REPUBLIC OF SOUTH AFRICA
IN THE HIGH COURT OF SOUTH AFRICA
GAUTENG LOCAL DIVISION, JOHANNESBURG

CASE NO: 2014/6003

REPORTABLE: YES
OF INTEREST TO OTHER JUDGES: YES
REVISED. NO
DATE: 19 March 2021

In the matter between:

PG obo TG Plaintiff

and

THE MEC FOR HEALTH, GAUTENG PROVINCE Defendant

JUDGMENT

Weiner J

Introduction

[1] Claims for medical negligence against the state rose to a staggering R104.5 billion in 2019, as patients seek redress for harm allegedly wrought by SA's overstretched public health system.[1] This case is another which comes before this court on the basis of the defendant's alleged medical negligence.

[1] Article in Business Live, dated 27 January 2020, titled 'Medical negligence claims against the state soar to R104bn' accessed at https://www.businesslive.co.za/bd/national/health/2020-01-27-medical-negligence-claims-against-the-state-soar-to-r104bn/.

[2] The plaintiff, as the mother and natural guardian of her minor child (TG) who was born on 22 November 2004 at the Mofolo Clinic (the Clinic) sought, in such capacity, to claim, against the defendant, damages suffered as a result of the alleged negligence of the medical and/or nursing staff (the defendant's employees) during the plaintiff's labour and delivery of TG at the Clinic. This resulted in TG suffering an hypoxic ischemic injury (HIE) resulting in neonatal encephalopathy (NE) and cerebral palsy (CP).

[3] The specific acts of negligence alleged are that the defendant's employees:

(a) failed to properly, sufficiently or adequately assess the plaintiff's stage of labour after her admission to the Clinic;
(b) failed to monitor the progress of the plaintiff's labour and the foetal well-being with sufficient regularity during the plaintiff's labour;
(c) failed to detect that the foetus was in foetal distress during the plaintiff's labour after admission to the Clinic;
(d) failed to timeously take appropriate and effective action to prevent further distress in the foetus or prevent the foetus from suffering any harm;
(e) intervened inappropriately and dangerously when they realised that TG was in distress by applying fundal pressure to deliver the baby.

[4] It is claimed that TG's condition is due to the negligent omissions and actions of the defendant's employees in circumstances in which such conditions were preventable.

[5] The defendant defended the claim by denying that any of its employees acted negligently, or that TG sustained an injury while the plaintiff was in labour and/or when he was delivered.

[6] In addition, the defendant pleaded that, even if negligence was proved, there was no causal connection between the negligence alleged and the CP suffered by

TG. The defendant submitted that the plaintiff was required to show that the CP was a product of an intrapartum insult caused by and/or connected to such negligence.[2]

The lack of medical records

[7] At the trial, the defendant's counsel submitted that the plaintiff could not prove her case as the Maternity Case record, including that of the antenatal period and intrapartum period, and the neonatal records, were missing and/or lost. These were accordingly not made available by the defendant. No further explanation was proffered by the defendant in this regard. In the defendant's argument, the submission was made that as there were no maternal records, the plaintiff could not show that the treatment she received was substandard and that it caused the injury to TG. This somewhat cynical attitude has been displayed by MECs in various cases in this, and other divisions, of our courts.

[8] In *Khoza v MEC for Health and Social Development, Gauteng,*[3] Spilg J set out possible implications of such missing records:

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

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[2] In relation to causation, Gorven AJA, writing for the majority, in *AN obo EN v Member of the Executive Council for Health, Eastern Cape* [2019] 4 All SA 1 (SCA) stated at paras 3-4:
'… The wrongful conduct must cause the wronged person to suffer loss. The first step in proving this is to prove that the wrongful conduct of the staff caused the baby to suffer brain damage. The appellant accordingly bore an onus to prove this. Wrongfulness should not be conflated with factual causation.
The test for factual causation is whether the act or omission of the defendant has been proved to have caused or materially contributed to the harm suffered. Where the defendant has negligently breached a legal duty and the plaintiff has suffered harm, it must still be proved that the breach is what caused the harm suffered….'

[3] *Khoza v MEC for Health and Social Development, Gauteng* 2015 (3) SA 266 (GJ) para 47.

(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* 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 contemporaneous 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.'

[9] In *Madida obo M v MEC for Health for the Province of Kwa-Zulu Natal*,[4] Pillay J discussed this issue as follows:

'In terms of ss 13 and 17 of the National Health Act 61 of 2003, the defendant's employees have a statutory duty to preserve and protect such hospital and medical records. Failure to do so opens the defendant's employees to criminal prosecution and liable on conviction to a fine or to imprisonment for a period not exceeding one year or to both such fine and imprisonment.
The Health Professions Council's Guidelines on the keeping of patient records dated May 2008 applies to health care practitioners in both the private and public service. It identifies what constitutes health records, why documents or materials should be retained and what information is compulsory for recording. It prohibits alteration of records and requires reasons for any amendments to be specified on the record. Errors may be corrected but the date of the change must be entered and the correction signed in full. The original record must remain intact and fully legible. Additional entries at a later date must be dated and signed in full. The guidelines also provide for the retention of health records, which must be stored in a safe place and if stored electronically then safeguarded by passwords. In the case of minors, their records must be kept until the minor's twenty-first birthday. For mentally incompetent patients the records must be kept for the duration of the patient's life.

[4] *Madida obo M v MEC for Health for the Province of Kwa-Zulu Natal* [2016] ZAKZPHC 27 paras 10-12.

Health records kept in a provincial hospital or clinic including the records of minors and mentally incompetent patients may only be destroyed with the authority of the Deputy Director General concerned.

I have detailed the National Health Act and Guidelines to emphasise their importance and the rationale and seriousness with which the health professions view the keeping of patients' records. So when they are not available when they should be there is potentially a breach of a rule of law and codes of good practice. Non-compliance with statutory requirements and codes of good practice that impact directly on the health of members of the public is cause on its own to refuse the adjournment. To do otherwise would lead to the mistaken inference that the court is prepared to condone or tolerate the illegality. The lack of a bona fide explanation for the unavailability of the records fortifies my opinion.'

[10] These remarks are pertinent in the present case, where the defendant, despite the absence of such records, pleaded a bare denial that its employees were negligent. As stated by Pillay J in *Madida*—

'To plead "no knowledge" and to put the plaintiff to the proof of facts that should be easily ascertainable was not a plea in good faith. It is hardly the response of a caring health service. Proof as to whether a medical doctor had attended to the plaintiff had to come from the hospital staff on duty at the time and from their records….'[5]

[11] In the present case, the only records that were discovered were:

(a) The birth register which showed:

i. The name of the plaintiff, date, and time of admission at 07h15 on 22 November 2004.
ii. The plaintiff tested negative for syphilis, positive for HIV, and she was not anaemic.
iii. A male child TG was delivered at 13h00 and both the mother and child were given a dose of nevirapine to prevent mother to child transmission of the HIV virus.
iv. Meconium Stained Liquor was noted when TG was delivered.

[5] Ibid para 20.

v. Meconium Aspiration.
vi. Apgar scores of 6/10 at one minute and 8/10 at ten minutes.
vii. TG had been immunised against polio and given a BCG inoculation at birth.

(b) The Neonatal discharge summary which showed that:
i. The Apgar scores were 6/10 at one minute and 8/10 at five minutes.
ii. Reference to body proportions/measurements at birth being normal (appropriate) for the gestation of 38 weeks.

(c) The Road to Health record was relied upon as a secondary factual record.

Witnesses

[12] The following expert witnesses were called to give evidence in support of the plaintiff's case:

(a)	Dr K Frank	Obstetrician Gynaecologist
(b)	Dr George Gericke	Paediatrician and Geneticist
(c)	Professor R.Solomons	Paediatric Neurologist
(d)	Prof J Smith	Neonatologist/ Paediatrician
(e)	Prof I G Nolte	Nursing Expert

[13] A radiologist appointed by the plaintiff, Dr Ranchod, compiled a radiology report and joint minute together with the defendant's radiologist, Dr Weinstein, which was accepted into evidence without the radiology experts having to give evidence. It was accepted that the MRI brain scan dated 30 June 2017 demonstrated features consistent with chronic sequela of a partial prolonged HIE.

[14] The defendant called the following expert witnesses:

(d)	Professor D Du Plessis	Nursing Expert
(e)	Professor K D Bolton	Paediatrician
(f)	Dr Koll	Obstetrician Gynaecologist
(g)	Dr V M Mogashoa	Paediatric Neurologist
(h)	Professor A L Christianson	Geneticist

[15] The plaintiff testified to the facts in the matter. Sister Elizabeth Nomathansanqa Ndlela (Sister Ndlela), called by the defendant, was the midwife who apparently attended to the plaintiff. She had no independent recollection of the plaintiff's case. Her evidence consisted of a description of how nurses in her position conduct themselves during a patient's labour. She doubted that the plaintiff's version of events was accurate. She however confirmed that the birth register referred to birth Asphyxia (BA), and Meconium Aspiration Syndrome (MAS) and Meconium Stained Liquor (MSL) and the Apgar scores as 6/10 after resuscitation and 8/10 at five minutes. She conceded that it was possible that the plaintiff and the foetus may not have been monitored for a period of two hours. This, she stated, could occur if the nurses were busy with other patients. She could not recall if this occurred in plaintiff's case. She agreed that the fact that TG's condition was poor, and they referred him to the Chris Hani Baragwanath Hospital (CHBH) meant that his situation was serious.

[16] It was submitted by plaintiff's counsel that, in the circumstances, the plaintiff was the only factual witness and that the Court would have to make a determination in respect of the veracity or reliability of the evidence of the plaintiff, in deciding the merits of the case.

Common cause or agreed issues

[17] The plaintiff was a primigravida (giving birth for the first time) at the time of the birth of TG on 22 November 2004. The plaintiff was admitted into the labour ward at the Clinic at approximately 07h15 on that day, her labour having already started and having progressed to the active phase of labour (her cervix had already dilated more than four centimetres).

[18] The baby was delivered by vaginal birth at approximately 13h00. It is recorded in the birth register that the baby suffered from birth asphyxia as a complication at birth. TG was born at full term and his anthropometric measurements and weight were within the normal range for a term baby according to the Road to Health Chart and the mode of delivery.

