IN THE HIGH COURT OF SOUTH AFRICA

(GAUTENG DIVISION, PRETORIA)

Case number: 31261/2015

Heard: 15 February - 18 February 2021

and 25 February 2021

Date of judgment: 8 March 2021

DELETE WHICHEVER IS NOT APPLICABLE
(1) REPORTABLE: YES/~~NO~~
(2) OF INTEREST TO OTHERS JUDGES: YES/~~NO~~
(3) REVISED

8/3/21 .....
DATE SIGNATURE

In the matter between:

LM on behalf of DM **Plaintiff**

and

THE MEMBER OF THE EXECUTIVE COUNCIL FOR HEALTH OF THE LIMPOPO PROVINCIAL GOVERNMENT **Defendant**

JUDGMENT

SWANEPOEL AJ:

INTRODUCTION

[1] This is a claim for delictual damages resulting from a birth injury that the plaintiff's minor child ("D") sustained during labour. Plaintiff arrived at the Dilokong Hospital in the early hours of the morning of 17 July 2010 suffering from lower abdominal pain. She was, she believed, in the early stages of labour. The plaintiff was assessed as being nine months pregnant by date, and 38 weeks by palpitation. By 14h00 the plaintiff had entered the latent first stage of labour. Her vital signs were normal, as were those of the foetus. By 18h00 the plaintiff was 4cm dilated, and she entered into the active first stage of labour. The foetus' baseline heart rate was normal with good variables and no noted decelerations.

[2] Labour progressed normally until 22h00. The plaintiff was attended to 2-hourly, and no concerns were noted. By 00h00 some concerns were noted. Although the partogram is contradictory in that multiple contradictory entries were made on it, it appears that the labour had slowed somewhat. By 01h20 there was no doubt that the progress of labour had slowed and had crossed the action line. The plaintiff had not yet fully dilated and the nursing staff felt it necessary to notify the doctor on call of the plaintiff's condition. It appears likely that the nursing staff were concerned about the plaintiff's condition because they called the doctor at 01h30 and again at 02h00. He promised he would come to see the plaintiff but there is no evidence that he did so.

[3] By 01h50 the membranes had ruptured and meconium-stained liquor (grade 2) was noted. At 02h00 it was recorded that the plaintiff was fully dilated, and she entered the second stage of labour. The last clinical entry was made at 02h00 and it records that the doctor was notified about the plaintiff's condition. The foetus' heart rate was 136 – 138 bpm and regular. The plan was to monitor the foetal and maternal condition. Thereafter there is no further entry in the clinical notes, and no indication that the plaintiff was monitored at all. Plaintiff started bearing down at either 02h15 or 03h15. The exact time is uncertain as the time on the summary of labour form was changed at some point. D was born at 03h35. Her 1 minute Apgar score was 5/10. Her heart rate was satisfactory and scored 2, while she scored 0 for respiration and muscle tone, 1 for response to stimulation, and 2 for colour. This assessment is in itself contradictory because it is unlikely that her colour would have been optimal if she was having trouble breathing.

[4] A second Apgar assessment at 5 minutes of life scored 7/10. D's breathing had improved somewhat, and she had a better muscle tone. The neonate assessment form shows that D had a slow respiration rate, a weak moro-reflex, and an absent grasp reflex and "cry". She was resuscitated and supplemental oxygen was administered via a headbox. D's blood glucose was high at 11.2 mmol/L.

[5] Later observations noted that she had suffered seizures. Her "cry" was still not audible, and a doctor noted hypertonia, rigidity and no reflexes-Moro. D was 'floppy". The evidence was clear: D had suffered birth asphyxia which

had caused a moderate hypoxic-ischaemic ("HI") injury which resulted in severe asymmetrical mixed type cerebral palsy, predominantly dystonic.

[6] Plaintiff's alleges that defendant was negligent in one or more of the following aspects in that he:

[6.1] Failed to employ the services of a suitably qualified medical practitioner who could examine the plaintiff and manage her labour;

[6.2] Failed to ensure that a medical practitioner was in attendance at all material times;

[6.3] Failed to employ suitably qualified nursing staff;

[6.4] Failed to ensure that the hospital was adequately equipped;

[6.5] Failed to take the required steps to ensure proper, timeous and professional assessment, monitoring and management of patients in labour;

[6.6] Failed to implement steps to prevent the occurrence of a complication;

[6.7] Failed to avoid the complication when he could have done so by exercising reasonable care and diligence.

