damage in view of the extended period that he was subjected to foetal distress prior to his birth and to therefore monitor him appropriately;

g) to administer timeous and appropriate treatment to the baby having regard to the clinical symptoms which were observed and observable; yet continued to administer Syntocinon (oxytocin) for the induction of the mother's labour under circumstances where it was not safe to do so.

[5] The defendant admits legal duty to render medical services with reasonable care, skill and diligence in accordance with acceptable medical standards as accepted in Charlotte Maxeke Johannesburg Academic Hospital and subject to availability of resources at that hospital. In this regard, the defendant denies negligence and pleads that the injury to the baby may have been caused by other factors. These factors include: -

a) the baby being post- term, (a pregnancy beyond 41 weeks)<sup>2</sup>;

b) mother's HIV status;

c) mother having an infection of a yellow discharge a few days prior to delivery of the baby;

d) mother was a social drinker;

e) probability of a calcified placenta;

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<sup>2</sup> During trial it was accepted that delivery was 41 weeks 2 days.

- f) hypoxia relating to partial prolonged watershed brain injury may have occurred during pregnancy and before the onset of labour.
- g) late booker (she was 30 weeks when she began attending at the ante-natal clinic) and accordingly this may prevent early detection of any problem and subsequent intervention;
- h) absence of a sentinel event, a possible umbilical cord compression, therefore the brain injury was unavoidable; and
- i) no record of decelerations of the fetal heart rate.

[6] The defendant further recorded in the pre-trial minute of 13 October 2021 that the agreed opinions between the experts are not admitted by the defendant insofar as they relate to the cause and timing of the injury as well as the nexus between the injury and the cerebral palsy of the child. Further, there is no written record of foetal distress to conclude negligence on the part of the defendant.

[7] The plaintiff testified on her own behalf regarding the factual occurrences surrounding her labour and birth of her baby. The plaintiff called Professor J Anthony (obstetrician, gynaecologist and fetal maternal specialist) and Professor J Smith (paediatrician and neonatologist) to testify in support of her claim. Their evidence formed the factual foundation for the plaintiff's claims; that the defendant's employees were negligent, and that the negligent conduct caused the global hypoxic injury which the baby sustained during the labour process and by the time of his birth.

[8] Hospital staff Matron Valencia Mothwane, a midwife who was present and responsible for the care of the plaintiff in the labour ward from 09h00 to 19h00 on 25 January 2007, during the plaintiff's labour gave evidence led by the defendant. She had no independent recollection of the events, but confirmed her written accounts on the record. The defendant also called three expert witnesses Dr T Kamolane (radiologist),

Dr V R Mogashoa (paediatric neurologist) and Dr Mashamba, (obstetrician and gynaecologist). Dr Mathivha, a Neonatologist, expert for the defendant did not give evidence in court, although his expert opinions in joint minutes with Prof Smith were considered on the basis of agreements between the experts.

[9] Both the plaintiff and the defendant had engaged experts in the same field who met, agreed, prepared and signed joint minutes. Save in respect of Prof Anthony (expert for the plaintiff) and Dr Mashamba (expert for the defendant), differences in opinions surfaced. Dr Mashamba's responses in the joint minute and his expert report fails to provide explanatory detail which were not set out with the facts and reasons in a summary, as required by Rule 36 (9)(b) of the uniform rules of court, instead he stated that it will be explained in court. Hence failing to give the plaintiff's expert an opportunity to agree or disagree. As a result, long and lengthy testimonies and cross examinations ensued to accommodate this deficiency.

#### *The value of Pre-trial conferences and agreements*

[10] The Supreme Court of Appeal in *Bee v The Road Accident Fund*<sup>3</sup> confirmed the approach of how a Court deals with joint minutes and agreements between experts engaged by parties. Referring to the judgment in *Thomas v BD Sarens (Pty) Ltd*<sup>4</sup> the court summarised the relevant principles and stated that:-

*where the parties engage experts who investigate the facts, and where those experts meet and agree upon those facts, a litigant may not repudiate the agreement 'unless it does so clearly and, at the very latest, at the outset of the trial. In the absence of a timeous repudiation, the facts agreed by the experts enjoy the same status as facts which are common cause on the pleadings or facts agreed in a pre-trial conference. Where two or more experts reach agreement on a matter of opinion, the litigants are*

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<sup>3</sup> 2018 (4) SA 366 (SCA)

<sup>4</sup> [2012] ZAGPJHC 161

*likewise not at liberty to repudiate the agreement. The trial court is not bound to adopt the opinion but the circumstances in which it would not do so are likely to be rare.*<sup>5</sup>

[11] The court further expounded that: -

*[68] There may be cases where the expert rather than the litigant wishes to depart from what he or she previously agreed. The same rules of fair play apply. The expert should notify the attorney through whom he or she was engaged and due warning should be given to the other side. In such a case there will often be a further procedural requirement, namely the furnishing of a supplementary report by the expert whose views have changed.*

*[69] The limits on repudiation, particularly its timing, are matters for the trial court. The important point for present purposes is that repudiation must occur clearly and timeously. The reason for insisting on timeous repudiation is obvious. If the repudiation only occurs during the course of the trial, it might lead to a postponement to allow facts which were previously uncontentious to be further investigated. It might be necessary for a party to recall witnesses, including his or her expert. Whether a trial court would allow this disruption would depend on the circumstances. The trial court would be entitled to insist on a substantive application from the repudiating litigant.'*<sup>6</sup>

[12] In this regard during the trial the defendant was at liberty to bring a substantive application to address its repudiation of any agreements, retracting of its expert opinion as well as providing a summary of facts and reasons for probability of other causes not fully pleaded and canvassed in reports and joint minutes. Plaintiff's Counsel raised objections that served as a caution to the Defendant of the repercussions of not complying with the provisions of Rule 36(9)(b) of the Uniform Rules of Court and attempts to lead her final witness on expressing expert opinion on new medical literature not canvassed in expert summaries and put in cross examination to plaintiff's

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<sup>5</sup> at para 64 referring to Thomas at paras 11-13 *supra*

<sup>6</sup> *Bee v The Road Accident Fund supra* para 68- 69

experts. It is trite that fairness of court proceedings requires the adjudicator to control the proceedings and ensure resources are not wasted.

[13] The defendant had admitted and agreed on the facts in the pre-trial minutes, joint minutes of experts, and in the amended plea delivered on 19 October 2021, and accepted the plaintiff's evidence on this issue by it not being challenged in cross-examination, and despite the defendant's witness Matron Mothwane having confirmed these facts in her evidence. Belatedly and after the plaintiff had closed her case, defendant sought to withdraw from the admission that Dr Rashid saw the patient at 15h10 on 25 January 2007 and directed that she should have a caesarean section delivery. Defendant failed to take the appropriate steps and argued that the decision in *Bee* is not applicable because she should be heard on all her defences, including those not pleaded and without due notice given.