[19] TG required resuscitation and oxygen at birth and was transferred to CHBH on the same day after a delay of approximately three hours. The reason for the transfer to CHBH was recorded as low Apgar scores and birth asphyxia. TG was admitted to the Neonatal Intensive Care Unit (NICU) immediately on arrival at CHBH and remained in the NICU for at least three days, whereafter he remained in hospital for a further three weeks.

[20] TG also presented with a subaponeurotic haemorrhage (bleeding into the skull) at birth, which resolved during TG's stay in CHBH.

[21] TG presented with feeding difficulties during his admission in the NICU and further stay in CHBH, in keeping with the consequences of birth asphyxia on the neonatal brain, and consistent with the sequela of NE.

[22] TG subsequently developed epilepsy, which is indicative of an encephalopathy. TG now suffers from mixed spastic CP and microcephaly with profound intellectual disability, as well as a mixed picture of asymmetric spastic quadriplegia and dystonia.

[23] The MRI brain scan dated 30 June 2017 demonstrates features consistent with chronic sequela of a partial prolonged HIE. There are no stigmata of intracranial syndromic disorder and there are no features to suggest complicated intracranial sepsis (infection). The implication of this is that the child does not suffer from a brain injury which was caused by HIV infection or AIDS, or inflammatory infections such as meningitis or syphilis.

[24] Although the plaintiff was HIV+, TG is not HIV infected and the plaintiff had been given a single dose of nevirapine to prevent mother to child transmission of the HIV virus, as was the practice at the time. There is sufficient evidence for timing of the partial prolonged HIE to the intrapartum period, birth asphyxia having been recorded in the discharge summary and notes. The paediatric neurologists for both parties are in agreement in this regard.

[25] There is no recorded evidence for the presence of the histological markers of Fetal Inflammatory Response Syndrome (FIRS) i.e. inflammation of the umbilical cord, acute chorioamnionitis, or chorionic villi vasculitis – all indicators of a pathological placental condition.

[26] Clinically, no genetic predisposing familial or antenatal factors could be identified. There are no obvious congenital genetic/syndromic causes for TG's neurological deficits.

Defendant's conduct of the trial

[27] Despite these issues being agreed upon as being common cause,[6] the defendant, in effect, 'threw the book' at the plaintiff. The defendant sought to introduce a myriad of evidence and literature to show the following: that there was insufficient evidence to time the HIE as being intrapartum; that the fact that the plaintiff was HIV positive was the cause of the CP; that there was a possibility of chorioamnionitis and/or chorionic villi vasculitis, Villitis of Unknown Etiology (VUE) and/or various other infections which may have caused the HIE.

[28] It must be noted that none of these issues were raised by the defendant on the pleadings. Furthermore, the defendant conducted this trial in a most unsatisfactory manner, in effect, a 'trial by ambush'. The defendant's counsel sought to introduce reams of literature whilst the plaintiff's witnesses were testifying. This resulted in the matter having to stand down on numerous occasions for the witnesses to familiarise themselves with the literature. In addition, the cross-examination of the plaintiff's witnesses was at times insulting and aggressive. On several occasions I had to admonish the defendant's counsel in this regard. It is pertinent to refer in this regard to *S v Azov*,[7] where the following was stated in regard to the attitude of the counsel in cross-examination:

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[6] And further confirmed in the defendant's heads of argument where it is stated: '6. The matters dealt with under the heading "The issues which are common cause or agreed in the joint minutes" are not in dispute.'

[7] *S v Azov* 1974 (1) SA 808 (T) at 810F – 811A.

‘I think it must be made clear to him, and perhaps to others, that witnesses who come into court, be they police witnesses or any other kind of witnesses, are entitled to the ordinary courtesy one extends to decent people. Witnesses who give evidence are assisting the court in arriving at the truth and in carrying out the administration of justice. No cross-examiner is entitled to insult a witness or to treat him in the manner in which these witnesses were treated, without there being a very good reason for it. Witnesses must be treated with courtesy and respect. They are doing a public duty in coming to court. That must be borne in mind by both cross-examiners and by presiding officers. It was clearly the duty of the magistrate here to protect these witnesses. I do not wish to be understood to say that a witness may never be attacked, but before you can attack a witness you must at least lay a foundation to the satisfaction of the presiding officer that you have grounds for attacking the witness. Otherwise witnesses must be treated with respect and with the same courtesy that you would extend to a man in civilised society. One is not rude to people when you speak to them during ordinary social intercourse, so why should it be any different in a court of law? Here this cross-examiner really shocked me in regard to the manner in which he treated these respectable men. He starts off by attacking them without any reason. He seems to assume that they are dishonest people and that he is entitled to attack and insult them….’

[29] Unfortunately, the defendant’s counsel was guilty of the same conduct; more particularly when he attacked some of the plaintiff’s experts.

Plaintiff’s evidence

[30] The plaintiff testified that she was 38 years old and had a Grade 11 level of education. She was 25 years old when TG was born. She has two other children aged six years and three years, who are both healthy and do not suffer from any of the health and neurological deficits which TG has.

[31] The plaintiff first attended at the Clinic in about February 2004, when it was found that she was pregnant. She was tested for HIV and was found to be HIV positive. The nursing sisters informed her after the examination that the baby was fine. She attended at the Clinic regularly for the next few months.

[32] On 15 November 2004, the plaintiff experienced some contraction-like pains in her abdomen and went to the Clinic where she was kept overnight. The nursing sister examined her in the morning and thereafter discharged her to go home.

[33] On 22 November 2004, she returned to the Clinic after she discovered a mucous and blood discharge on her panties just before 06h00. She also started having contractions and proceeded to the Clinic. She arrived at the Clinic at approximately 07h00. She was attended to by a nursing sister who checked her cervical dilatation manually and then advised the other nurses present that she was about to give birth. She was then taken into a labour room just behind the reception area.

[34] She was told to lie on a bed and a drip was put into her arm. The nurse left the room at some stage and only returned to check if the drip was working properly, but did not examine the plaintiff. She testified that the nurse did not examine her again from the time when the drip was inserted until she gave birth. The plaintiff was left in the labour room until she started screaming because the pains she was experiencing in her abdomen had become unbearable. During the intervening period, neither she nor the foetus were monitored. She is not sure of the time when she started screaming, but thinks that it was around 12h00.

[35] Three nurses came into the labour room when she started screaming and they then started telling her to push, which she then did. When she was told to push, one of the nurses placed her hand on the plaintiff's abdomen and pressed onto her abdomen just below her breast. The nurse continued to press until TG was delivered.

[36] The plaintiff estimated that the time from when she started screaming until the baby was delivered was approximately 30 minutes. When the baby was delivered, he did not cry, and she heard the nurses saying that one of them should go and fetch the oxygen.

[37] A while later, the plaintiff got off the bed and then saw her baby with an oxygen mask on his face. She was not allowed to touch or handle him. She noticed that the baby was still and was not moving, nor was he making any sounds. One of the nurses told her that they were being transferred to CHBH. The nurses did not inform her why they were taking her and the baby to CHBH, save to say that the baby did not cry.

[38] The ambulance arrived some three hours later at approximately 16h00 and they then drove to CHBH in the ambulance, accompanied by a nurse who held the baby in the ambulance. On arrival, TG was taken away by the nurses who were still administering oxygen to the baby.

[39] The plaintiff was later informed that TG was in ICU and that she could only see him the next day. The plaintiff saw TG in the ICU the next day in an incubator with pipes inserted into his nostrils and running into machines and into a bottle. She was informed that TG had not yet cried and that he was unable to feed normally, and was therefore being fed by means of the pipes which she was seeing. TG was kept in ICU for about a week, and was then taken to a ward where he was kept for a further two weeks.

[40] The plaintiff was discharged from CHBH after two days while TG was in ICU. She visited the baby every day in the ICU and in the ward. TG was kept on an oxygen mask while in the ward. Whilst in the ward, people from the speech therapy department assisted her with training on how to feed TG with a feeding bottle as TG had difficulty sucking.

[41] A week after TG was discharged from the hospital, the plaintiff took him to the antenatal clinic. TG attended there approximately once a week for one month. At three months of age, TG was referred to the CP clinic at CHBH where the plaintiff was informed that TG suffered from CP.

[42] TG is now 13 years old and he cannot walk, talk, or sit up straight without support. At present, he crawls around on his knees and cannot do anything for himself, like feeding or cleaning himself.

[43] The plaintiff gave evidence that with the births of her two other children she experienced the same symptoms before and during labour as she did with TG. In those cases, however, she and her babies were monitored and referred to the hospital for the children to be delivered in the hospital.

[44] The plaintiff stated that she clearly remembered everything about the birth of TG because the treatment which she received during his birth was so different to that which she received with the birth of the other two children, and her recall was also clear because of the trauma surrounding his birth.

[45] In cross-examination, other than repeating the plaintiff's version and then accusing her of being coached, the defendant did not put a version or a defence to the plaintiff. The Court commented that counsel had traversed each and every one of the plaintiff's statements without putting forward a version. The position regarding unchallenged evidence was set out in *President of the Republic of South Africa & others v South African Rugby Football Union & others*[8] to the following effect:

'The institution of cross-examination not only constitutes a right, it also imposes certain obligations. As a general rule it is essential, when it is intended to suggest that a witness is not speaking the truth on a particular point, to direct the witness's attention to the fact by questions put in cross-examination showing that the imputation is intended to be made and to afford the witness an opportunity, while still in the witness-box, of giving any explanation open to the witness and of defending his or her character. If a point in dispute is left unchallenged in cross-examination, the party calling the witness is entitled to assume that the unchallenged witness's testimony is accepted as correct.'

Expert witnesses

Evidence of Dr Frank – obstetrician and gynaecologist called by the plaintiff

[8] *President of the Republic of South Africa & others v South African Rugby Football Union & others* 2000 (1) SA 1 (CC) para 61.

[46] Dr Frank had obtained a history of what had happened during labour and the delivery of the baby from the plaintiff by way of an interview conducted on 19 February 2018. She opined that it could be deduced that the plaintiff was in active labour when she arrived at the Clinic at approximately 07h00 on 22 November 2004. She was at least four centimetres dilated on admission. As a primigravida, the plaintiff would have dilated at one centimetre per hour. She should therefore have taken between six to eight hours to dilate fully.

[47] She referred to what was contained in the birth register. It was noted that there was meconium aspiration, meaning that TG had aspirated meconium at some stage during the delivery. Such aspiration can present as pneumonia-like symptoms. As a child only starts breathing after delivery, TG could not have aspirated the meconium prior to his delivery and must have aspirated the meconium during or after delivery when he took his first few breaths. The maternity register further indicated that Meconium Stained Liquor was noted when the child was delivered, indicating that the child was probably in distress during the labour and/or delivery period.