[7] Plaintiff alleges that the defendant's employees were negligent in one or more of the following respects, in that they:

[7.1] Failed to properly and sufficiently assess and examine plaintiff upon her admission;

[7.2] Failed to monitor plaintiff's labour and foetal well-being appropriately and with sufficient regularity or at all;

[7.3] Failed to note sufficiently, timeously or at all that plaintiff's labour was not progressing appropriately;

[7.4] Failed to request assessment or examination by a medical practitioner upon admission;

[7.5] Failed to perform accurate and proper cardiotocographic ("CTG") tracings of the foetal heart rate and maternal contractions, and/or failed to recognize that the foetal heart pattern on the CTG was unsatisfactory;

[7.6] Failed to monitor the foetal heart rate appropriately, timeously or with sufficient frequency and/or at all, and/or failed to detect that D was in foetal distress;

[7.7] Failed to note and/or appreciate the significance and/or timeous progress of labour;

[7.8] Failed to monitor plaintiff's labour, either appropriately, timeously with sufficient frequency and/or at all;

[7.9] Failed to perform a proper and accurate partogram;

[7.10] Failed to perform, or request to be performed, a caesarian section in circumstances where it would have been appropriate to do so;

[7.11] Failed to provide the requisite reasonable medical, nursing and midwifery services with such professional skill and diligence as could reasonably be expected of medical practitioners, nurses and midwives;

[7.12] Failed to obtain a comprehensive obstetric history from the plaintiff;

[7.13] Failed to render reasonable medical, nursing and midwifery services with professional skill and diligence;

[7.14] Failed to prevent D from suffering a hypoxic ischaemic incident, which resulted in severe brain damage.

[8] It is not in dispute that defendant had a duty of care to ensure that plaintiff received proper medical care and that defendant is vicariously liable for the acts or omissions of the hospital staff. It is also not in dispute that plaintiff received sub-standard care. There is no evidence that she was monitored at all from 02h00 onwards, at a time when the protocols require constant monitoring of the mother and foetus. Defendant accepts that the nursing staff were negligent.

[9] The sole question for determination is whether the negligent omission resulted in the hypoxic ischaemic injury and whether, with proper care, the injury could have been prevented.

[10] At the outset the parties moved for an order that the merits of the matter be separated from the quantum in terms of rule 33 (4) of the Uniform Rules of Court. I granted the order. Therefore, it is only the issue of liability that has to be determined at this stage.

THE EVIDENCE

[11] I must point out that none of the witnesses were present at the birth. They have formulated their opinions based upon the plaintiff's medical records, the antenatal card, the partogram, other labour records, and the neonatal records. They also relied on an MRI scan which was performed on 4 February 2015.

DR LINDA MURRAY

[12] Dr. Linda Murray was plaintiff's first witness. She is an obstetrician and gynaecologist in private practice at the Life Vincent Pallotti Hospital. She lectures in obstetrics and gynaecology at the University of Cape Town, as well as at the University of Stellenbosch. Dr. Murray's expertise was not in dispute. She testified that plaintiff was, at the time of D's birth, a 26 year old woman in her second pregnancy (a multigravida). She was a so-called 'late booker', only presenting herself for examination when her pregnancy was already well advanced. She was HIV positive, and slightly anaemic. Plaintiff was placed on

highly active antiretroviral therapy. None of these risk factors played any role in the later events. Plaintiff's pregnancy progressed normally, and upon admission to the hospital she was classified as a 'low risk' patient. The foetus was lying normally, in a cephalic presentation. Amniotic fluid (or liquor) was normal, and the membranes were intact. In short, there was nothing of concern during the early part of the labour.

[13] Dr. Murray testified that the foetus of a low risk patient may be monitored intermittently by auscultation, by listening to the foetal hart rate before, during and after a contraction. She explained that during a contraction placental perfusion falls and there is less oxygenated blood available in the placenta. The foetus suffers brief episodes of hypoxia, although under normal circumstances this does not cause harm to the foetus. She also explained that it was important to establish the baseline heart rate of the foetus. During a contraction the foetal heart rate might decelerate due to the brief hypoxic event, and it is important to establish whether the heart rate returns to the baseline after a contraction. If it does not, then there is cause for alarm and the patient must be more closely monitored. If necessary there must be emergency intervention to protect the foetus from suffering further harm, either by way of an instrument delivery, or by way of a caesarian section.