[14] The importance of *Bee* is aptly expressed in *Van Zyl N.O obo v MEC for Health*<sup>7</sup>, as

*Bee does not relate to the admissibility of expert opinions, but to the fairness of the trial. Expert opinion evidence should only be excluded when it impacts adversely on the latter.*<sup>8</sup>

[15] In evaluating expert evidence the purpose is to determine whether the opinion advanced by the experts are found on logical reasoning and, if so, to what extent. The examination of the opinions and the analysis of their essential reasoning assists the court in reaching its own conclusion on the issue. If the court concludes that the opinion is one that can reasonably be held on the basis of the facts and the chain of reasoning, the threshold will be satisfied. In this regard, the court in *Michael & Another v Linksfeld Park Clinic (Pty) Ltd & Another* stated that: -

*The assessment of medical risks and benefits is a matter of clinical judgment which the court would not normally be able to make without expert evidence and it would be*

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<sup>7</sup> [2022] ZAWCHC 133. Referring to *Bee* and English case *Huntley v Simmons* [2010] EWCA Civ 54

<sup>8</sup> Van Zyl at para 230

*wrong to decide a case by simple preference where there are conflicting views on either side, both capable of logical support. Only when opinion cannot be logically supported at all will it fail to provide 'the benchmark by reference to which the defendant's conduct falls to be assessed.'*<sup>9</sup>

### *The Injury*

[16] The Magnetic Resonance Imaging (MRI) scans are common cause radiological evidence by the experts of both plaintiff and defendant who concluded that a mixed pattern or combined form of injury to the brain occurred. In this regard two types of injuries to the brain are distinguished. An acute profound hypoxic ischaemic event which occurs suddenly and not progressive, it affects the grey deep structures of the brain and is called basal ganglia thalamic (BGT). The other type is the partial prolonged hypoxic ischaemic event which causes damage to the white matter or peripheral structures of the brain and occurs over a prolonged, progressive period of time. In the present case both types of injury occurred to the baby. The MRI scans show damage to both the grey matter and the white matter of the child's brain.

[17] Dr Kamolane, expert for the defendant, having performed an MRI on the child 14 years after birth reported that the radiological findings support a combined form of hypoxic insult (partial prolonged and acute profound) in a term baby. He testified that he was unable to determine a sentinel event. If a MRI was done within 3-5 days of life one could determine when the injury took place. He just interprets the image before him.

[18] Dr Mogashoa, the paediatric neurologist, expert for the defendant agreed that the impairments; cerebral palsy, mental retardation and marked developmental delay which the child suffers from were caused by intrapartum hypoxia and no other element contributed significantly to this outcome. Intrapartum period refers to the period from the onset of labour until delivery.

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<sup>9</sup> *Michael & Another v Linksfeld Park Clinic (Pty) Ltd & Another* 2001 (3) SA 1188 (SCA) at para 39



[19] Dr Lippert, a radiologist for the plaintiff further supported this finding, that the child has mild cerebral palsy of a mixed spastic dystonic type with a clumsy and proficient gait. His Apgar scores being 3/10 at 1 minute after birth, 4/10 at 5 minutes after birth, and 4/10 at 10 minutes after birth<sup>10</sup> were indicative of a neurologically depressed newborn infant and that the insult most likely occurred during the intrapartum period. One criteria for hypoxia is the baby is depressed at birth with low Apgar scores.

[20] Dr Mogashoa deferred to the obstetricians and neonatologist for expert opinion on the management of the intrapartum period. In this regard, both Prof Smith and Dr Mathiva, paediatricians and specialist neonatologists of both parties agreed that the Apgar scores were indicative of a severely neurologically depressed newborn baby and that the baby's condition had been compromised prior to delivery during the intrapartum period. As a result, the key issues in the trial were the cause and timing of the mixed pattern hypoxic ischemic brain injury suffered by the child.

#### *The medical record*

[21] The management of the intrapartum period is evident from the available discovered medical records. The medical records are incomplete. The cardiotocography (CTG)<sup>11</sup> tracings and the entries on the partogram, which record key data during labour made on 25 January 2007 are missing. This data includes foetal heart rate, contractions during labour and vital signs that would provide the essential detail of the progress of the plaintiff's labour and the condition of the unborn baby and signs of any distress.

[22] The defendant had initially taken the view that the CTG tracings and partogram existed and had been discovered. During the trial the court ordered the defendant to produce the partogram, non-stress test (NST) and CTG records. Dr Patricia Africa, medical doctor and clinical manager at the Charlotte Maxeke Johannesburg Academic

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<sup>10</sup> It became common cause during the trial that the contemporaneously recorded Apgar scores were correctly recorded. The Apgar score of 7/10 at 10 minutes appearing in the Road to Health Chart on the discharge of the baby had no source document and it was taken at face value by the evaluating experts.

<sup>11</sup> CTG is defined as a "continuous recording of the foetal heart rate obtained via an ultrasound transducer placed on the mother's abdomen. It is widely used in pregnancy as a method of assessing foetal well-being, predominantly in pregnancies with increased risk of complications."

Hospital (the Hospital) deposed to an affidavit that these documents are not available and have been destroyed without copies been made. She stated that the partogram must have been deliberately torn out of the book because they were not scanned on microfilm. Despite this, a diligent search no copies of the partogram, CTG and NST printouts were found.

[23] There is a duty on the defendant, more so an obligation upon the person in charge of a health establishment to keep records, protect such records and provide access to those records. This is legislated in sections 13 and 17 of the National Health Act 61 of 2003, which provide for the records of clinics and hospitals to be maintained and stored as prescribed.<sup>12</sup> In addition, the South African Maternity Guidelines of 2007 emphasize the importance of proper record keeping. The documents that were destroyed if produced would materially have shortened the trial.

[24] When statutory obligations are breached without reasonable explanations appropriate consequences are required. In *Khoza v MEC for Health and Social Development*<sup>13</sup>, Splig,J held that:-

*‘in summary the failure to produce the original medical records which are under a hospital’s control and where there is no acceptable explanation for its disappearance or alleged destruction*

*a) may result in the inadmissibility of ‘secondary’ evidence if the interests of justice so dictate, whether such evidence is of a witness who claims to have recalled the*

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<sup>12</sup> <sup>12</sup> It provides as follows: -

**13 Obligation to keep record**

*Subject to National Archives of South Africa Act, 1996 (Act 43 of 1996), and the Promotion of Access to Information Act, 2000 (Act 2 of 2000), the person in charge of a health establishment must ensure that a health record containing such information as may be prescribed is created and maintained at that health establishment for every user of health services.*

**17 Protection of health records**

*(1) The person in charge of a health establishment in possession of a user’s health records must set up control measures to prevent unauthorised access to those records and to the storage facility in which, or system by which, records are kept.*

<sup>13</sup> 2015 (3) SA 266 (GJ) para 47

*contents of the lost document or to have made a note of its contents on another document;*

*b) cannot of its own be used to support an argument that a plaintiff is unable to discharge the burden of proof because no one now knows whether the original records would exonerate the defendant's staff from a claim of negligence;*

*c) may result in the application of the doctrine of res ipsa loquitur<sup>14</sup> in an appropriate case;*

*d) may result in an adverse inference being drawn that the missing records support the plaintiff's case in matters where the defendant produces other contemporary documents that have been altered, contain manufactured data or are otherwise questionable irrespective of whether the evidence of secondary witnesses called in support is found to be unreliable or untruthful.'*

[25] The missing records are serious as it is prima facie proof of the truth of its content. In *Khoza*, the CTG tracings were not produced by the defendant, and the partogram had been altered. As a result, the secondary hearsay notes of the interpretation of the CTG tracings (original evidence) were held to be inadmissible and could not be relied upon by the defendant unless confirmed under oath by a witness. In the current matter, failure to produce these important documents could be prejudicial to the defendant and therefore could be the reason why they were destroyed. This contributes to an adverse inference on the treating doctor's assessment and management of the labour without producing the CTG tracings and partogram. There is no confirmation under oath of the treating doctor's assessment and remains inadmissible evidence.