[48] Meconium aspiration is preventable by either meconium infusion, or the more practical method of suctioning the meconium out of the baby's trachea once the baby is delivered, thereby preventing the meconium from entering the respiratory system. In general, the nursing sisters delivering the baby would suction the baby immediately on noticing the presence of meconium at birth.

[49] TG suffered birth asphyxia, which is a condition where the baby is depressed, has low Apgar scores; the baby would be flat, not crying, have low tone, and be blue instead of pink, and would need assistance with breathing or resuscitation – which occurred in this case with TG.

[50] There are several causes of birth asphyxia. In the present case, there is no record or evidence of intrauterine infection, cord around the neck, an abnormal presentation or shoulder dystocia, which are some of the causes. In Dr Frank's opinion, the birth asphyxia could have been caused by the meconium aspiration. The thick meconium could have occurred because the baby was compromised during the course of labour, and it could have occurred at the time of delivery as well. It would

have been possible to detect that the baby was being compromised before delivery if correct monitoring occurred.

[51] The protocol for the care of a pregnant woman admitted into the labour ward before giving birth is as follows:

(a) Her vital signs i.e. blood pressure, pulse, urine, and temperature would be recorded every two hours.
(b) She will have a vaginal examination hourly from six centimetres dilated.
(c) The foetal heart rate will be listened to a half hour before, during, and after contractions.
(d) When the cervix is fully dilated (ten centimetres), then the foetal heart rate must be monitored at intervals of five minutes.

[52] The purpose of this protocol is to detect any abnormalities in the condition of the mother and the foetus. From such closer monitoring the doctor or midwife would get an indication of the condition of the baby. It will give the practitioner an idea if the baby is coping with the labour, or is in distress during the contractions.

[53] If the baby is in distress in this period before its birth and is imminently deliverable, i.e. the mother is ten centimetres dilated, then the nurses will encourage her to bear down or push the baby out. If she is not imminently deliverable, i.e. she is about seven centimetres dilated, if the foetal heart rate is not reassuring, then she must be transferred to a hospital where a caesarean section can be performed.

[54] Foetal monitoring and foetal heart rate monitoring is critically important as it would indicate the condition of the baby, and if not reassuring, would prompt an intervention to deliver the baby.

[55] Based on an assumption that the plaintiff was admitted to the Clinic at approximately 07h00, she would have had to be examined at least three more times before she was fully dilated. The foetus should then have been monitored every 30 minutes, at least seven times before 12h00, and at least ten more times in the period from 12h00 to when TG was delivered at 13h00. That is 17 examinations in the

period from 08h30 to 13h00. If the plaintiff was allowed to labour by herself for six hours without being attended to, and without the foetus being monitored, then her treatment was substandard.

[56] Dr Frank did not believe that the use of fundal pressure was safe. Fundal pressure means that the uppermost part of the uterus is being squeezed manually in order to get the baby out. The use of fundal pressure often resulted in damage to the mother and child, such as a ruptured liver, fractured ribs, ruptured uterus and foetal injuries and conditions, such as the subaponeurotic haemorrhage, which TG presented with at birth. Fundal pressure could cause a cord compression if the cord was in a position where it could be pushed against the maternal pelvis. The cord compression could have caused the birth asphyxia if the foetus was compressed against the bony pelvis and the cord is trapped between the foetus and the pelvis. This did not appear to be the situation in the present case.

[57] One of the reasons why fundal pressure would be employed, is if the doctors or midwives were worried about the well-being of a baby and could not get the baby out fast enough, or could not get the mother to a theatre for a caesarean section or deliver the baby by vacuum or forceps. In other words, it would be used if there was an emergency situation.

[58] In this case, Dr Frank believes that when the nurses saw the meconium stained liquor, they realised that this baby was in trouble and needed a rapid delivery, and that is why fundal pressure was applied.

[59] As the injury was a partial prolonged HIE brain injury, adequate monitoring of the mother and foetus would have shown that TG was in distress, was having decelerations or prolonged bradycardia (low heart rate). If the nurses had been monitoring the baby appropriately, they would have picked this up timeously; they would have been able to arrange to transfer the mother and baby to a tertiary centre such as CHBH, where the mother and baby could have received a better standard of care during the labour and delivery.

[60] Neither Dr Frank, nor Dr Kohl (the obstetrician and gynaecologist called by the defendant) dealt with the effect of the plaintiff's HIV status and/or the presence of infection on TG's condition in their reports or the joint minutes. Despite this, the defendant's counsel cross-examined Dr Frank extensively in this regard. Dr Frank noted that the plaintiff was HIV positive but was only placed on ARV's seven years after the birth of TG, and deduced from this fact that the mother was healthy, and had a high CD4 count with a low viral load. Both mother and child were also given nevirapine during the labour and after delivery, to prevent mother to child transmission of the HIV virus.

[61] In commenting on the mother's health status at the time of the birth of TG, Dr Frank noted that the mother's haemoglobin level was 10.7. She further noted that, in general, women who are infected with HIV, who are immune compromised generally have a haemoglobin lower than 10.7, the deduction from this being that the plaintiff was not yet immune compromised, although infected at the time.

[62] If one took into consideration the objective facts recorded, such as the fact that the baby was not crying at birth, that the baby required resuscitation and needed oxygen immediately at birth, that the baby was transferred to CHBH and was admitted to the NICU and remained in the hospital for three weeks, the Apgar scores of 6/10 at one minute, 8/10 at five minutes and 10/10 at ten minutes (from the discharge summary), are not credible.

[63] Dr Frank believes the Apgar scores may have been inflated and a baby with birth asphyxia and meconium aspiration would certainly not be able to score 10/10 on an Apgar within ten minutes. Further, that common logic dictates that a healthy baby would not be taken to the ICU.

[64] Under cross-examination, which was in the main aimed at Dr Frank's lack of expertise, the defendant's counsel questioned the expertise of Dr Frank by suggesting that she had ventured an opinion in the area of HIV in which she did not have a full qualification. He further accused her of not having credibility amongst her peers, in that she had not published relevant research in peer reviewed journals. Dr Frank responded that her expertise and training was in the field of obstetrics and

gynaecology, but that it was necessary for her to have knowledge of HIV treatment protocols as treating and delivering HIV positive women was part of her training and her daily tasks as an obstetrician and gynaecologist.

[65] Dr Frank practiced as an obstetrics and gynaecology specialist in the labour ward at CHBH from 2006 to 2012, having qualified as a medical doctor in 1999, and having been an intern from 2000 to 2006 when she became a specialist. In addition, she taught obstetric students at the hospital, performed surgery and supervised Master's in Medicine students in their research for their theses, and was also an examiner at the University of KwaZulu-Natal.

[66] Dr Frank was published in the American Journal of Obstetrics and Gynaecology, which Mr Pauw himself had acknowledged as one of the highest ranking peer review bodies. In addition, she was published in the South African Journal of Obstetrics and Gynaecology and the British and Italian Journals of Obstetrics and Gynaecology.

[67] Dr Frank conceded that her knowledge and experience of CP was limited as it was not her area of specialisation, but she was also not totally uninformed as to the causes of CP or how it related to antenatal and intrapartum causative factors.

[68] Counsel for the defendant further suggested that the finding by Dr Frank that at birth there was thick meconium present was false as it is not mentioned in the available records. Dr Frank replied that the fact that the child suffered meconium aspiration at birth, according to the records, indicates that there was thick meconium. In addition, Dr Frank pointed out that the child was diagnosed with respiratory distress syndrome which could very easily have been caused by Meconium Aspiration Syndrome.

[69] To the suggestion that the child had congenital pneumonia at birth, Dr Frank responded that the note was merely a query and not a diagnosis of pneumonia. Further, that the results of tests done on the baby were put into the hospital file at least three weeks after the tests were done. If the child had congenital pneumonia, it would have been investigated and confirmed. There was no such note in the file.

[70] It was put it to Dr Frank that HIV exposure (i.e. when the mother is HIV positive while the child is in utero) is associated with an adverse outcome or morbidity in new born babies. This he based on a study done in Botswana (the Botswana study).[9]

[71] Dr Frank responded that the sample size in the study for the article was too small to be of much significance, and the results were confounded by a high number of premature births which were included in the sample cohort, and that prematurity was found to be a significant risk factor for CP in Botswana.

[72] The article, in fact, concluded as follows:

'Cerebral Palsy is an important and common contributor to childhood disability in low-resource settings. The relative contributions of major risk factors for cerebral palsy in Botswana differ significantly from those described in high resource settings, with infection, birth complications and maternal HIV being major contributors. Of note, these are all potentially preventable risk factors, suggesting that interventions in birth practices and neonatal care could make a significant impact in decreasing rates of cerebral palsy in Botswana**.** *Further studies are necessary to confirm maternal HIV as a risk factor for cerebral palsy and to characterize the mechanism by which HIV increases the risk of cerebral palsy.*' [Emphasis added]

[73] Dr Frank conceded, in regard to fundal pressure, that the literature was inconclusive in relation to the possible injury to the foetus. However, she stated that, in 2004, fundal pressure was an acceptable practice in hospitals, but not in clinics.

[74] Dr Frank was recalled at a later date and confronted with articles with which the defendant attempted to show that maternal recall of labour and birth was poor.[10]

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[9] Baphaleng Mononkwane *et al* 'Risk Factors for Cerebral Palsy in Children in Botswana' (2017) 77 *Paediatric Neurology Journal* 73.

[10] BP Yawn *et al* 'Maternal Recall of Distant Pregnancy Events' (1998) 51 *Journal of Clinical Epidemiology* 399.

Dr Frank commented that the articles merely showed that some women remember some aspects of birth and labour better than others, but all women remembered some aspects of the labour better, especially if it was associated with a traumatic or negative outcome.

[75] Despite the relentless challenge to her expertise, Dr Frank's evidence remained consistent that, based on the narrative given by the plaintiff, as well as the information obtained from the available medical records, the care given to the plaintiff and the baby was substandard.

Evidence of Dr Kohl – obstetrician and gynaecologist called by the defendant

[76] Dr Kohl's initial evidence was that he was unable to give an opinion due to the lack of obstetric records. His further comments were based purely on hypothetical scenarios put to him by counsel for the defendant.