[14] Dr. Murray testified that there were two 'red flag', indicators which should have alerted the nursing staff that the patient was at a heightened risk as the labour progressed. The first was the lack of progress in the labour. Dr. Murray said that by 01h20 the progress of labour had crossed the action line. This was in itself unusual as the plaintiff was a multigravida, having given

natural birth previously. The second was the presence of meconium stained liquor grade 2. This is a possible indicator of foetal stress. The staff were sufficiently alarmed that they notified the doctor of the plaintiff's condition. Dr. Murray is of the view that from then onwards the foetus should have been monitored constantly by means of a cardiotocograph ("CTG"). There is no evidence that a CTG was ever employed. The second stage of labour, commencing at full dilation at 02h00 was mildly prolonged, but not unduly so.

[15] Dr. Murray is of the view that there was insufficient monitoring of the foetus after 02h00, and that the partogram is incorrect inasmuch as it records that the baseline heart rate was normal throughout. She says this in light of the fact that D was born in a compromised state, and had to be resuscitated. The child could not have been born in such a compromised state if her heart rate had been normal before birth. Dr. Murray's opinion is in all likelihood correct given the fact that the partogram records that the foetus' heart rate was normal at 04h00, 25 minutes after the baby had already been born. Very little reliance can be placed on the partogram.

[16] Dr. Murray testified that an MRI scan of the child's brain was performed on 4 February 2015 and was diagnostic of an "acute-profound" (central) hypoxic ischaemic injury ("HI injury") of the Basal Ganglia-Thalamic areas of the brain ("BGT"). Often an acute-profound injury occurs fairly quickly as a result of a sudden sentinel event. In this case there is no evidence of a sentinel event. In Dr. Murray's view, the same type of injury may result if the foetus suffers ongoing subthreshold hypoxia over a period of time, which builds up to a point where the injury occurs.

[17] It has thus far been accepted that damage to the BGT structures, the deep grey matter of the brain, always resulted from a sudden total interruption of blood supply to the brain. In Dr. Murray's view, however, the partial intermittent interruption of blood supply to the brain can also result in the type of HI injury that presented itself on the MRI scan as an acute-profound injury.

[18] In order to examine this theory Dr. Murray, in conjunction with Professor Johan Smith and a number of other medical experts, conducted a study of ten specifically identified patients. The purpose of the study was to investigate cases of acute-profound HI injuries in cases where there was no identifiable sentinel event. The findings were published during November 2020 in a peer reviewed article, and concluded that:

"……*if a non-reassuring fetal status develops during labour, and is prolonged, a BGT pattern HI injury may result, in the absence of a perinatal sentinel event.*"

[19] I will deal with the study more fully hereunder. Dr. Murray pointed out that the lack of monitoring after 02h00 had the result that there was no data available as to the status of the foetus, and whether it was in distress. However, in the absence of a sentinel event, and given the evidence produced in the study, Dr. Murray was of the view that the foetus must have been in distress after 02h00, because there is no evidence of distress before that. Prof. Lombaard, defendant's expert agreed with Dr. Murray's view in a joint minute, and added that the insufficient monitoring of the plaintiff of the second stage could have resulted in the foetal distress being missed. In Dr.

Murray's opinion D's BGT HI injury was the result of repeated hypoxic events which built up over a period of time between 02h00 and 03h35, and which culminated in the type of BGT injury commonly referred to as an acute-profound injury.

[20] In Dr. Murray's view the injury would likely not have occurred just before birth. There was no sentinel event that could have caused a sudden injury in a short space of time. Considering the severity of the injury, the hypoxic episodes must have started some time before birth, causing a gradual change in the foetal status. She was furthermore of the view had the foetal heart rate been properly monitored it would have revealed that the foetus was in distress, which would have resulted in medical intervention in the form of an instrument delivery. Had the foetus been delivered immediately upon the distress being noted, the HI injury would in all likelihood have been prevented.

[21] It was put to Dr. Murray in cross-examination that the injury could have been caused by cord compression which would have left no footprint behind. She conceded that there would be no evidence of a cord compression once the compression ceases. In her view that is an unlikely cause of the injury. Typically, a cord compression that is equal to a sentinel event occurs when the cord prolapses and comes out in front of the baby's head. This is not a sudden nor silent event. The probability of a cord becoming so severely compressed that it cuts off blood supply completely is, in Dr. Murray's view, extremely unlikely. In her view the injury was the result of labour stress in a compromised foetus.