[26] The narrative of what transpired is extracted from the available doctors and nurses' hospital records. The plaintiff was admitted to the hospital on 24 January 2007,

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<sup>14</sup> 'The nature of the negligence reasonably fits within the bounds of the defendant's duty to the plaintiff.'

where she was assessed by a midwife to be 40 weeks pregnant. Dr Mokotedi diagnosed her to be post-date at 41/40 weeks. The plaintiff was discussed with Dr Ramsamy and she was to be induced for labour. She was given the first dose of 10ml of misoprostol at 14h30. At 20h30 the dosage was increased to 20ml. At 23h00 the nursing note reports that the plaintiff's condition was stable, vital signs monitored and an NST was done and she still needs to be seen by a doctor.

[27] On 25 January 2007 at 06h45, the nursing progress report reflects that an NST was done and was not okay. Dr Koch was informed who made an entry on the antenatal admissions record that the NST was non-reactive. It was discussed with the Registrar on call and the plaintiff was to be transferred to labour ward for continuous foetal monitoring. The nursing report at 08h15 recorded that the plaintiff was having moderate contractions, with cervical dilation being 1cm and membranes were intact and NST was non-reactive.

[28] At 09h00 the foetal heart rate was recorded as 140 beats per minute (bpm) and cervix was 1cm dilated. Matron Mothwane testified that no CTG was available at 09h00 and she went around the ward to find one. Plaintiff was eventually put on a CTG monitoring machine at 10h00. A doctor's note at 10h00 records that the cervix was 1 cm dilated and the foetal heart rate was +/- 130bpm and will re-assess in 2 hours. At 12h30 the same doctor recorded that the foetal heart rate was 130bpm with good accelerations and no decelerations. Areas of loss of contact were recorded and that the plaintiff had mild pain. The plan was to re-assess later.

[29] The next recording refers to a time of '13h00' which is altered to read '15h00'. The '5' is written over the '3'. Matron Mothwane testified that the note was written by Dr Rashid but could not independently recall the time at which the note was made. Neither was Dr Rashid called to testify on the time of the note or its content. The note records that the NST was initially non-reactive and non-pathological and to re-assess in 2 hours.

[30] However, a further entry follows 10 minutes later and is made at 15h10. Matron Mothwane confirms this entry is made by her and that it is her handwriting. She recorded that the patient was seen by Dr Rashid and booked for a caesarean section. The consent was not yet signed and the pre-medication for caesarean was administered.

[31] When asked why such an entry was made, she could not recall instead explained the procedure that takes place when a decision for a caesarean section is taken. She testified that the same doctor speaks to the patient about the baby's condition and either the doctor or midwife makes the patient sign the consent. The very same doctor goes to theatre and lists the patient on the board. The midwife prepares the patient for the caesarean operation with an IV-line, catheter and waits for the theatre to call.

[32] At 16h00 there is an entry written in by Matron Mothwane which she confirmed she wrote. The entry records that Dr Rashid doesn't seem to know whether the plaintiff was booked for a caesarean section or not, accordingly "c/s not to be done". Matron Mothwane immediately went to Dr Rashid to show her the NST (CTG) because it was 'flat' and she was told to tell Dr Dube.

[33] At 16h50 Dr Dube was informed of the flat NST(CTG) and promised to come and check the patient. It is not documented at what time Dr Dube saw the patient. Matron Mothwane testified that if Dr Dube had come before 18h00 she would have seen.

[34] The next note is made at 19h20 after a gap of two and half hours. The records indicate that plaintiff was taken over by the night shift nursing staff. That she was 2cm dilated following the induction of labour with misoprostol. She was assessed by the night staff to be very distressed ("distressed ++"). The plaintiff was put on a CTG monitor and the tracing was non-reactive at the start.

[35] During an unrecorded time, Dr Dube examined the plaintiff and made a note that the CTG tracing was recorded to be reactive with no decelerations in the foetal heart

rate, which had a baseline of 140 bpm. Dr Dube prescribed the administration of Syntocinon (oxytocin) to further augment labour, administration of a sedative drug, and administration of Atarax. Dr Dube directed that the plaintiff be kept on continuous foetal monitoring.

[36] Syntocinon in a drip commenced at 19h30. Foetal heart rate of 121 bpm at 19h45 is recorded. At 19h45, within 15 minutes of the commencement of the Syntocinon infusion, the plaintiff's cervix from 2cm went to 6 – 7cm dilated. At 19h50 the plaintiff's membranes ruptured. After the administration of Syntocinon commenced, but before the Atarax was administered (i.e. between 19h30 and 20h10), the CTG started to be non-reactive and Dr Dube was notified and was "*still to see the patient*". The Atarax was administered intramuscularly with an injection at 20h10. Plaintiff was assessed to be very, very distressed ("distressed +++"). At 20h15 she was fully dilated 10cm and the second stage of labour commenced. The baby was delivered by normal vaginal delivery at 20h40 and the third stage of labour, being the expulsion of the placenta after birth, ended at 20h50.

[37] The hospital note at 21h00, describes the newborn baby as 'flat infant'. Due to his unsatisfactory condition, he was shown to his mother to identify his gender, and immediately transferred to the transitional unit. It was recorded that the placenta was delivered with apparently complete membranes. The final diagnosis on the discharge summary of the baby records 'birth asphyxia, convulsions and feeding difficulties.'

#### *Applicable Legal principles*

[38] Success of a delictual claim rests with the plaintiff establishing a link with probability that the wrongful act or omission of the defendant caused the injury.

[39] In *Lee v Minister of Correctional Services*,<sup>15</sup> the State's liability in delict for a negligent failure to put in place a reasonable system to guard against the contagion of

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<sup>15</sup> 2013 (2) SA 144 CC

tuberculosis among prisoners at the Pollsmoor Prison was questioned. In regard to causation the court held the following: -

*The point of departure is to have clarity on what causation is. This element of liability gives rise to two distinct enquiries. The first is a factual enquiry into whether the negligent act or omission caused the harm giving rise to the second enquiry, a juridical problem, arises. The question is then whether the negligent act or omission is linked to the harm sufficiently closely or directly for legal claim. If it did not, then that is the end of the matter. If it did, the liability to ensue or whether the harm is too remote. This is termed legal causation.*<sup>16</sup>

[40] The court further stated that: -

*although different theories have developed on causation, the one frequently employed by courts in determining factual causation, is the conditio sine qua non theory or but-for test. When the facts point to an omission, the 'but-for test' requires that a hypothetical positive act be inserted in the particular set of facts, the so-called mental removal of the defendant's omission. This means that reasonable conduct of the defendant would be inserted into the set of facts.*

*Whether an act can be identified as a cause depends on a conclusion drawn from available facts or evidence and relevant probabilities. Factual causation, unlike legal causation where the question of the remoteness of the consequences is considered, is not in itself a policy matter but rather a question of fact.*<sup>17</sup>

[41] Subsequent to the *Lee* decision, the Constitutional Court in *Mashongwa v Passenger Rail Agency of South Africa*,<sup>18</sup> dealt with a claim of negligence where a passenger was thrown out of a train having its doors open. The court held that on the basis of the traditional causation test, had the doors of the coach in which Mr Mashongwa was travelling been closed, it is more probable than not that he would not

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<sup>16</sup> *Supra* para 38

<sup>17</sup> *Supra* para 39

<sup>18</sup> 2016(3) SA 528 (CC) at para 66-67

have been thrown out of the train and sustained the injuries that led to the amputation of his leg. In the result the defendant was held liable for negligence.