[77] Dr Kohl stated that it was highly unlikely that plaintiff was left unattended for six hours. In addition, he had no reason to doubt the Apgar scores, despite the evidence that TG was asphyxiated at birth and taken to CHBH as he was considered very ill. He did, however, made the following concessions:

(a) That continuous foetal monitoring was critical to detect foetal distress.
(b) There should have been foetal monitoring at least every five minutes in the second stage of labour.
(c) That even though low Apgar scores were not conclusive of the poor condition of the baby, they are a strong indicator and useful tool to assess the condition of the child at birth.
(d) That if the evidence of the plaintiff is accepted by the Court, the care given to the plaintiff and the foetus was substandard.

[78] Dr Kohl furthermore testified that, with proper monitoring, if there was foetal distress, it could and should have been detected at an early stage, and that intervention would have led to a positive result for the newborn. The process of labour should be monitored through any of the known mechanisms of assessment to

monitor foetal heart rate patterns and the increase and decrease thereof before, during, and after contractions. He did not dispute the evidence of Dr Frank that if foetal distress was suspected, plans could have been made to transfer the plaintiff to CHBH.

Evidence of Professor Nolte – nursing specialist called by the plaintiff

[79] The evidence of Professor Nolte was clear, that based on the history given by the plaintiff, the nursing sisters were grossly negligent in the following respects:

(a) They did not monitor the plaintiff.
(b) They did not monitor the foetus and therefore missed important indicators of foetal distress.
(c) They did not identify the significant risk factors in the maternal HIV when the plaintiff was admitted.
(d) The failed to follow the correct protocols in delivering the baby by using fundal pressure when it is specifically prohibited in the training of nurses and midwives.
(e) They allowed the plaintiff to give birth without any medical assistance, when clearly there was a difficulty in the birth which necessitated a resort to the dangerous procedure of fundal pressure.

Evidence of Professor du Plessis – nursing specialist called by the defendant

[80] Professor du Plessis did not dispute the proper protocol for the management of labour and maternal and foetal monitoring. She commented that HIV holds the potential of opportunistic infection, but could take the matter no further.

Evidence of Professor Johan Smith and Professor Bolton – paediatric and neonatal specialists

[81] Professor Johan Smith, the paediatrics and neonatology expert called by the plaintiff, is the head of Neonatal Services at the Tygerberg Hospital and the head of the neonatal intensive care unit at the University of Stellenbosch University Hospital since 1987.

[82] In the pretrial minute, Professor Smith and Professor Bolton (called by the defendant) could not find common ground in relation to the cause of TG's CP:

(a) Professor Smith stated that the Apgar score of 6/10 at one minute was probably an 'assisted' score after resuscitation. Professor Bolton disagreed, stating that that scenario is only one such possibility. It was equally probable to speculate that the child's clinical condition at birth reflected the presence of severe respiratory distress.

(b) Professor Smith opined that moderate and severe encephalopathy is attributable to asphyxia in 60% of cases, most of which evolve during labour.[11] Professor Bolton partially agreed, but stated that these authors defined NE as being caused by asphyxia according to blood gas and CTG anomalies, which are not available in this case. The authors noted that their patients with asphyxia and NE had low five-minute Apgar scores (<7/10 in 77% and <4/10 in 44% of cases). In this case the five-minute Apgar score was 8/10. Professor Smith doubted the Apgar score at five minutes, having regard to the condition of TG and the fact that he was immediately referred to CHBH.

(c) Professor Bolton stated the following in the joint minute:

'Children born to HIV mothers in low-resource settings in Southern Africa [the Botswana study] have been shown to be at increased risk for developing CP and this risk is not affected through the transmission of the HIV to the foetus, but likely to be due to the effect that the HIV infection has on the placenta.
The mother's HIV status probably had a deleterious effect on placental function and this then played an important role in the development of brain damage and that infection with HIV is associated with chorioamnionitis which in turn is associated with cerebral palsy….'

[11] Jonsson M *et al* 'Neonatal encephalopathy and the association to asphyxia in labor' (2014) 211 *American Journal of Obstetrics and Gynecology* 667.e1-667.e8.

Professor Bolton referred to a study regarding the effect of HIV on placenta from Bangkok, Thailand (the Bangkok study)[12] which showed that:
'Although HIV-infected women were more likely than HIV-uninfected women to have placental inflammatory lesions, but that placental inflammatory lesions were not associated with increased perinatal HIV transmission.'

(d) Professor Smith noted that in the Bangkok study, placentas from the HIV-positive group were characterised by decreased weight and an increased number of marginal infarcts relative to the HIV-negative group.

(e) Professor Bolton stated that (1) recent evidence from Southern Africa (the Botswana study) has shown that postnatal infection including early-onset pneumonia was strongly associated with later development of CP; and (2) that in this case the baby was thought to have evidence of congenital pneumonia; and (3) this early infection may have been associated with chorioamnionitis and probably played an important role in the aetiology of CP.

(f) Professor Smith was of the view that the Botswana study included the whole spectrum of gestations, including premature births of gestations below 32 weeks, which are known to influence incidences of CP. Of the subjects with CP studied (56), only eight had gestational age <32 weeks. The authors acknowledged that they did not have sufficient data in this study to allow them to differentiate between potential mechanisms causing CP related to HIV.

(g) Professor Bolton stated that the major risk factors for CP in Botswana included birth complications (50%), neonatal infection (23%) and maternal HIV. Serious neonatal infection was defined as culture-proven sepsis, severe pneumonia, or meningitis, but the actual percentages were not reported.

(h) Professor Bolton observed that in this case, the severity of the respiratory distress was not commented on in the neonatal discharge summary. Professor Smith noted that the discharge summary indicated a possibility, rather than a probability,

[12] DA Schwartz *et al* 'Placental abnormalities associated with human immunodeficiency virus type 1 infection and perinatal transmission in Bangkok, Thailand' (2000) 182(6) *The Journal of Infectious Disease* 1652-1657.

that there was congenital pneumonia, which is different to an infection acquired after birth, and there certainly was no meningitis or culture-proven septicaemia.

(i) Professor Smith noted that if there was possible chorioamnionitis as referred to by Professor Bolton, chorioamnionitis among neonates with encephalopathy has been associated with a lower risk of brain injury and adverse outcomes, whereas signs of neonatal sepsis carried an elevated risk.[13] He noted that neonatal infection was not defined as pneumonia, but according to the presence of a positive blood culture or combined haematological derangements associated with neonatal fever. A positive blood culture was not reported in the present matter, otherwise the neonatal summary would have recorded it, and the reference to 'congenital pneumonia?' indicates that this diagnosis was uncertain, or not proven.

(j) Professor Bolton disagreed. He stated that the neonatal caregivers noted respiratory distress and ascribed this to pneumonia. He quoted a study which stated that:

'Pneumonia in newborn infants is often difficult to diagnose and often difficult to distinguish from other causes of respiratory distress... Although many investigations including white cell counts, blood cultures, C-reactive protein, etc are performed, they lack the necessary sensitivity and specificity to accurately diagnose pneumonia'.[14]

(k) Professor Smith stated that it is alleged that external abdominal pressure was applied. If this allegation is borne out in Court, the following should be noted:-

i. Fundal pressure is understudied and remains controversial in the management of the second stage of labour;
ii. Application of fundal pressure on a delivering woman was ineffective in shortening the second stage of labour;

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[13] Jenster, M *et al* 'Maternal or neonatal infection: association with neonatal encephalopathy outcomes' (2014) 76 *Pediatric Research* at 93.
[14] Edwards MO *et al* 'Respiratory Distress of the Term Newborn Infant' (2013) 14 *Paediatric Respiratory Reviews* 29-37.

iii. Fundal pressure is associated with shoulder dystocia and foetal acidosis;

iv. There was evidence for a traumatic delivery if one considers the recording of a subaponeurotic haemorrhage (SAH) and one has to consider a contributory role for external abdominal pressure;

v. The mechanism through which external abdominal pressure probably contributed towards the development of HIE was recently described as 'cranial compression ischemic encephalopathy'.[15] They opined that intrapartum events, including asphyxia in term foetuses, account for significant amounts of subsequent neurological handicap, including CP. In their review, they trace the development of the understanding of the forces of labour as a mechanism of foetal head trauma and subsequent foetal neurological injury.

(l) Professor Bolton stated that the lack of contemporaneous records of the labour makes it speculative regarding the allegation that abdominal pressure was applied. According to him, this seems unlikely if, as recorded by Dr Mogashoa (the Paediatric Neurologist called by the defendant), 'the delivery was not difficult'. Thus, Professor Bolton concluded, the mother's various versions should be tested in Court.

(m) Professor Smith summarised the position as follows:

'…considering the available limited factual records, the allegation that there was application of external fundal / abdominal pressure, his own experience with alleged birth asphyxia cases and the scientific literature, he concludes that the foetal brain was probably subjected to partial prolonged hypoxic ischaemia during the period between 07:00 and 13:00. It is likely (probable) that the combined effects of uterine contractions (placental blood flow) during the active phase of labour and external fundal pressure during the second stage of labour, significantly contributed towards the development of cerebral (brain) ischaemia, cranial compression ischaemic encephalopathy and birth trauma (SAH), which ultimately lead to the development of cerebral palsy.'

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[15] BS Schifrin *et al* 'Cranial compression ischemic encephalopathy: Fetal neurological injury related to the mechanical forces of labor and delivery' in L Zhang and LD Longo (eds) *Stress and Developmental Programming of Health and Disease: Beyond Phenomenology* (2014) at 651-688.

(n) Professor Bolton disagreed in this regard. He was of the view that—

'In the absence of appropriate, contemporaneous clinical records of the labour and delivery, it is speculative as to whether there was sub-optimal care offered by the defendants during labour. The obstetric experts, rather than a paediatrician and neonatologist, should be relied upon to comment on care of the mother.'

(o) Professor Smith further noted:

'When an acute sentinel event such as uterine rupture, placenta praevia, abruption placenta or cord prolapse is reasonably excluded, the most common causal factor underlying intrapartum hypoxic ischaemic brain injury (neonatal encephalopathy) is suboptimal obstetric care. In the present case none of the aforementioned sentinel factors were recorded in the available medical notes…'

(p) Professor Smith concluded that, based on a balance of probabilities, foetal distress and a non-reassuring foetal condition probably occurred before birth because the probability that early neonatal encephalopathy occurs with foetal distress is higher than the probability that encephalopathy occurs without foetal distress. Professor Bolton disagreed with this statement, regarding it as speculation.