PROF. JOHAN SMITH

[22] Professor Johan Smith testified that he is a specialist neonatologist in the Department of Paediatrics at the University of Stellenbosch. He holds the degrees MB ChB, M Med (Paed) and PhD. He attends weekly meetings dealing with high risk pregnancies as well as weekly perinatal mortality meetings during which cases of stillborn babies and neonatal deaths are discussed. Prof. Smith's expertise was not in dispute. He has been a medical practitioner for 40 years, and a neonatologist for 27 years. Prof. Smith confirmed the correctness of his expert summary, as well as the joint minutes between himself and Prof Cooper.

[23] Prof. Smith concurs with Dr. Murray that by 1h20 the plaintiff's labour had crossed the action line and thick meconium stains were observed in the liquor, which indicated that cardiotocograph monitoring was necessary. Between 02h00 and delivery at 03h35 the plaintiff was in the second stage of labour and required continued and careful monitoring. He says that there is no evidence that the foetus was monitored at all during the latter period.

[24] Prof. Smith testified that at 1 minute after birth D's Apgar score indicated a heart rate score of 2, which means that the foetus had not suffered bradycardia. The baby was in respiratory distress and required suctioning. The baby had no grasp reflex, was neurologically depressed, exhibited no "cry", and was floppy. Her blood glucose levels were raised, which is often a stress response. D had difficulty initiating and maintaining respiration and required supplemental oxygen. In Prof. Smith's view, D exhibited a moderate

neonatal encephalopathy. All of her symptoms were consistent with an HI injury resulting from birth asphyxia. The diagnosis of moderate early onset encephalopathy establishes a 'causal pathway' to cerebral palsy.

[25] Prof. Smith testified that there are different types of brain injury which result from different clinical causes. The first is the so-called acute-profound injury which results from a sentinel event, and which causes damage to the gray matter of the brain, the BGT area and the brain stem. The sentinel event causes an abrupt drop in the foetal heart rate to somewhere between 60 and 80 beats per minute. This bradycardia lasts until delivery. The resulting lesions are generally isolated BGT and brainstem related. Prof. Smith says that such injuries are rare because sentinel events are a relatively rare occurrence.

[26] The second type of injury results from a subacute intermittent hypoxic insult which occurs over an extended period of time. In his view, this type of HI insult fluctuates above and below the threshold for hypoxic-ischaemic injury and results in injury to the perirolandic and the BGT areas of the brain. Prof. Smith believes that D's injury falls into this latter category.

[27] The third type of hypoxic insult is not as devastating as, for instance, an acute-profound incident, and results in the brain shunting blood from the watershed areas of the brain to the central deep structures in order to preserve life. This manifests in damage to the white peripheral areas of the brain. This type of injury is not relevant to this matter.

[28] Prof. Smith testified that a sentinel event is a specifically medically defined type of occurrence, which includes uterine tears, abruption placenta, placenta praevia, feto-maternal haemorrhage, maternal cardio respiratory collapse, and an umbilical cord prolapse. In this case there was no sentinel event. In Prof. Smith's view this foetus suffered prolonged intermittent hypoxia, which accumulated and eventually resulted in the so-called acute-profound injury. The MRI scan revealed a Rolandic Basal Ganglia-Thalamic injury (acute-profound pattern).

[29] Prof. Smith says that it is well-known in the literature that in the absence of a sentinel event an "acute-profound" type brain injury may occur over several hours. His contention is partially based upon the article which he and his colleagues have recently published, as well as on other recent publications. He also concedes though that there are experts who disagree with him.

[30] In Prof. Smith's view D suffered repeated hypoxic insults over at least two hours, as a result of which the hypoxic injury developed gradually. He also believes that the HI insults were subthreshold/subacute and intermittent, and that they culminated in a final profound brain injury.

PROF. COOPER

[31] Prof. Peter Cooper testified for the defendant. He simply confirmed that he had read his expert summary, as well as the joint minutes of the meetings between himself and Prof. Smith. He confirmed the contents of those documents. He gave no further evidence and he was not cross-examined. It is

a pity that he did not expound on his opinions, as it would have been useful to hear him on aspects on which he disagreed with Prof. Smith.