[42] Limitless liability, however, cannot be imputed to a wrongdoer. In this regard it was stated in *Mashongwa* that: -

*The imputation of liability to the wrongdoer depends on whether the harmful conduct is too remotely connected to the harm caused or closely connected to it. When proximity has been established, then liability ought to be imputed to the wrongdoer provided policy considerations based on the norms and values of our Constitution and justice also point to the reasonableness of imputing liability to the defendant.*<sup>19</sup>

[43] Molemela, JA writing the dissent at the Supreme Court of Appeal in *AN V MEC for Health, Eastern Cape*,<sup>20</sup> succinctly explained the conclusion on causation that is set out in *Mashongwa* as follows:-

*“In re-stating the ‘but-for’ test in Mashongwa, the Constitutional Court settled the law on this aspect. It pointed out that the imputation of liability to the wrongdoer depends on whether the harmful conduct is either too remotely or sufficiently closely connected to the harm caused. It emphasised that where the traditional but-for test is adequate to establish a causal link, it may not be necessary to resort to the Lee test. It is the facts of the case that will dictate which test is more appropriate.”*<sup>21</sup>

[44] In the current matter, like *AN v MEC* the question must be asked: Would the injury to the brain of the baby been avoided if the hospital staff had acted appropriately on the non-reassuring foetal status that warranted urgent attention. Did the nursing staff or Dr Rashid or Dr Dube or any other Doctor not do something which was expected of him or her? If so, factual causation is established which is sufficiently and closely

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<sup>19</sup> At para 68

<sup>20</sup> *AN V MEC for Health, Eastern Cape* [2019] 4 All 1 (SCA)

<sup>21</sup> *Supra* at para 49



connected to the harm caused. This is in effect an allegation that “*but for*” the negligent conduct, the injury and consequent conditions would not have ensued.

*Causation- What caused the injury?*

[45] Foetal monitoring initially detects warning signals, but it is the treating doctor’s response to those warnings that raises inspection and scrutiny. Dr Mashamba argued that a change in the foetal condition was not observed because there were no decelerations in the foetal heart rate.

[46] Prof Anthony testified that ‘there is a critical distinction between knowing that the foetus is alive and knowing that there is foetal wellbeing. The fact that one can measure a foetal heartbeat at some point, indicates that the foetus is alive. If you want to know about foetal wellbeing, you need to know what the relationship is between the foetal heart rate and the uterine contractions that are taking place during labour. So there always needs to be measurement of the foetal heart rate before and then after the contraction to see whether there is any evidence of deceleration. The recording of a foetal heart rate in the nursing notes every hour cannot be interpreted without having known what was happening in relation to contractions. ‘The recording of those heart rates as stand-alone values is insufficient to allow an assessment to be made and is substandard care.’ The missing CTG tracings and partogram would have indicated the critical evidence.

[47] Prof Anthony explains that it is internationally accepted that cerebral palsy of the spastic quadriplegic type, presents as hypoxia in the intrapartum period. Such insults are associated with a period of foetal compensation that may last several hours during which time a diagnosis of a developing metabolic acidosis can be made and is the clinician’s window of opportunity for necessary intervention before the threshold of decompensation has been reached.

[48] In the baby the acidosis base deficit ( $> 12$  mmol/L) within the first hour or two of birth confirms that severe intrapartum hypoxia was present, but the amount of hypoxia

that may cause brain damage will vary from infant to infant. The brain damage would have started when an individual threshold was attained. Therefore, the degree of acidosis associated with the mixed pattern suggests that the damage to the brain became more and more likely as time passed. This opinion is further supported by Prof Smith and Dr Mathiva that the baby's nucleated red blood cell count determined approximately 2 ½ hours after birth is in keeping with either relatively short (up to 2 hours) or longer duration (but less than 24 hours) in utero hypoxaemia.

[49] In cross examination, Prof Anthony told the court that continued restriction in blood supply leads to a lack of oxygen supply. Blood from the mother through the placenta along the umbilical cord supplies oxygen to the brain of the baby. If the foetus does not get enough oxygen it moves into anaerobic metabolism and one gets the production of lactic acid which elicits autonomic responses and changes in the heart. This causes a slowing of the foetal heart rate. While the mother is in labour and her uterus was contracting, the abnormalities in the tracings which were being detected are those related to those features that would be indicative of hypoxia.

[50] During active labour if there is insufficient supply of blood carrying oxygen, the brain shunts the limited blood from the peripheries to the deep grey matter. This is designed to protect the deep grey matter which is the most vulnerable matter due to its higher metabolic rate. When shunting takes place, damage occurs to the white matter of the brain. This means that if there is some blood supply, but it is inadequate damage occurs to the white matter. If there is no blood supply at all, none is available to shunt to the deep grey matter. In this instance only the grey matter will be damaged. Where this takes place, bradycardia occurs.

[51] During contractions, intermittent interruptions to the blood supply to the brain occurs. Blood supply is restored between contractions. Hypoxia results from a sustained reduction in the supply of oxygen to the brain, resulting in the HIE injury to the baby. This has a cumulative effect which ultimately in the active phase of labour, during the bearing down, led to the damage in the deep grey matter called the basal ganglia

thalami (BGT) injury. The BGT is a very vital part of the brain in the sense that it sustains life in the cortex of the brain, responsible for regulating of blood pressure and heart rate. Severe injury resulting in loss of function of the basal ganglia of a foetus will result in death.

[52] The mixed pattern of brain injury as reported on by the neuroradiologists is incompatible with a sudden acute event described as a sentinel event. In this case, the injury to the BGT was not the first injury. The first injury was the partial prolonged watershed injury to the white matter of brain, followed by a profound BTG injury. The existence of the watershed injury implies that there was a lapse of time and a hypoxic process which began earlier in the labour, during which the foetus tried to compensate but failed to do so sufficiently to prevent subsequent injury to the BGT, which occurred towards the end of labour.

[53] After the Syntocinon (Oxytocin) infusion was put up at 19h30, there was a rapid evolution of labour under the influence of the oxytocin, the cervix dilated from 2 cm to 6 within 15 minutes and full dilatation 30 minutes thereafter. The foetus probably suffered a profound terminal insult to the BGT in that time. Under cross-examination, Dr Mashamba testified that the BGT injury probably occurred during the last part of the second stage of labour, and he agreed with Prof Anthony and Prof Smith on the mechanism and probable timing of the brain injury.