(q) Professor Smith was of the opinion that: 'Suboptimal obstetric care was probably the avoidable causal factor and therefore had birth been appropriately expedited, the outcome was probably completely avoidable.' Professor Bolton disagreed with this conclusion.

Evidence of Professor Smith (additional to that contained in the joint minutes)

[83] TG suffered from birth asphyxia and HIE Grade 2. This, in layman's terms, is an injury to the child's brain caused by deprivation of blood and oxygen to the brain.

[84] 'Grade 2' refers to the severity of the injury being a moderate injury on a sliding scale from one to three, with one being mild and three being the worst kind of injury. When the clinicians write this diagnosis, it means that they were under the

impression that this HIE is the mechanism of the brain injury and the encephalopathy.

[85] When the congenital pneumonia occurs during the pregnancy near to the time of delivery, one would link it to a maternal infection; the probability is that there is chorioamnionitis in a mother who would present with fever, tender uterus, pus draining from the vagina, foul smell, or a combination of such things. If there was chorioamnionitis there would be a footprint or record of the infection with regard to treatment and antibiotics for the infection. None of these was present with the plaintiff.

[86] In the absence of infection, just pure asphyxia *per se* will result in respiratory distress. It is more likely on the probabilities, and supported by his experience and the literature, that when encephalopathy is diagnosed during the early neonatal period, that there must have been foetal distress during labour and birth.

[87] Having regard to the history that the plaintiff gave regarding the condition of the child at birth i.e. that the child did not cry, the child was lying very still and not moving, he was resuscitated, placed in an incubator and transferred to CHBH, where he was placed in ICU with tubes in his nose for feeding and breathing, and after his discharge was referred to the CP clinic – the probable time at which the hypoxia and encephalopathy occurred is in the intrapartum phase. That is, during labour and birth at which the child sustained the asphyxia and the development of the CP.

[88] The Apgar scores recorded in the available hospital records do not make sense if one has regard to the known objective factors, such as the condition of the child at birth and the need to admit the child to the NICU at CHBH immediately after birth.

[89] It does not make sense that they would resuscitate the baby and then immediately decide to send the baby to CHBH with an Apgar score of 8/10. There was no clear reason to refer TG to CHBH, as 8/10 is a normal score. If the baby arrived at CHBH and scored 10/10 then that would indicate that TG was perfectly

healthy. In his experience he has found that clinics and midwife clinics sometimes overestimate or inflate the Apgar scores.

[90] From the perspective of a neonatologist, the HIV-positive status of the mother did not highlight a higher risk for abnormal neurodevelopmental outcome for the child, the mother having taken nevirapine before the birth. He stated that there are no studies that show conclusively that HIV is noted as an independent cause for CP. HIV did not give you a higher risk profile, so there is no conclusion that can be drawn from studies from Botswana and other places, as obstetric care in South Africa is better than that in Botswana.

[91] Growth restriction is the long standing effect of inadequate placental function. Infection like HIV kills a baby if it is severe enough and is contracted in close proximity to labour and delivery; whereas placental pathology caused by some infection is contracted weeks before delivery in the majority of cases, and would be evident from the mother's symptoms. There is no evidence that this occurred.

[92] Various articles[16] were put to Professor Smith by the defendant's counsel for comment. When presented with the article titled 'The Villitis of unknown etiology: noninfectious chronic villitis in the placenta' (the third Redline article)[17] Professor Smith stated that if Villitis of Unknown Etiology (VUE) was implicated in having caused a placental pathology which resulted in the hypoxia and CP, the VUE would cause preterm labour, still birth and intrauterine growth restriction – none of which are present in this matter. In addition, if the CP was caused by VUE, it would be of a different kind from the spastic quadriplegic and/or dyskinetic CP. Linking VUE and CP causally by way of a hypoxic mechanism involving the placenta would be a quantum leap as there is no clear cut definite brain pathology for such a link. The

[16] RW Redline & MA O'Riordan 'Placental lesions associated with cerebral palsy and neurologic impairment following term birth' (2000) 124 *Archives of Pathology & Laboratory Medicine* 1785-1791; RW Redline 'Severe fetal placental vascular lesions in term infants with neurological impairment' (2005) 192 *American Journal of Obstetrics and Gynaecology* 452-457; and RW Redline 'Villitis of unknown etiology: noninfectious chronic villitis in the placenta' (2007) 38 *Human Pathology* 1439-1446. (The Redline articles).

[17] 'The Villitis of unknown etiology: noninfectious chronic villitis in the placenta' (note 16 above).

proposition is all speculation. There is not a specific categorisation of the type of CP that may be caused by VUE. The article does not include any HIV cases.

[93] Professor Smith discounted the Botswana study as neonatal infections were defined as culture proven sepsis, severe pneumonia or meningitis. But the actual percentages were not reported, and complications occurred in half of those cases. Association between maternal HIV and CP has not been previously described and required further investigation.

[94] In regard to the Bangkok study, Professor Smith commented that the article states that with an HIV positive population, there was more chorioamnionitis. There was no chorioamnionitis present in the plaintiff's case.

[95] If there was a placental abnormality causing hypoxia, the foetus would show that by an abnormal foetal heart rate and heart rate pattern which is then equated to foetal distress and would then be detectable, if proper monitoring occurred. In any event, if the brain injury was sustained in the weeks before delivery there would, on the probabilities, be a non-reassuring foetal heart rate pattern and the probability that the brain would have already started shrinking in the weeks prior to the labour. In other words, the baby would have had a much smaller head circumference out of proportion to what TG had.

[96] There is evidence of meconium aspiration, which negates the diagnosis of congenital pneumonia. The query of congenital pneumonia is just a query and not a diagnosis. It was not confirmed in the discharge summary.

[97] Having considered the multi-pronged, multi-faceted, multi-disciplinary involvement and what respective experts for the plaintiff described, including genetics, neurological outcome, and the radiology picture (of which there is agreement between the plaintiff and defendant) one has to conclude that intrapartum asphyxia is the most likely cause of the child's injury. As this was a partial prolonged HIE brain injury, the failure to monitor the foetus' heart rate amounted to substandard care, as proper monitoring would have picked up the foetal distress in a timely way, allowing the defendant's employees to refer the plaintiff to CHBH timeously.

Evidence of Professor Bolton (additional to that contained in the joint minutes)

[98] Professor Bolton conceded that he is not a neonatologist and that he is careful not to claim to be what he is not. However, within the field of his expertise as a paediatrician, he confirmed that the HIE occurred in the intrapartum phase.

[99] He opined that, in view of the plaintiff's HIV positive status, the condition of the child may have been caused by exposure of the child to HIV – although this did not mean that the child was HIV infected. However, he conceded that given the status of the available research material in respect of the association between HIV exposure and possible adverse birth outcomes, he was unable to say that the Court should consider the HIV as a probable cause of the outcome.

[100] Professor Bolton explained that chorioamnionitis had an effect on the amniotic sac, placenta, and umbilical cord and was the most common cause of prematurity, especially in developing countries. He stated further that it is the response to an infection that causes the damage, and that there is no way of detecting this damage during pregnancy. However, the latest consensus statement from the American College of Obstetricians and Gynaecologists in 2014,[18] says that unless you have examined the placenta histologically, one cannot determine the cause of CP.

[101] The examination would include histological chorioamnionitis, microscopic histological chorioamnionitis, microscopic histology, culture histology and clinical histology. Professor Bolton states that on the medical evidence available, the baby most likely had pneumonia. An X-Ray can confirm pneumonia, or it can look like meconium aspiration. But at this point, it is all speculation.

[18] 'Neonatal Encephalopathy and Neurologic Outcome, Second Edition' (2014) 133 Pediatrics e1482-e1488 (the ACOG statement).

[102] On the Mowbray study,[19] Professor Bolton stated the article 'supports the fact that HIV positive mothers have got more hypoxic ischemia in their entrance than HIV negative mothers…. In the worst case scenario that hypoxic ischemia kills the baby and it is born stillbirth.' With reference to the Botswana study, Professor Bolton stated that a lot more evidence needs to be published but that that they 'found a significant increased incidence of CP rather than neonatal encephalopathy, the end result.' In response to Professor Smith's criticism of the Botswana study, Professor Bolton responded, 'So we have got two articles neither of which were the greatest articles, but showing evidence that, at least in an African context in the developing world, certain causative factors are associated with CP.'

Evidence of Dr Solomons – the paediatric neurologist called by the plaintiff

[103] Dr Solomons testified that based on his examination of the child as well as his consideration of the respective expert reports, by a process of deduction and elimination of causal factors, he had reached the conclusion that TG had suffered HIE in the intrapartum period. He further explained that based on the finding that there was bifrontal narrowing, the probability that the HIE occurred in the intrapartum phase was stronger. In addition, he indicated that the finding of multi-cystic encephalomalacia[20] was also a further indicator that the HIE occurred in the intrapartum phase when looked at in conjunction with the history of the labour as given by the plaintiff. This is also confirmed by the radiologists in their reports.

[104] The cross examination of Professor Solomons did not focus on his findings, but rather sought to establish the possibility of an infectious cause for the HIE. He considered other possible causes, such as infection, by indicating that the fact that the baby was of normal weight is in contradiction to an antepartum event or to a placental problem as that would have resulted in intrauterine growth restriction.

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[19] D Kennedy *et al* 'The effect of maternal HIV status on perinatal outcome at Mowbray Maternity Hospital and referring midwife obstetric units, Cape Town' (2012) 18 *South African Journal of Obstetrics and Gynaecology* 6-10.

[20] The softening or loss of brain tissue after cerebral infarction, cerebral ischemia, infection, craniocerebral trauma, or other injury.

[105] With regard to the causal implication of chorioamnionitis, his evidence was that there would have been specific symptoms for chorioamnionitis in the mother, which include a foul smelling discharge and maternal fever, none of which were reported. If sub-clinical chorioamnionitis (no symptoms evident) was suggested, it could only be determined by a histology. He concluded that there was no evidence of chorioamnionitis, and the suggestion in the circumstances is merely speculative and not probable. All the literature referencing chorioamnionitis or foetal inflammatory response syndrome indicates that the outcome would be a preterm infant.

[106] In relation to pneumonia being a possible cause, his evidence was that you would need a very severe pneumonia, and a child that is shocked enough to require cardio compressions to be administered, in order to lead to postnatal HIE.