[32] Prof. Cooper's summary of the events is substantially the same as that of Dr. Murray and Prof. Cooper. He is of the view that the plaintiff's HIV status was a significant risk factor for an adverse pregnancy outcome. However, there is no evidence that the HIV played any role in the eventual outcome. Prof. Cooper also believes that the foetus displayed an asymmetrical intrauterine growth restriction which was likely due to late placental insufficiency, which would compromise the foetus' ability to tolerate normal labour.

[33] Prof. Cooper's view was that D was at the mild end of moderate encephalopathy. He says that if the encephalopathy can be traced to a hypoxic event or events, the neurological handicap could be linked to this. However, he points out that there are a number of other causes of neonatal encephalopathy, such as meningitis, metabolic causes, vascular events and structural brain abnormalities. Prof. Cooper states that D was not examined for other causes of encephalopathy. Prof. Cooper's expert summary does not show that he had had sight of the radiological report of 4 February 2015, although the joint minute of 11 February 2021 seems to suggest that he knew about the report. The MRI report specifically states that the MRI scan showed no congenital abnormalities or genetic disorders, nor any inflammatory brain disease. In this regard Prof. Cooper's expert summary seems clearly contradicted by the evidence of Prof. Smith and the MRI scan.

[34] At best for defendant, Prof. Cooper only says that the neurological features are suggestive of a peripartum hypoxic ischaemic event, but that other causes than intermittent hypoxia may have been responsible for the injury.

[35] The joint minute of the meeting between Professors Smith and Cooper on 25 June 2020 records that they agree on the basic facts relating to the pregnancy. Prof. Cooper was of the view that the foetus suffered from placental insufficiency, a view that Prof. Smith agreed with in the second minute of 11 February 2021. Both experts agreed that D's condition was in keeping with birth asphyxia, although Prof. Cooper continued to express the view that there might have been other causes of the respiratory distress. Both experts agreed in the second minute that there was no evidence that infection, genetic or anatomical abnormalities had played a causal role in the injury.

[36] The second joint minute also records that Professors Cooper and Smith agree that early onset encephalopathy establishes a "doorway in a causal pathway between intrapartum asphyxia and cerebral palsy." Although Prof. Cooper did not disagree with this basic hypothesis, he noted that this only applied in cases of moderate encephalopathy. However, Prof. Cooper had already expressed the opinion in his expert summary that D had suffered a moderate encephalopathy, albeit on the mild side.

[37] Both experts agreed that there had not been a sentinel event. Prof Smith opined that in the absence of a sentinel event, the probable cause of

the injury was suboptimal/substandard intrapartum obstetric management. Prof. Cooper was of the view that substandard intrapartum management had to be demonstrated in each specific case. Prof Cooper also expressed the view that current neonatal opinion was that an acute-profound HI injury of this type could only occur in cases of total or near total cessation of blood flow for a period of 10 to 45 minutes.

[38] Mr. Rossouw argued on behalf of defendant that Prof. Cooper's statements in the joint minutes and in the expert summary must be accepted as evidence, and that, in the absence of cross-examination it stands uncontroverted. He says so on the basis of ***BEE v Road Accident Fund*** [1], a matter in which the evidence contradicted the joint report of forensic accountants and facts that had been agreed in a pre-trial minute. I do not agree. The following principles relating to expert witnesses can be extracted from ***BEE*** and other authorities:

[38.1] An expert witness is there to assist the court and not to usurp the function of the court.

[38.2] Expert witnesses are required to lay a factual basis for their conclusions and explain their reasoning to the court;

[38.3] The court must be satisfied as to the correctness of the expert's reasoning;

[1] 2018 (4) SA 366 (SCA)

[38.4] Absent any reasoning, the opinion is inadmissible;[2]

[38.5] A court is not bound by an expert's opinion;[3]

[38.6] For an expert's evidence to be helpful he or she has to be neutral;[4]

[39] It is not correct therefore to understand ***BEE*** to mean that a court is bound to blindly accept whatever an expert says.