[54] Hypoxia is the golden thread that runs through all what happened in terms of the neuropathology, and if a partial prolonged hypoxia precedes the onset or exacerbation of more profound hypoxia, then the insult is going to occur more quickly and be more profound than it would do under other circumstances. In this case, one had hypoxia that developed gradually during the course of the labour, and which then worsened because of the onset of the second stage labour and the administration of oxytocin which caused an exacerbation in the hypoxia, and in the absence of a sentinel event that would be enough to have given rise to the BGT injury which occurred.

[55] Continuous CTG monitoring was directed by the treating doctor, because it is a useful tool for identifying a foetus that is becoming hypoxic and acidotic. It will not identify the point at which injury takes place, but it is indicative of an increased probability of adverse outcome. An abnormal pathological tracing is associated with a high probability of the baby being hypoxic and acidotic but will not inform one about the extent of that acidosis or hypoxia. It will not necessarily determine whether or not the particular foetus is going to end up with a hypoxic brain injury. The tracing simply indicates that there is a problem at that time related to the oxygen content of the blood, the hydrogen ion content of the blood, the amount of acid in the blood, and it is an indication for assessment and for an intervention in terms of intrauterine resuscitation or expedited delivery. The obligation on the part of the treating doctor is to react to what she sees in front of her, if what she sees in front of her is an abnormal tracing which is thought to have a high probability of an adverse outcome, then intervention is necessary.

[56] Prof Anthony further testified that if you diagnose foetal distress, or signs of foetal hypoxia, there are multiple ways in which you can intervene. You can intervene by trying to stop the contractions, which is the most important thing that you need to do. You can give the mother oxygen and you certainly will not let her go through the second stage of labour and bear down. The introduction of oxygen therapy at 19h20 is evidence of the fact that foetal distress and foetal hypoxemia, which is a progressive event, had developed during labour. It is not readily reversible simply by giving oxygen, as the mechanism giving rise to the hypoxia is the contractions themselves. So, there is a need to not only try and supplement the oxygen that the foetus gets, but also to stop the contractions. Giving oxygen to the mother by face mask will only result in about 3 % of the oxygen getting into the maternal blood, which is a very small amount, and will not resolve the problem. The fact that the CTG tracing improved was indicative of the fact that the diagnosis was probably correct, that this baby was hypoxic and acidotic.

*Sentinel event*

[57] Dr Mashamba took the view that for an acute profound injury to occur there must be a sentinel event, but a sentinel event is not noticeable. He explains that an occult cord prolapse is an un-identifiable sentinel event which cannot be detected after the baby has been born because the cord does not remain compressed when the pressure on it is alleviated. The medical article by Smith and others demonstrates that there can be a BTG injury in the absence of a sentinel event.<sup>22</sup> All experts, save for Dr Mashamba, took the expert opinion that the brain injury was caused by intrapartum hypoxia during labour in the absence of a sentinel event. Prof Smith testified that an undiagnosed compression of the umbilical cord alongside the head of the foetus resulting in total persistent occlusion of the blood supply to the fetus, is unlikely and if it occurred would probably result in a terminal bradycardia, of which there is no evidence in this case. It is therefore improbable that the profound BGT injury was caused by a cord compression. Dr Mashamba eventually under cross examination conceded and did not give any contrary evidence, that an 'occult cord' was a probable cause of the BGT injury in this case.

[58] Dr Kamolane also testified that on the child's MRI there was no touch congenital infection from mother to child's brain that is from placenta to baby during pregnancy, and therefore cannot detect a sentinel event on the MRI scan but BGT pattern was found in the child's brain scan. The brain stem controls heartbeat which appears normal on the MRI. If an MRI scan was done within 5 days from birth it could be detected when the injury occurred as the hospital has all the facilities to do.

[59] Dr Mashamba testified that if there is a progressive reduction in oxygen as a result of calcification of the placenta the foetus will suffer a partially prolonged insult. However, there is no evidence to suggest that there was a calcification of the placenta. The hospital notes record that the placenta was delivered with apparently complete membranes.

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<sup>22</sup> Smith, Solomons et al, 'Intrapartum Basal Ganglia- Thalamic Pattern Injury and Radiologically Termed "Acute Profound Hypoxic-Ischemic Brain Injury" Are not Synonymous' American Journal of Perinatology, November 2020

[60] Dr Mashamba said it is impossible to determine if the brain was not injured before labour. Prof Smith and Dr Mathiva had agreed in their joint opinion that there was no history of an identifiable intrapartum sentinel event such as a ruptured uterus, severe abruption placentae, umbilical cord prolapse, amniotic fluid embolus with coincident severe and prolonged maternal hypotension and hypoxaemia, maternal cardiovascular collapse and foetal exsanguination from either vasa praevia or massive foetal-maternal haemorrhage which could explain the outcome of neonatal encephalopathy and subsequent cerebral palsy. They further agreed that there is no evidence on the records in support of a congenital infection or early neonatal sepsis.

[61] Both Prof Anthony and Dr Mashamba agree there is importance for antenatal care and late booking is a factor associated with adverse outcome. And this is relevant in circumstances where pre-term birth is evident but in the current matter it is not the case. In the uncontested cross examination Prof Anthony said that the fact that there was also a watershed injury to the white matter is further evidence to the effect that this was likely a prolonged period of hypoxia in this labour, which allowed the redistributive blood flow to take place and the watershed injury to develop. He continued that it is very improbable that the hypoxic injury took place before the onset of labour during the pregnancy.

[62] On the question of social drinking, the plaintiff testified that once she became aware that she was pregnant she did not consume alcohol. Prof Anthony testified that he was not aware of any literature linking the neuropathology described in this case with the delivery of a hypoxic baby with the use of alcohol in pregnancy. He could not link the hypoxia that the baby suffered to the consumption of alcohol as a social drinker in this case.

[63] Defendant argued that the plaintiff had an infection when she visited the clinic, complaining of spontaneous rupture of membranes, and this could have led to brain damage to the child in utero. However, no reasonable proposition and evidence was put forth by the defendant on this score. At the clinic the plaintiff was examined by a

staff who found no evidence of ruptured membranes. There was evidence of a yellow vaginal discharge for which she was treated and sent home. Prof Anthony testified that the yellow discharge is indicative of a vaginitis and in addition thereto several days later when she was sent in for induction of labour there was no indication of any systemic infection. Plaintiff testified that they made her drink something to start the labour, if there was an infection it would be unlikely to have induced labour.

[64] Further to the submission of the defendant that the plaintiff may have had 'chorioamnionitis' which is regularly treated with antibiotics, Prof Anthony explained that a patient would usually be systemically ill by having a fever, tender abdomen and there is a likelihood that the patient will go into labour shortly after developing such an infection. Further the baby would show signs of foetal tachycardia (fast foetal heart rate) because the baby is in an infected environment. However, there was no evidence of any of that because the plaintiff had her labour induced and didn't go into labour on her own. Also, Dr Mogoshoa testified that the inflammatory markers did not suggest an infection.

[65] The child does not have HIV, but could the mother's HIV status, as an existing condition lead to the foetus having hypoxia? Dr Mashamba testified that the HIV of the mother and prolonged pregnancy is associated with adverse outcomes. Mogashoa deferred to obstetrics to the effects of maternal HIV on the status of the pregnancy.