[107] With regard to the suggestion of intercranial sepsis, Professor Solomons pointed out that the MRI excludes sepsis as a cause. If the injury had occurred after the child had been delivered, it would have been a significant event and would have been something clearly mentioned in the discharge summary.

[108] Professor Solomons differed from Dr Mogashoa, the paediatric neurologist called by the defendant. He opined that a baby born HIV negative, but suffering from CP, could only be connected to the mother's HIV positive status, with premature birth, low weight with a specific brain injury and MRI picture-periventricular leukomalacia.[21] This did not occur in TG's case.

[109] Professor Solomons testified that the mother's narrative of labour is consistent with the MRI 'holes in the frontal area of the brain'. It was consistent with partial prolonged HIE and occurred for 15-30 minutes. Professor Solomons stated that that, in the absence of supporting evidence, he had to rely completely on the mother's history.

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[21] A type of brain injury that affects premature infants. The condition involves the death of small areas of brain tissue around fluid-filled areas called ventricles. The damage creates 'holes' in the brain.

[110] Professor Solomons maintained that, in the literature, there is no support for a link between HIV and a term infant. On HIV, the following articles were put to Professor Solomons and he maintained that HIV is not associated with this case:

(a) The Botswana study associating HIV exposure with encephalopathy: he indicated that the article is unreliable as the sample size was too small to draw a valid conclusion; the type of CP involved is not indicated; and the study clearly refers to an association and not causation. He refuted the attempt to implicate HIV infection or HIV exposure as a probable cause of the encephalopathy. He stated that if HIV was somehow implicated, it would have manifested in premature labour. Further that the MRI would have shown a specific pattern which does not appear in this case. Professor Solomons maintained that, in the literature, there is no support for a link between HIV and a term infant.

(b) The Bangkok study: he pointed out that the high water mark of the study is an association, not causation, and that it is based on placental histology which is not available in this case. The reference is therefore speculative. Professor Solomons discounted this article as it is not applicable, as TG does not have specified spastic CP, which was the issue considered in the Bangkok study.

(c) A study titled 'Placental pathology in full-term infants with hypoxic-ischemic neonatal encephalopathy and association with magnetic resonance imaging pattern of brain injury':[22] Professor Solomons stated it does not speak to HIV itself but rather analysis of placentas in relation to CP, and at term CP's.

[111] The Redline articles: Professor Solomons discounted as being studies on placentas and chorioamnionitis but not specific to CP.

Evidence of Dr Mogashoa – paediatric neurologist called by the defendant

[112] Dr Mogashoa conceded that the baby suffered a HIE in the intrapartum

[22] JC Harteman *et al* 'Placental pathology in full-term infants with hypoxic-ischemic neonatal encephalopathy and association with magnetic resonance imaging pattern of brain injury' (2013) 163 *The Journal of Pediatrics* 968-995.

phase, although she stated that it is difficult to accurately time such an injury. Dr Mogashoa further conceded that the evidence which she gave with regard to the implication of HIV and chorioamnionitis, based on the literature, cannot be used to prove causation, as it only rises to the level of an association. Dr Mogashoa referred to the ACOG criteria that a depressed baby does not mean it is HIE, there are many causes. Children with low Apgar scores turn out normal; children with high Apgar scores can develop CP.

[113] She stated that maternal HIV infection could still be an associated factor, as just because the baby is HIV negative, does not mean that there are no problems during labour. She referred to the ACOG statement, which provides that there should be a multi-dimensional assessment. The take home message of the Botswana article is that maternal infection plays a role in placental pathology and the outcome of the child and subsequent CP. The Bangkok article stated that with HIV positive mothers, there was more chorioamnionitis. In many cases, sub-clinical chorioamnionitis occurred; no symptoms were present which would alert the nurses to foetal distress.

[114] Dr Mogashoa indicated that, in the absence of records, one cannot come to a definite conclusion. Without knowing the effect of HIV, it is difficult, with certainty, to say what caused the HIE.

[115] Dr Mogashoa stated that there are various forms of placental abnormalities. The fact that the baby was HIV negative does not mean that there was no problem with the pregnancy. One may not understand the pathology and the mechanisms behind it, but the study suggests that the HIV, even though it is not transmitted to the baby, is associated with a lot of problems in the placenta. They found abnormality in the placenta in the form of chorioamnionitis. Chorioamnionitis and villitis of unknown origin (VUE) are examples of these. Chorioamnionitis is more common. In the Bangkok Study it is said that VUE is actually a remote issue and it can therefore be excluded in a scenario where HIV may have affected the placenta.

[116] There are three possible, most likely causes of the respiratory distress. Firstly, MAS; secondly, HIE, because the babies are born with metabolic acidosis and they have difficulty in breathing; and, thirdly, pneumonia.

[117] HIV causes lots of problems with infection ‘but what we do not know at this point in time is how it affects brain injury, we are still in the dark about the mechanisms that it affects and therefore we cannot draw conclusions from it and the studies as they stand at present. The association between maternal HIV and CP requires further investigation. Because there is a higher chance of a HIV infected mother having chorioamnionitis, chorioamnionitis as a cause for the HIE, must be one of the considerations in this case.’

[118] The plaintiff’s counsel suggested to Dr Mogashoa that the Bangkok study suggests that the chorioamnionitis has the opposite effect; it says it has a protective effect. At the end of the study, it says chorioamnionitis was associated with lower risk of moderate to severe brain injury. The study also stated that with ‘neonatal encephalopathy, chorioamnionitis was associated with no risk of brain injury’. Therefore, plaintiff’s counsel contended that one cannot draw any firm conclusions from the study that the Court can rely on. One study says chorioamnionitis causes brain injury, the other says there is lesser risk for brain injury where there is chorioamnionitis. In addition, the study indicated that they are unsure whether the condition resulted from HIV itself, or as a result of the anti-retroviral treatment that HIV positive mothers were given.

[119] Dr Mogashoa responded that she ‘was quite satisfied to conclude that HIV is associated with increased intrapartum morbidity, increased mortality, increased problems in the infants who are HIV exposed even though they are uninfected because ‘we have to use what we have in front of us, some of it may be conflicting but we know from the countries with increased prevalence of HIV, that we have got problems.’

Evidence of Dr Gericke and Professor Christianson

[120] The two geneticists excluded infections or genetics as probable causes for the HIE.

Literature

[121] Professor Solomons, Professor Bolton and Dr Mogashoa referred to the article 'Neonatal Encephalopathy and Neurological Outcome, Second Edition: Report of the American College of Obstetricians and Gynaecologists' Taskforce on Neonatal Encephalopathy' from the American Academy of Pediatrics (the ACOG statement), which states:[23]

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

[122] Of importance are the following points made in the article:

'There are several well-defined patterns of brain injury and their evolution on MRI that are typical of hypoxic–ischemic cerebral injury in the newborn, including deep nuclear gray matter or watershed cortical injury. If a different pattern of brain injury or evolution of injury exists on MRI, then alternative diagnoses should be actively pursued (eg, metabolic and genetic investigations).'

[123] It appears from the ACOG statement that the definition of neonatal encephalopathy is—

'…a clinically defined syndrome of disturbed neurologic function in the earliest days of life in an infant born at or beyond 35 weeks of gestation, manifested by a subnormal level of consciousness or seizures, and often accompanied by difficulty with initiating and maintaining respiration and depression of tone and reflexes.'[24]

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[23] The ACOG statement (note 18 above).
[24] Ibid at e1484.

[124] One has to look at the clinical status of the baby during labour, during and at birth in the neonatal period, and the subsequent outcome to determine if the baby fits the criteria for intrapartum hypoxia. There are certain requirements before one can diagnose neonatal encephalopathy and the cause thereof. ACOG looks at, inter alia:

(a) the condition of the baby at birth and refers to the Apgar scores;
(b) the condition of the baby in the neonatal period that speaks to neonatal encephalopathy and the staging;
(c) whether there was a sentinel event.

[125] Dr Mogashoa stated that in the 2014 ACOG guidelines, the ACOG task force on neonatal encephalopathy and CP reflects that multiple causes can lead to brain injury in term infants, not just oxygen deprivation around the time of birth. The task force states that a comprehensive, multidimensional assessment should be performed, as described above. The task force considers that multiple factors act together with resultant encephalopathy and resultant injury to the brain.

[126] ACOG requires that the medical practitioners look for risk factors during the labour, called proximal risk factors, and then look at how the labour was managed. This helps one to conclude when the insult occurred. According to the ACOG statement, an Apgar score less than five at five and at ten minutes is supportive of intrapartum hypoxia. The Apgar score remain helpful to determine what the condition of the child was at birth, the need for resuscitation and the response to resuscitation and as one goes on with the resuscitation, the lower the Apgar remains, the more likely the chance of morbidity.

Fundal pressure

[127] The plaintiff contended that the literature in respect of the risks associated with the application of fundal pressure support the version or proposition of the plaintiff that the most probable cause of the injury is a mechanical event such as cord compression or meconium aspiration. I am unable to decide, on the probabilities, whether the fundal pressure contributed to or caused the injury. There was insufficient evidence in this regard. I therefore will not take this issue into

account in analysing the evidence and coming to a conclusion.

The effects of the plaintiff's HIV positive status on TG's condition

[128] The defendant referred to several articles, some of which have been dealt with above, in support of its proposition that there was an association between HIV exposure, intrapartum asphyxia and the development of CP in children born to HIV infected mothers.

[129] The plaintiff argued that the articles referred to by the defendant all clearly indicate that at this point in time much more research has to be done before any conclusions can be drawn with regard to a causative connection between HIV exposure in neonates, and the development of neonatal encephalopathy resulting in CP.

[130] The other articles referred to by the defendant:

(a) 'The effect of maternal HIV infection on maternal conditions and perinatal deaths in southwest Tshwane':[25] The plaintiff submitted that what the article demonstrated is the occurrence of a higher rate of spontaneous preterm births, infection and intrapartum asphyxia in the HIV mothers coupled with a higher rate of antepartum deaths, spontaneous abortion or stillbirths. Further, it referred to an increase of intrapartum asphyxia in HIV infected babies and not to HIV exposed but uninfected babies such as in the present case. The article concluded that:

'The significant increase of intrapartum asphyxia in HIV infected babies was unexpected and unexplained…. A possible explanation is that these fetuses had severe congenital infections that were mistaken for intrapartum asphyxia. Alternatively, previous intra-amniotic infections might make the fetus more susceptible to hypoxia during labour due to the fetal immune response syndrome. The numbers of fetuses involved are small and this observation will need to be

[25] RC Pattinson *et al* 'The effect of maternal HIV infection on maternal conditions and perinatal deaths in southwest Tshwane' (2010) 2 *Facts, Views & Visions in ObGyn* 227-231.

confirmed by other studies.'