[40] Prof. Cooper states that D's placental insufficiency would have exacerbated any foetal distress. He says that if the neonatal encephalopathy was due to peripartum HI, D's subsequent neurological encephalopathy could be linked to this. However, he says, there are many other causes of neonatal encephalopathy, such as meningitis, septicaemia, metabolic causes, vascular events and structural brain abnormalities. Prof. Cooper then says:

> *"There were no details in the neonatal or follow up record to indicate that she was investigated for other causes of encephalopathy."*

[41] If one has regard to the documents that Prof. Cooper examined in order to prepare his expert report, it appears that he did not consider the MRI scan and the three radiological reports dated 13 June 2019, 18 June 2020, and 21 January 2021 respectively. The first joint minute refers to the

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[2] Masstores (Pty) Ltd v Pick and Pay Retailers (Pty) Ltd 2016 (2) SA 586 (SCA)
[3] Road Accident Appeal Tribunal v Gouws and another [2017] ZASCA 188; [2018] 1 ALL SA 701 (SCA); Michael and another v Linksfield Park Clinic (Pty) Ltd and another [2002] 1 ALL SA 384 (A); 2001 (3) SA 1188 (SCA)
[4] Stock v Stock 1981 (3) SA 1280 (A) at 1296 E-F; Jacobs v Transnet Ltd t/a Metrorail 2015 (1) SA 139 (SCA); [2014] ZASCA 113

"subsequent reported MRI", and therefore Prof. Cooper was presumably aware at that stage that there had been an MRI scan. The radiological report of 13 June 2019 records that no genetic disorders or congenital anomalies were found, and no sign of inflammatory brain disease. When Prof. Cooper says in the expert summary that there were no investigations into other possible causes he is plainly wrong. Prof. Cooper contradicted his expert report by agreeing with Prof. Smith in the first joint minute that there is no evidence to show that infection, genetic or anatomical abnormalities played a causal role.

[42] In the first joint minute Prof. Smith stated that a diagnosis of early onset encephalopathy establishes an essential 'doorway' in a causal pathway between intrapartum asphyxia and cerebral palsy. Prof. Cooper disagreed, suggesting that the mild HI encephalopathy found in this case would not have resulted in later major neurological handicap and cerebral palsy. The difficulty is that in Prof. Cooper's own expert report he says:

> *"However that fact that she was able to feed from a cup on the third day, was well enough to receive her first immunizations on the fourth day and could be discharged on the sixth day indicated that this was at the mild end of the spectrum <u>of moderate encephalopathy.</u>*

[43] Prof. Cooper was in agreement that no sentinel event occurred. However, he makes no suggestion as to a possible cause of the injury.

[44] In the second joint minute Prof. Cooper's attention was specifically drawn to the study by Smith *et al.* His only response was to say that the paper

had only been published online and only when it was published in print would peer review be able to take place. He said that until peer review had taken place, the paper could not be accepted. I find it strange, in the era of the internet, that comment would only follow on publication in hard copy. This criticism was also put to Prof. Smith. He explained that the paper had been submitted to the American Journal of Perinatology, where it had been subjected to comprehensive peer review before it was published. He said that Prof. Cooper was confusing pre-publication peer review with post-publication comment.

[45] I find it hard to believe that a journal of such standing would publish a paper without proper peer review having first taken place. In any event, Prof. Cooper is one of Prof. Smith's peers. If he had any qualms about the study, its methodology, the data, or its interpretation, he would no doubt have raised his concerns.

[46] Prof. Cooper failed to deal with any of the other recent publications (ACOG and Volpe) which contradict his position. Mr. Rossouw submitted that these studies could not be relied upon because they based their findings on experiments on monkeys and sheep. Firstly, this is not correct. The studies also included human subjects. As I understand, Volpe is reliant on studies by a number of experts in the field who have observed numerous human cases. Secondly, Prof. Smith testified that animal studies were relevant albeit that one had to be careful what conclusions were drawn from those studies. The Smith *et al* study included animal experiments, but was also based, as Prof.

Smith explained, on his experience (and that of his co-authors) over many years, involving human cases.

[47] The main bones of contention between the parties were therefore the following:

[47.1] Has plaintiff demonstrated that subthreshold/subacute intermittent HI insults that build up over an extended period of time can cause this type of acute-profound type injury of the BGT and perirolandic brain structures?

[47.2] If the answer to the above question is in the affirmative, then is there evidence to suggest that such insults occurred in D's case?

[47.3] If there is evidence that there were such insults, then would proper monitoring have alerted the nursing staff to the fact that the foetus was in distress?

[47.4] If the nursing staff had been forewarned that the foetus was in distress, would immediate intervention have prevented the injury?

[48] If all of the above questions are answered in the affirmative, causation would have been established.