[66] According to the Kennedy article<sup>23</sup> hypoxia is a significant risk in HIV infected women. Prof Anthony testified that the mother's HIV status should have raised the risk of HIE which confers an added responsibility on those who are providing intrapartum care. This would necessitate more careful monitoring during the labour and birth processes of any evident hypoxic change. It is not evident in the available treating doctors' notes whether they took this into account. There is no relevant and available evidence, tests or investigations carried out to establish the adverse effect of maternal

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<sup>23</sup> Kennedy et al, 'The effect of maternal HIV status on perinatal outcome at Mowbray Maternity Hospital and referring midwife obstetric units, Cape Town', South African Journal of Obstetrics and Gynaecology – January 2012, vol 18, No. 1

HIV to foetal hypoxia in the current case. Speculation is insufficient. In the Pretoria pilot study carried out, the authors also concluded in their research that 'the mechanisms linking HIV exposure in the womb with poor offspring development *in utero* and after birth remain unresolved.'<sup>24</sup>

[67] In *Price Waterhouse Coopers v National Potato Co-operative Ltd*<sup>25</sup> the court held that: -

*before any weight can be given to an expert's opinion, the facts upon which the opinion is based must be found to exist. As long as there is some admissible evidence on which the expert's testimony is based it cannot be ignored, but it follows that the more an expert relies on facts not in evidence, the weight given to his opinion will diminish. An opinion based on facts that are not in evidence in the matter has no value to the court. The opinions of expert witnesses involve the drawing of inferences from facts. Contextually and not isolation. The inferences must be reasonably capable of being drawn from those facts. If they are tenuous, or far-fetched, they cannot form the foundation for the court to make any finding of fact. In the process of reasoning the drawing of inferences from the facts must be based on admitted or proven facts and not matters of speculation.*<sup>26</sup>

### *Findings*

[68] When the factual evidence is compared with all opinions of the experts the most plausible inference is that the injury took place during the intrapartum period and highly probable not before that. There is no pre-existing condition of the plaintiff or the foetus, nor any event preceding the plaintiff's admission to hospital, which could constitute a probable cause of the hypoxic brain injury sustained by the baby prior to his birth on 25 January 2007.

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<sup>24</sup> White et al, 'Does *in utero* HIV-exposure influence infant development and immune outcomes? Findings from a pilot study in Pretoria, South Africa', *Medi Rx1v* (September 6<sup>th</sup> 2019) at 8

<sup>25</sup> [2015] 2 All SA 403 (SCA)

<sup>26</sup> *Supra* 326



[69] Having been booked into hospital on 24 January 2007 and assessed by a midwife at 12h20, the foetal condition was reassuring at the time. Plaintiff was under the care of the defendant for management of induced labour and delivery of her baby for a period of approximately 18 hours while there was foetal well-being. The first signs of concern were detected at 06h45 on 25 January 2007. Monitoring of the foetus was undertaken, correctly so under the Maternity Guidelines, as required. The CTG was reported to be non-reactive at various times. The non-reassuring foetal status was recorded in the hospital records at 15h10, 16h00, 16h50, 19h20, 19h30, 19h45.

[70] Matron Mothwane testified that continual foetal monitoring means that the patient should be left on the CTG recording machine and shouldn't be taken out of the machine as long as she's on induction with Misoprostol. The plaintiff testified that the machine was removed at a certain point. It was only put back later by the night shift staff.

[71] In closing arguments the defendant argued that there is no evidence of foetal distress recorded on the available records and the only references are made to foetal distress is the non-reactive NST or CTG and they were not pathological. Poor contact does not mean foetal distress. Dr Rashid as the treating doctor planned for reassessment in 2 hours and to be placed on continuous foetal monitoring. At 12h30 and 15h00 the NST was initially non-reactive but non-pathological. Defendant's submission is that probable cause was the foetus was sleeping. It is improbable that the foetus was sleeping because while the mother was in labour and her uterus was contracting under the influence of Misoprostol, the abnormalities being detected in the tracings by the nursing staff related to features that are indicative of hypoxia and not merely a sleeping foetus. The nursing staff were concerned and brought it to the attention of the treating doctors.

[72] At 15h10 on 25 January 2007, the plaintiff was seen by Dr Rashid, who decided that she should have a cesarean section delivery. The decision to do a cesarean section delivery was probably based upon an assessment of the presence of foetal distress made by Dr Rashid, whose responsibility it was as the treating doctor, to decide

whether a cesarean section delivery should be done. The South African Maternity Guidelines 2007 indicates the circumstances when a caesarean section should be performed, when there is a failed induction of labour or foetal distress as is present in this case. The plaintiff was prepared by Matron Mothwane for theatre by the administration of pre-medication and shaved. Plaintiff had testified to being shaved for the caesarean surgical incision.

[73] At 16h00 on 25 January 2007, the CTG tracing was flat, poor variability and no accelerations, which means there was a loss of beat to beat variability in the foetal heart rate, indicative of possible foetal distress. Matron Mothwane showed the flat tracing to Dr Rashid, who told her to tell Dr Dube, who came on duty at 16h00. The flat CTG tracing was a concern to both Dr Rashid and Matron Mothwane. The matron as an experienced midwife for 7 years at the time, testified that the cause of a flat CTG is either decreased blood flow to the uterus or to the foetus.

[74] A cesarean section was planned by Dr Rashid at 15h10 on 25 January 2007, but never followed up and the indication (or reason) for cesarean section was not documented. The hospital falls into the category of level 1 hospitals that have properly trained and qualified doctors and nurses, medical equipment, 24-hour labour and delivery service and a theatre to provide proper obstetric care. It is also a major regional teaching hospital and the reasonable and achievable period from decision to do a cesarean section delivery to the actual delivery should not exceed 1 hour, according to the evidence of Dr Mashamba and Prof Anthony.

[75] The delivery should therefore have taken place by no later than 16h10. The cesarean section was not performed. There is no explanation in the hospital records or by oral evidence as to why the cesarean section was not performed. The defendant failed to call Dr Rashid, as a witness, who had knowledge and was in the defendant's employ at the time.<sup>27</sup> The probable inference is that there is no justifiable explanation

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<sup>27</sup> *Raliphaswa v Mugivhi and others* 2008(4) SA 154 (SCA) where failure to call a witness can have an adverse inference in particular circumstances.

other than a negligent failure to perform the caesarean section delivery, despite the clinical judgment and decision by Dr Rashid that it should be performed.

[76] Even at 16h50, the NST was reported as flat which is a sign that something is not well with the baby which was another window of opportunity for a caesarean section to be performed. Had the baby been delivered by caesarian section as suggested by Dr Rashid at 15h10, the experts are of the opinion the injury would probably not have resulted. Even if the baby was delivered by caesarean section as soon as possible after 16h50 which was still an option before 19h30, the deep grey matter injury would not have occurred. The risk of hypoxic damage would have been less and the risk proportional to the time passed.

[77] Dr Mashamba in his written report expressed the expert opinion that a cesarean section delivery could and should have been carried out by 16h10, but certainly by no later than 16h50. Although he retracted his opinion in evidence in chief based on him not being given the full record. There are no reasons why the delivery of the baby was not expedited by caesarean section since the defendant took the view that the pregnancy was post-date, induced using Misoprostol and later Syntocinon (Oxytocin), both of which are drugs known to be associated with an increased risk of uterine tachysystole leading to foetal hypoxia. Dr Mashamba himself told the court that the risk factor of any drug can lead to hyper stimulation and slow progress in induced-labour is concerning because response to drugs is unpredictable.