(b) The Mowbray study: the defendant sought to show, by way of this study, that there was an association between HIV exposure and morbidities such as neonatal encephalopathy. Whilst the study purported to confirm such an association, the results of the study reports: 'In 2008, there were 54 babies diagnosed with neonatal encephalopathy (predominantly hypoxic ischaemic encephalopathy), 16 in HIV positive. 31 in HIV negative and 7 in untested women.' It is argued by the plaintiff that the quoted statistic runs counter to the case which the defendant wishes to make for the proposition that HIV exposed children are more likely to suffer a neonatal encephalopathy.

(c) 'Meconium aspiration syndrome in infants of HIV-positive women: a case-control study':[26] The purpose of referring to this study was to show that HIV positive women had significantly more meconium-stained amniotic fluid than HIV negative women and that infants born to HIV positive women showed higher rates of Meconium Aspiration Syndrome.

While the study found that there was more meconium-stained amniotic fluid and higher MAS in HIV positive as opposed to HIV negative mothers and infants, the authors stated that—

'We were not able to determine whether it was the HIV infection itself or the fetal exposure to ART that led to meconium passage, possibly through fetal distress.
What is however significant is the repetition of the fact that fetal distress can cause increased production of meconium by the fetus. Therefore, it is important to prevent fetal distress.'

(d) 'Placental pathology in HIV infection at term: a comparison with HIV-uninfected women':[27] the plaintiff contended that this article debunks the suggestion

[26] SK Gupta *et al* 'Meconium aspiration syndrome in infants of HIV-positive women: a case-control study' (2016) 44 *Journal of Perinatal Medicine* 469-475.
[27] E Kalk *et al* 'Placental pathology in HIV infection at term: a comparison with HIV-uninfected women' (2017) 22 *Tropical Medicine & International Health* 604-613.

by the defendant that a positive HIV status will result in more placental lesions than for HIV negative women. The study is significant as it was conducted at hospitals in Cape Town and makes the following conclusions:

'Our findings are notable for the limited differences observed at the placental level between the HIV- infected and uninfected women without adverse birth outcomes.'[28]

The legal framework

[131] As stated above, the plaintiff is the only factual witness who gave evidence. Sister Ndlela, had no recollection of the plaintiff's case and could only confirm what appeared on the birth register.

[132] In a trial action—

'…it is fundamental that the opinion of an expert must be based on facts that are established by the evidence and the court assesses the opinions of experts on the basis of "whether and to what extent their opinions advanced are founded on logical reasoning". It is for the court and not the witness to determine whether the judicial standard of proof has been met.'[29]

[133] Adapting the approach taken by the Supreme Court of Appeal (SCA) in *MV Pasquale*:

'[T]he court must first consider whether the underlying facts relied on by the witness have been established on a prima facie basis. If not then the expert's opinion is worthless because it is purely hypothetical, based on facts that cannot be demonstrated even on a prima facie basis. It can be disregarded. If the relevant facts are established on a prima facie basis then the court must consider whether the expert's view is one that can reasonably be held on the basis of those facts. In other

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[28] Ibid 611.

[29] *MV Pasquale della Gatta; MV Filippo Lembo; Imperial Marine Co v Deiulemar Compagnia di Navigazione Spa ZASCA* 2012 (1) SA 58 (SCA) para 25. See also *Michael & another v Linksfield Park Clinic (Pty) Ltd & another* 2001 (3) SA 1188 (SCA) paras 34-40.

words, it examines the reasoning of the expert and determines whether it is logical in the light of those facts and any others that are undisputed or cannot be disputed. If it concludes that the opinion is one that can reasonably be held on the basis of the facts and the chain of reasoning of the expert the threshold will be satisfied. This is so even though that is not the only opinion that can reasonably be expressed on the basis of those facts. However, if the opinion is far-fetched and based on unproven hypotheses then the onus is not discharged.'[30]

[134] In *PriceWaterhouse Coopers Inc v National Potato Cooperative Limited*,[31] the court said:

'The basic principle is that, while a party may in general call its witnesses in any order it likes, it is the usual practice for expert witnesses to be called after witnesses of fact, where they are to be called upon to express opinions on the facts dealt with by such witnesses.'

[135] Similarly, Wessels JA, in dealing with the nature of an expert's opinion, in *Coopers (South Africa) (Pty) Ltd v Deutsche Gesellschaft für Schädlingsbekämpfung MBH*,[32] stated—

'…an expert's opinion represents his reasoned conclusion based on certain facts on data, which are either common cause, or established by his own evidence or that of some other competent witness. Except possibly where it is not controverted, an expert's bald statement of his opinion is not of any real assistance. Proper evaluation of the opinion can only be undertaken if the process of reasoning which led to the conclusion, including the premises from which the reasoning proceeds, are disclosed by the expert….'

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[30] Ibid para 26.
[31] *PriceWaterhouse Coopers Inc & others v National Potato Cooperative Ltd & another* [2015] ZASCA 2; [2015] 2 All SA 403 (SCA) para 80.
[32] *Coopers (South Africa) (Pty) Ltd v Deutsche Gesellschaft für Schädlingsbekämpfung MBH* 1976 (3) SA 352 (A) at 371F-G.

[136] An opinion of an expert must therefore be based on facts which have been proven before the court. An opinion based on facts not in evidence has no value for the court.[33] A court has to ascertain whether the opinions expressed by the experts are based upon facts proved to it by way of admissible evidence. It is with this principle in mind that the facts of the matter, as well as an analysis of the experts' evidence, must be considered.

[137] As set out above, other than Sister Ndlela, the defendant called no other factual evidence to dispute the plaintiff's version. Such version was, in effect, not challenged in cross-examination.

[138] In *Ratcliffe v Plymouth and Torbay Health Authority*,[34] Lord Justice Brooke made the point that:

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

[139] It suffices for the plaintiff to convince the court that the inference that they advocate is the most readily apparent and acceptable inference from a number of possible inferences.[35] In *Caswell & Powell Duffryn Associated Collieries*,[36] Lord Wright remarked—

'Inference must be carefully distinguished from conjecture or speculation. There can be no inference unless there are objective facts from which to infer the other facts

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[33] *PriceWaterhouse* (note 31 above) para 99.
[34] *Ratcliffe v Plymouth & Torbay Health Authority & Anor* [1998] EWCA Civ 2000 (11 February 1998)
[35] *AA Onderlinge Assuransie-Assosiasie Bpk v De Beer* 1982 (2) SA 603 (A); see also *Cooper & Another NNO v Merchant Trade Finance Ltd* 2000 (3) SA 1009 (SCA).
[36] *Caswell v Powell Duffryn Associated Collieries Ltd* [1940] AC 152 at 169–170.

from which it is sought to establish. In some cases, the other facts can be inferred with as much practical certainty as if they had been actually observed. In other cases, the inference does not go beyond reasonable probability. But if there are no positive proved facts from which the inference can be made, the method of inference fails and what is left is mere speculation or conjecture.'

[140] When a plaintiff is not in position to produce evidence on a particular issue, less evidence will suffice to establish a prima facie case where the matter is peculiarly within the knowledge of the defendant. A shifting burden will be placed on the defendant to show that steps were taken to comply with the standards to be expected. The overall onus nevertheless remains with the plaintiff.[37] In *McIntosh v Premier, Kwazulu-Natal and Another*,[38] the SCA held that:

'The second inquiry is whether there was fault, in this case negligence. As is apparent from the much-quoted dictum of Holmes JA in *Kruger v Coetzee* 1966 (2) SA 428 (A) at 430E - F, the issue of negligence itself involves a twofold inquiry. The first is: was the harm reasonably foreseeable? The second is: would the *diligens paterfamilias* take reasonable steps to guard against such occurrence and did the defendant fail to take those steps? The answer to the second inquiry is frequently expressed in terms of a duty. The foreseeability requirement is more often than not assumed and the inquiry is said to be simply whether the defendant had a duty to take one or other step, such as drive in a particular way or perform some or other positive act, and, if so, whether the failure on the part of the defendant to do so amounted to a breach of that duty. But the word "duty", and sometimes even the expression "legal duty", in this context, must not be confused with the concept of "legal duty" in the context of wrongfulness which, as has been indicated, is distinct from the issue of negligence….

…

The crucial question, therefore, is the reasonableness or otherwise of the respondents' conduct. This is the second leg of the negligence inquiry. Generally speaking, the answer to the inquiry depends on a consideration of all the relevant

[37] *Monteoli v Woolworths (Pty) Ltd* 2000 (4) SA 735 (W) para 27.
[38] *McIntosh v Premier, Kwazulu-Natal and Another* 2008 (6) SA 1 (SCA) paras 12 & 14.

circumstances and involves a value judgment which is to be made by balancing various competing considerations, including such factors as the degree or extent of the risk created by the actor's conduct, the gravity of the possible consequences and the burden of eliminating the risk of harm….'

[141] In *Goliath v MEC for Health Eastern Cape*,[39] Ponnan JA stated:

'When an inference of negligence would be justified and to what extent expert evidence would be necessary would no doubt depend on the facts of the particular case. Questions of absolution from the instance at the close of the plaintiff's case aside, a court is not called upon to decide the issue of negligence until all of the evidence is concluded (*Arthur v Bezuidenhout and Mieny* at 573H). Thus any such explanation as may be advanced by a defendant forms part of the evidential material to be considered in deciding whether a plaintiff has proved the allegation that the damage was caused by the negligence of the defendant or its servants (*Osborne Panama SA v Shell & BP* at 897G – H). Here, although the procedure performed on Ms Goliath was under the control of the MEC's employees, and what they did or did not do was exclusively within their direct knowledge, none of those employees were called to testify.'