CAN A SUBACUTE INTERMITTENT HYPOXIC ISCHAEMIC INSULT OVER A PERIOD OF TIME RESULT IN AN ACUTE-PROFOUND TYPE INJURY?

[49] It has thus far been widely believed that only an acute-profound insult such as a sentinel event can cause this type of BGT HI injury. In ***AN o.b.o. EN v Member of the Executive Council for Health, Eastern Cape***[5] the Supreme Court of Appeal ("SCA") dealt with a similar claim for damages resulting from birth asphyxia where the medical care had been substandard. The central point for determination was whether causation had been established.

[50] In ***AN*** the foetus had suffered an acute-profound HI insult, a cord compression, which resulted in a total cessation of blood supply to the brain. The SCA accepted that only an acute-profound total interruption of the blood supply could have caused the type of BGT injury found in that case. It accepted that if the interruption of blood supply was intermittent, the white peripheral matter in the watershed areas would have been injured. In other words, the SCA accepted the conventional view as it was at that time. Prof. Smith testified in that case. His evidence was rejected as being speculative and his opinion was rejected on the basis that it was not supported by authoritative peer-reviewed literature.

[51] The SCA therefore upheld the finding by Dawood J in the court *a quo* where she said (regarding the defendant's witness, Buchman)[6]:

> *"Professor Buchman however stated that although that is what he relied on, the exclusion of that would not change his opinion since it was still an acute-profound event that would have occurred in the*

[5] [2019] ZASCA 102; [2019] 4 ALL SA 1 (SCA)
[6] [2018] 2 ALL SA 678 (ECM)

last half an hour prior to delivery according to the articles relied upon and therefore even with no monitoring the outcome would have been the same.

(h) The sub-standard care and failure to adequately monitor was not a causative factor in this case according to him.

(i) His opinion as to how the insult and resultant injury occurred and that it was an acute-profound is as already indicated in keeping with the medical authorities cited and I find it the more probable explanation as these are the only available medical authorities at this time that have been peer reviewed and despite the criticism levelled that it was not of a big enough sample and old, it is authoritative until contrary findings and outcomes are made in peer reviewed published articles.

(j) The plaintiff's expert's opinions in other litigated matters accordingly cannot be accepted as being authoritative without knowing the full history of each case and having that data checked by experts in the relevant fields and peer reviewed."

[52] The ***AN*** case is distinguishable from this case for a number of reasons. Firstly, in ***AN*** there had been a sentinel event, a cord compression, which resulted in the injury. Secondly, the latest authorities on this issue were not yet available to the SCA, and it based its finding on authority which is now very much in question. Thirdly, the defendant in ***AN*** called a witness who gave convincing evidence that a sentinel event had occurred. Prof. Johannes

Buchanan testified that the injury was sudden, without warning and severe. Intervention by Ventouse or forceps delivery or caesarian section could not have occurred quickly enough to prevent the harm in his view, and consequently there was no causal connection between the sub-standard care and the outcome.

[53] In ***The Member of the Executive Council for Health, Eastern Cape v Mpetsheni***[7] the SCA was faced with a similar case. The plaintiff had given birth to a severely brain damaged baby as a result of a hypoxic ischaemic insult. In that matter the accepted evidence was that the foetus had suffered an acute-profound injury, probably a compression of the cord, akin to a sentinel event. This proposition was accepted by the plaintiff's expert. That matter is equally distinguishable from this matter as the experts agree that in this case that there was no sentinel event.

[54] I was handed a series of articles dealing with so-called 'acute profound' injuries, commencing with Joseph Pasternak *et al's* publication in 1998[8]. Pasternak studied eleven infants that had sustained an acute near-total intrauterine asphyxia at the end of labour, due to bradycardia. Pasternak's study postulated that in these cases there had been an acute near-total intrauterine asphyxia at the end of labour. According to Pasternak imaging studies documented a consistent pattern of injury in subcortical brain nuclei, including thalamus, basal ganglia and brainstem. In contrast the cerebral cortex and white matter were completely or relatively spared.