[78] The experts testified that the foetus suffered partial prolonged hypoxia during the period from about 15h10 to 19h30 on 25 January 2007, resulting in a progressive white matter ("watershed") injury to the brain. This partial prolonged hypoxia also caused the threshold or ability of the foetus to cope with the hypoxia, by shunting blood to the brain and vital organs being diminished and impaired. Plaintiff testified that she was experiencing severe pain which caused her to scream, and it felt to her as if she had no time to breathe between contractions. According to the medical experts, this suggests strong uterine contractions which result in a restriction of blood flow to the foetus

through the placenta, resulting in a relative or partial hypoxia of the foetal brain, which recovers to an extent between contractions as blood flow is restored. The stronger and more frequent the contractions, the greater the restriction in blood flow to the foetus, and the greater the degree of hypoxia to which the foetal brain is subjected.

[79] From 19h20, no steps were taken to protect the foetus, such as the administration of a tocolytic drug to stop the uterine contractions while preparing for a cesarean section delivery, but an intravenous administration of Syntocinon was commenced, which increased the strength and frequency of the contractions and added to the foetal hypoxia. The partial prolonged hypoxia continued after 19h30, and the foetus sustained a further hypoxic injury to the deep grey matter of the brain (a BGT injury) during the period from 19h30 and the birth at 20h40 and according to Dr Mashamba most probably during 2nd stage of labour between 20h15 and 20h40.

[80] Prof Smith and Dr Mathiva agreed that if the cervix will normally dilate at about 1cm per hour in the active phase of labour, and the second stage of labour is normally up to 2 hours' duration. After the administration of Syntocinon the plaintiff progressed from a cervical dilatation of 2 cm at 19h20/19h30, to 6/7 cm at 19h45, and to full dilatation (10 cm) by 20h15. This is 8cm in under an hour and clear evidence of tachysystole.

[81] The experts agree that generally a human foetus could withstand about 10 minutes of acute hypoxia. This 10-minute rule originates from the Rennie and Rosenbloom's article where the experiments conducted on animals indicated that an acute profound insult happens in approximately 10 minutes.<sup>28</sup> The foetus must be delivered within 10 minutes from onset of bradycardia to avoid brain injury. From the time of brain injury, it takes 30 min for the foetus to survive in utero depending on the severity of the insult and the threshold of such a foetus.

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<sup>28</sup> Rennie J, Rosenbloom L. 'How long have we got to get the baby out? A review of the effects of acute and profound intrapartum hypoxia and ischaemia' Royal College of Obstetrician and Gynaecologist (2011) 13: 169

[82] The drugs were used inappropriately because they were used persistently despite the advent of an active labour and may also have acted synergistically since Syntocinon was given following doses of Misoprostol even if it was hours later. The drugs were given despite evidence of an abnormal CTG tracing. The drugs were continued even after intrauterine foetal resuscitation commenced. Dr Dube prescribed the intravenous administration of Syntocinon to augment labour at about 19h30 and at a time when the plaintiff was already experiencing strong and frequent uterine contractions and severe pain, and Dr Dube and the nursing personnel continued the administration of Syntocinon, under circumstances where it was not safe to do so and its foreseeable results brought about an increase in the strength and frequency of contractions and consequent foetal hypoxia.

[83] Further, when Syntocinon is used, the maternal guidelines instruct that 'there must be no evidence of foetal distress.' In this case the alarm bells were ringing from 06h45 and intermittently through-out the day before Syntocinon was prescribed and given at 19h30. Dr Dube had to ensure there was no foetal distress which s/he failed to do. Dr Mashamba testified that the risk factor of any drug can lead to hyper stimulation and slow progress in induced labour is concerning because response to drugs is unpredictable.

[84] It was not established exactly what time Dr Dube did see the patient. Matron Mothwane testified that if Dr Dube had come before 18h00 she would have seen. This is unlike a reasonable treating obstetrician in the current circumstances and is gross negligence.

[85] One of the important consequences is the note by Dr Dube, that the FHR and CTG tracing on 25 January 2007 was normal with no actual decelerations, is improbable. There is no partogram to show the heart rate before and after a contraction, we only have a narrative. It is further probable that Dr Dube relied on a stand-alone foetal heart rate without assessing the heart rate before and after a contraction to determine a deceleration. This is sub-standard care. Dr Mashamba also agreed without

CTG tracings one cannot tell if the interpretation was wrong. Especially since it was an induced labour and caution was raised since 06h45. It is evident in the result of a hypoxic baby that a 15- point drop is a deceleration, even if it is above 110bpm. The evidence of Prof Anthony on this point was not challenged by the defendant.

[86] The non-reassuring foetal status warranted urgent attention. The condition of the baby at birth was consistent with intrapartum hypoxia, and the most probable cause of the baby's condition at birth. The nursing personnel and medical doctors failed to react to the suspicious CTG tracings indicative of possible foetal distress, to act thereto appropriately which the law expects of him or her to and to institute appropriate intrapartum resuscitation methods, including immediate and continuous administration of oxygen to the plaintiff, placing the plaintiff on her left side, administering a tocolytic drug to stop uterine contractions, and ensuring an expedited cesarean section delivery.

[87] Once the inference of negligence has been drawn, the defendant may offer an explanation of how the accident occurred. Such an explanation must be reasonable, and not speculative. Ogilvie Thompson JA held in *Arthur Bezuidenhout and Mieny* that '*the onus rests on the defendant to establish the correctness of his explanation on a balance of probabilities.*'<sup>29</sup> The destroyed CTG tracings would have provided critical evidence bearing the truth of its content. Failure to produce it supports the plaintiff's case as explained at paragraph 25. The fact that the defendant failed to lead the evidence of Dr Dube or Dr Rashid who were in her employ and no reasons are advanced for their decisions is unacceptable and fails to discharge the onus placed on the defendant.

[88] Dr Mashamba did not persist that the brain injuries may have been caused by factors other than intrapartum hypoxia and could not have occurred without there having been a sentinel event. He agreed with the opinions of Prof Anthony and Prof Smith as to the probable mechanism and timing of the partial prolonged hypoxic brain injury followed by a more profound brain injury to the BGT region of the child's brain.

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<sup>29</sup> *Arthur v Bezuidenhout and Mieny* 1962 (2) SA 566 (A)

[89] The court in *SARFU v President of SA* held that there is a duty to cross-examine which ‘must obviously not be applied in a mechanical way but always with due regard to all facts and circumstances of each case’ and referred to *Browne v Dunn* where it was held that the rule is ‘essential to fair play and fair dealing with witnesses’.<sup>30</sup> The consequences of a failure to fully and effectively challenge the evidence of a witness in cross-examination, is that the plaintiff is entitled to assume that the unchallenged witness’s testimony is accepted as correct.<sup>31</sup> Defendant’s Counsel failed to fully and effectively challenge the evidence of all three witnesses of the plaintiff. The defendant unreasonably prolonged the trial by repetitive and irrelevant cross-examination of Prof Anthony and Prof Smith, without challenging the essence of their opinion evidence in the manner required in terms of the principles set out in *SARFU v President of the RSA*.