[142] Ponnan JA referred to and adopted Lord Justice Brooke's comments in *Ratcliffe v Plymouth and Torbay Health Authority.*[40]

[143] In *Meyers v MEC, Department of Health, EC,*[41] Ponnan JA, in dealing with the onus of proof found that, once the plaintiff had given an acceptable explanation for her claim, it—

'was sufficient as to place an evidentiary burden upon [the doctor] to shed some light upon the circumstances attending [the plaintiff's] injury. Failure to do so meant that, on the evidence as it then stood, he ran the risk of a finding of negligence against

[39] *Goliath v Member of the Executive Council for Health, Eastern Cape* [2014] ZASCA 182; 2015 (2) SA 97 (SCA) para 17.
[40] *Ratcliffe* (note 34 above).
[41] *Meyers v MEC, Department of Health, EC* [2020] ZASCA 3; 2020 (3) SA 337 (SCA) para 71.

him. For, whilst … the plaintiff, bore the overall onus in the case, [the doctor] nonetheless had a duty to adduce evidence to combat the prima facie case made by [the plaintiff]. It remained for him to advance an explanatory (though not necessarily exculpatory) account that the injury must have been due to some unpreventable cause, even if the exact cause be unknown.'

[144] The defendant conceded that the plaintiff's evidence was uncontroverted. It was however submitted that, in spite of this, her evidence should not be accepted. Reference was made to *Siffman v Kriel*.[42] The defendant contended that the version put up by the plaintiff was so improbable that it should be rejected. It reiterated the submission that the plaintiff was a rehearsed witness, without offering any reason therefor.

[145] The submission was that one cannot infer that once something has gone wrong or not in accordance with what was expected, there must have been negligence on the part of the defendant. *Buthelezi v Ndaba*[43] was referred to in this regard. It was submitted that the appropriate approach to the evidence would be to ask, with reference to all the evidence that was admitted, whether a reasonable person in the position of the defendant would have foreseen injury to the plaintiff and have taken steps to guard against it. Reference was made to the *locus classicus* in this regard *Kruger v Coetzee*,[44] as refined in *Mukheiber v Raath & Others*.[45] If this test is applied to the evidence at hand and, in particular, to the fact that there is no complete record of what had transpired during birth and the neonatal period, the answer is that one cannot know what happened. In the absence of such primary knowledge, the defendant contended that it is difficult (if not impossible) to draw any inference of negligence on the part of the defendant.

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[42] *Siffman v Kriel* 1909 TS 538.
[43] *Buthelezi v Ndaba* [2013] ZASCA 72; 2013 (5) SA 437 (SCA), where it was held that negligence was not to be inferred from a concession by the surgeon that something must have gone wrong during the operation. It was dependent on evaluation of the reasoning underlying conflicting expert opinions.
[44] *Kruger v Coetzee* 1966 (2) SA 428 (A) at 430E-F.
[45] *Mukheiber v Raath and Others* [1999] ZASCA 39; [1999] 3 All SA 490 (A).

[146] This approach would depend upon the Court rejecting the plaintiff's evidence as highly improbable. Unfortunately, our court records are filled with cases in which the negligence of the defendant's employees in failing to monitor patients properly, recording issues inaccurately (if at all) and generally displaying conduct that does not befit medical practitioners, is proven. On Sister Ndlela's own version, expectant mothers can be left unattended and unmonitored for two hours on occasion, if the nurses are busy with other patients. There appears to be no reason to reject the plaintiff's version of events, which is not improbable.

Analysis of possible results of the plaintiff's HIV positive status

[147] The plaintiff contended that the defendant's case for implicating HIV as a distal factor in causing the encephalopathy suffered by the child, is that the HIV positive status of the mother may have resulted in the placenta having been affected to the extent that it may have resulted in reduced blood flow to the foetus, and therefore less oxygenation and perfusion. This reduced perfusion then led to the brain of the foetus being affected and resulting in an encephalopathy.

[148] The plaintiff submitted that the literature referred to by both parties indicated that the theory of causation of injury suggested by the defendant is unlikely, and that the more probable cause is a mechanical injury or event which resulted in a deprivation of blood to the foetal brain intrapartum which in turn led to hypoxia, foetal distress, and a resulting encephalopathy.

[149] The plaintiff contended that the injury shown on the MRI, and on which the radiologists agree, is a partial prolonged HIE. This is an important criterion for establishing the timing of the injury to the intrapartum or peripartum phase of labour and the nature of the injury as a partial prolonged HIE, and not one caused by a metabolic or infectious agent (as suggested by the defendant).

[150] The literature referred to by the defendant eliminates the likelihood of VUE having been present in the plaintiff; alternatively, there is insufficient evidence to be drawn from the literature for either VUE or chorioamnionitis to be the cause of the HIE.

[151] In relation to the Bangkok study, the plaintiff pointed to the finding that:

'Our findings that villitis occurred less often in HIV infected women may be related to HIV associated immunosuppression and suggests a diminished capacity to develop an inflammatory response.'[46]

[152] In relation to the Botswana study, the plaintiff submitted that the defendant sought to show, by way of this article, that there was an association between HIV exposure in neonates from HIV positive mothers and the development of neonatal encephalopathy and the sequela of CP. The plaintiff submitted that the study is misplaced in the present case, as the purpose of the study is aimed at low resource settings and obtaining a better understanding of the unique risk factors affecting children with CP in such low resource settings, and how to optimise resource allocation and preventative strategies.

[153] That objective is stated very clearly in the abstract on the first page of the article. The conclusion of the study is stated as follows:

'Modifiable risk factors such as maternal HIV infection should be targeted as a potential strategy to reduce the incidence of cerebral palsy in Botswana. Further studies are necessary to determine the optimal preventative and treatment strategies in this population.'[47]

[154] The conclusion of the Botswana study states—

'Cerebral Palsy is an important and common contributor to childhood disability in low-resource settings. The relative contributions of major risk factors for cerebral palsy in Botswana differ significantly from those described in high-resource settings, with infection, birth complications, and maternal HIV being major contributors. Of note, these are all potentially preventable risk factors, suggesting that interventions

[46] The Bangkok study (note 12 above) at 1655.
[47] The Botswana study (note 9 above) at 73.

in birth practices and neonatal care could make a significant impact in decreasing rates of cerebral palsy in Botswana.'[48]

[155] The plaintiff contended that the study cannot be applied to a South African setting, as there are major differences between the resources available in South Africa as opposed to Botswana. It was further argued that the conclusion sought by the defendant that HIV exposure is associated with neonatal encephalopathy is not one of the conclusions reached in the study. The study also makes the point that further studies are necessary to confirm maternal HIV as a risk factor for CP and to characterise the mechanism by which HIV increases the risk of CP.

[156] It was argued by the plaintiff that the defendant raised the issue of HIV as a possible distal factor merely to muddy the waters as it has not placed any cogent evidence before the Court to show that HIV exposure may have caused the neonatal encephalopathy and CP the child suffered.

[157] The defendant's witnesses, Professor Bolton and Dr Mogashoa, both conceded that HIV exposure as a contributing factor required more research.

Conclusion

[158] It was submitted that by the plaintiff that it is more probable that the negligence of the midwives is the proximal cause of the injury which the child suffered in that they:

(a) Failed to monitor the plaintiff properly during her labour;
(b) Failed to monitor the unborn child regularly and appropriately to detect any foetal distress and therefore did not note that the child was in distress timeously;
(c) Failed to take appropriate action or interventions to stop the child from suffering distress;
(d) Applied a risky and dangerous procedure to deliver the child when they discovered the distress thereby injuring the child further.

[48] Ibid at 76.

[159] Despite vigorous and extended cross-examination, the defendant did not succeed in establishing that the plaintiff was dishonest or coached in presenting her testimony. The plaintiff claimed that neither she nor TG were examined or monitored as required by the maternity guidelines.

[160] The radiological evidence is not disputed. The injury was a partial prolonged ischaemic event. Properly monitored, the foetal distress during this period should have been picked up and the necessary interventions should have occurred.

[161] As stated earlier in this judgment, it is of significance that very fundamental issues were agreed to by the parties before the commencement of the trial. These included:

(a) It is recorded in the birth register that the baby suffered from birth asphyxia as a complication at birth.
(b) TG required resuscitation and oxygen at birth and was transferred to CHBH on the same day after a delay of approximately three hours.
(c) The reason for the transfer to CHBH was recorded as low Apgar scores and birth asphyxia.
(d) The MRI brain scan, dated 30 June 2017, demonstrates features consistent with chronic sequela of a partial prolonged HIE. There are no stigmata of intracranial syndromic disorder and there are no features to suggest complicated intracranial sepsis (infection). The implication of this is that the child does not suffer from a brain injury which was caused by HIV infection or AIDS, or inflammatory infections such as meningitis or syphilis.
(e) There is sufficient evidence for timing of the partial prolonged HIE to the intrapartum period.
(f) There is no recorded evidence for the presence of the histological markers of Foetal Inflammatory Response Syndrome (FIRS) i.e. inflammation of the umbilical cord, acute chorioamnionitis or chorionic villi vasculitis – all indicators of a pathological placental condition.

[162] Despite these agreed issues, the defendant sought to introduce evidence in contradiction to the agreement reached in relation to the timing of the partial prolonged HIE to the intrapartum period; the presence of acute chorioamnionitis or chorionic villi vasculitis; the fact that the child does not suffer from a brain injury which was caused by HIV infection or AIDS, or inflammatory infections.

[163] The evidence introduced by the defendant is inconclusive on all those issues and in most cases, speculative. There is simply no conclusive evidence that the plaintiff's positive HIV status had the consequences suggested by the defendant. In the absence of contradictory evidence, the plaintiff's version (supported by the expert evidence on the consequences of substandard care) that she was not monitored and that such substandard care resulted in TG suffering birth asphyxia and the resultant neonatal encephalopathy and CP must be accepted.

Therefore, the following order is made:

1. It is declared that the defendant is liable for 100% of the plaintiff's damages, as proved or agreed, arising from the birth of TG born on 22 November 2004.
2. The defendant shall pay the plaintiff's costs.

S E WEINER
JUDGE OF THE HIGH COURT
GAUTENG LOCAL DIVISION, JOHANNESBURG

This judgment was handed down electronically by circulation to the parties' and/or parties' representatives by email and by being uploaded to CaseLines. The date and time for hand-down is deemed to be 10h00 on 19 March 2021.

Dates of hearing: 24 – 26 April 2018
13 – 17 August 2018
20 – 24 August 2018
28 January – 1 February 2019

	2 December 2020
Defendant's supplementary heads of argument:	22 December 2020
Plaintiff's supplementary heads of argument:	16 January 2021
Date of judgment:	19 March 2021

Appearances:

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Instructing Attorneys:	State Attorney