[90] I find that the plaintiff has established on a balance of probabilities, that the defendant’s employees were responsible for the care and treatment of the plaintiff and her child and that they were negligent as expounded at paragraphs 68 to 88 above. The negligence caused the hypoxic brain injury sustained by the child in that, but for the negligent conduct, the child would not have sustained the global hypoxic (mixed pattern) brain injury and the consequent condition of spastic quadriplegic cerebral palsy.

#### Costs

[91] There is no reason to depart from the principle of costs following the cause, as the Plaintiff has succeeded to prove on a balance of probabilities on the evidence that the most probable cause of injury to the baby, was caused in the intrapartum period by the negligence of the defendant’s employees.

[92] The parties having set the matter down for trial exceeded their allocated time and were indulged to ventilate the defendants’ further causes including those that were not pleaded. In this regard, a substantial amount of the trial time was taken by the

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<sup>30</sup> *Supra* para 65, 62 referring to the rule as held in *Browne v Dunn* (1893) 6 The reports 67 (HL).

<sup>31</sup> *President of the RSA v SARFU* 2000(1) SA 1 (CC) paras 61 and 63.

defendant for this purpose. The defendant prolonged the trial by repetitive and at times irrelevant cross-examination of Prof Anthony and Prof Smith, without challenging the essence of their opinion evidence. The same was done to defendant's witness Dr Mashamba, by leading his evidence in chief over a period of several days on matters not in dispute and on issues on which he agreed with the testimony and opinions of Prof Anthony. He was taken at length through his and Prof Anthony's joint minute to confirm in respect of most of what Prof Anthony had stated, that he agrees with or does not differ from Prof Anthony. The same result could have been achieved by leading him only on matters which he disputed and disagreed with. This resulted in prolonging the trial unnecessarily and such prejudice to the plaintiff is appropriately addressed with an attorney and client cost award.

*Application for leave to amend the Defendant's Plea*

[93] The defendant upon the close of the defendant's case brought an application for leave to amend its plea with an offer to pay the plaintiff's costs. The reason for the amendment is the defendant had mistakenly pleaded admitting that Dr Rashid had booked the patient for a caesarean section whereas there is no note in the handwriting of Dr Rashid that the patient be booked for a cesarean section. The note is in the handwriting of Matron Mothwane as confirmed by her in testimony. It is for this reason to set the record straight that the defendant seeks leave to amend its plea by deleting pleaded paragraph 14.2 that reads: -

'subject to what has been pleaded above, the defendant takes no issue with the remainder of the allegations made herein.'

[94] Irrespective of who wrote the note in the hospital records, it indicates a cesarean section was planned and such a decision is only taken by a treating doctor and not a midwife. Even if the paragraph is removed, it does not take away the evidence of a planned caesarean section and the admissions made in the signed pre-trial minute of the parties. The witness of the defendant was not discredited to have lied about the caesarean section. The application is made when the parties have closed their case



and informed the court that it will not be re-opening its case and therefore can no longer add any benefit to the trial. 'Pleadings are made for the court and not the court for the pleadings.'<sup>32</sup> Issues pleaded have long been canvassed and expanded. Proverbially 'the horse has bolted'. Section 15 of the Civil Proceedings Evidence Act 25 of 1965, provides that it is neither necessary for a party to prove, nor competent to disprove, a fact admitted on the record of any civil proceedings. No convincing reason for leave to amend in these circumstances are present and in my view the court shall not encourage amendments that are merely technical. The application for leave to amend the plea is dismissed with costs.

**[95] The order is as follows: -**

[95.1] In terms of the provisions of Rule 33(4) the issues arising from the following paragraphs of the plaintiff's amended particulars of claim and the defendant's plea thereto are hereby separated for initial determination

1.1. Paragraphs 1, 2, 3, 4, 5, 6, 7, the introductory portion of paragraph 8 up to: "... A[...] suffered the injury and consequent conditions" and paragraph 11 of the amended particulars of claim, and

1.2. Paragraphs 17; 18; 19; 20; 21; 22 and 23 of the defendant's plea insofar as it relates to the paragraphs set out above of the plaintiff's amended particulars of claim.

1.3. The remaining disputes as defined in the pleadings are postponed *sine die*;

[95.2] The defendant is ordered to pay 100% of the plaintiff's proven or agreed damages in her representative capacity as parent of the minor child A[...] N[...] who was born on 25 January 2007, suffered as a result of the injury sustained by them and consequences as pleaded in the paragraphs of the particulars of claim referred to in paragraph 1.1. above. The damages amount, once determined, shall be paid to the credit of the plaintiff's attorney's trust account, details of which are the following:

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<sup>32</sup> *Shill v Milner* 1937 AD 101 105

**NAME: JOSEPH'S INC, TRUST ACCOUNT**

**BANK NAME: RMB PRIVATE BANK, JOHANNESBURG**

**ACCOUNT NO: 5[...]**

**BRANCH NO: 2[...]**

[95.3] The defendant is ordered to pay the plaintiff's taxed or agreed attorney and client costs on the High Court scale up to date of this order, which costs will include but will not be limited to:

3.1. The costs consequent upon the obtaining of the medico legal reports and expert summaries and the reasonable qualifying fees (if any) of:

3.1.1. Dr M. van Rensburg, neuro-radiologist

3.1.2. Prof J. Smith, neonatologist

3.1.3. Dr M.M. Lippert, paediatrician and paediatric neurologist

3.1.4. Prof J Anthony, maternal and foetal specialist

3.1.5. Prof D. du Plessis, nursing expert

3.1.6. Dr G.S. Gericke, paediatrician and geneticist

3.1.7. Prof J.W. Lotz, neuro-radiologist of whom the plaintiff has given notice in terms of the provisions of Rule 36(9)(a) and (b);

3.2. The costs consequent upon the employment of two counsel.

[95.4] The following provisions shall apply regarding the determination and payment of the plaintiff's abovementioned taxed costs:

4.1. the plaintiff's attorney shall timeously serve the notice of taxation on the defendant's attorneys of record;

4.2. the plaintiff's attorney shall allow the defendant 60 (SIXTY) calendar days to make payment of the taxed costs from date of settlement or taxation thereof;

4.3. should payment of the plaintiff's taxed or agreed costs not be effected timeously, the plaintiff will be entitled to recover interest at the mora interest rate, calculated from the 31<sup>st</sup> calendar day, after the date of the Taxing Master's *allocatur*, or after the date of settlement of costs, up to the date of final payment.

[95.5] The Minister of Health litigates using public funds and there is a duty upon this department to be aware of the manner in which litigation is advanced on its behalf. The Registrar of the Gauteng Division, Pretoria is ordered to bring this matter under the attention of the Office of the Health Ombud and the Office of the Health Standards Compliance.

**R. FRANCIS-SUBBIAH, AJ**  
**THE GAUTENG DIVISION OF THE HIGH COURT, PRETORIA**

**APPEARANCES:**

Counsel for the Plaintiff:	Adv. NGD Maritz SC Adv. MM Lingenfelder SC
Instructed by:	Joseph's Incorporated
Counsel for the Respondent:	Adv. N Manaka Adv. I Tshoma

Instructed by:	The State Attorney
Date of hearing:	25-29 October 2021
2– 5 November 2021	
	24-28 January 2022
	23-27 May 2022
	23 June 2022
Date of Judgment:	22 July 2022