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[7] [2020] ZASCA 169 (14 December 2020)
[8] Pasternak, J and Gorey, M: *The Syndrome of Acute Near-Total Intrauterine Asphyxia in the Term Infant*, Pediatr Neurol 1998 18: 391-398

[55] This study was criticized by Alistair Mc Lennan in 1999 on the grounds that he believed that of the eleven babies studied, ten did not meet the strict perinatal definition for asphyxia. Prof. Smith agrees with this criticism. Pasternak, however, later reaffirmed his belief that all of the babies had suffered asphyxia. Prof. Smith also criticized the basis of the study, and specifically Pasternak's claim that imaging studies documented a pattern of injury consistent with BGT and brainstem injury, as opposed to cerebral cortex damage:

[55.1] Of the eleven patients in the study, four had no radiological evidence for any brain injury whatsoever;

[55.2] Only one case had a BGT type mid-brain injury which Volpe calls the deep nuclei stem pattern injury;

[55.3] Three cases had BGT and perirolandic cortex deep nuclei pattern injuries, similar to this case;

[55.4] Three patients had an isolated BGT injury.

[56] Therefore only 4 out of 11 cases had the deep nuclei-brainstem type injury which is associated with acute-profound type events according to Volpe and the American College of Obstetricians and Gynaecologists ("ACOG"). Pasternak's general statement regarding the consistency of the injuries through the cases is shown to be inconsistent with the data. Only in five of the cases was a sentinel event identified. In all of the Pasternak cases a terminal fixed bradycardia occurred which lasted between 10 and 45 minutes. That

distinguishes the Pasternak cases from this case where it is unlikely that a fixed bradycardia occurred.

[57] Okumura[9] reported on a study of two patients. Both had allegedly suffered a sudden fall in foetal heart rate and had suffered an HI insult. In referring to the Pasternak study, Okumura remarked that the foetuses had suffered the same type of sudden hypoxic incident as in the Pasternak study. Both cases revealed injury to the BGT areas. On this basis, Okumura postulated that sudden acute hypoxia leads to injury to the BGT structures, while gradual intermittent hypoxia resulted in injury to the watershed areas. Okumura's study however shows that the second foetus did not suffer a sudden bradycardia. It displayed an erratic heart rate which fluctuated significantly, until the mother suffered prolonged repeated contractions. At that point the foetus suffered a sudden bradycardia.

[58] Essentially only one of Okumura's patients is comparable to the Pasternak group. Nevertheless, after the Pasternak and Okumura studies were published, it seems to have been widely accepted that only acute-profound insults result in BGT injury. Radiologists started referring to the pattern on an MRI scan which involved the BGT structures as an acute-profound injury, the name suggesting the pathophysiology of the injury. Thereafter, when an MRI scan produced an image showing BGT injury, the injury was referred to as acute-profound and it was accepted that the insult that caused the injury had been acute (sharp or severe) and profound (intense).

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[9] A Okumura, F Hayakawa, T Kato & K Watanabe, *Bilateral Basal Ganglia-thalamic Lesions Subsequent to Prolonged Fetal Bradycardia*, (2000) 58 Early Human Development 111

[59] In 2011 Rennie and Rosenbloom published a study[10] which was aimed at determining the time from insult to foetal HI injury in animals and human babies. The article was restricted to the acute-profound HI model. Again, as with Okumura, the authors accepted the existing view that only acute-profound insults resulted in injury to the BGT structures. The study was aimed at determining the time available to deliver a foetus in the event of an acute-profound event. In itself the study does not provide authority for the proposition that only an acute-profound event would result in a BGT injury.

[60] Deidre Murray *et al* published a study[11] in 2009 which sought to examine foetal heart rate patterns during labour and to relate those findings to neurodevelopmental outcome. Of 35 foetuses studied, the second group relates most closely to this case: Those were infants who had been admitted with a normal CTG trace, who then deteriorated over a period of time to become pathological. Only four of the subjects of the study suffered sentinel events. Those insults resulted in the most severe injury, but the study did not exclude the possibility that prolonged and intermittent HI incidents could result in BGT injury. Murray is neither supportive nor dismissive of the Pasternak theory, in my view.

[61] Later publications discussed the possibility of injury to the BGT areas other than through an acute-profound incident or a sentinel event. ***Volpe's***

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[10] Rennie J and Rosenbloom L, *How long have we got to get the baby out? A review of the effects of acute and profound intrapartum hypoxia and Ischaemia,* The Obstetrician & Gynaecologist, 2011 13: 169 - 174

[11] Murray, D, O'Riordan, M, Horgan, R, Boylan, G, Higgins, J, Ryan, C: *Fetal Heart Rate in Neonatal Hypoxic-Ischemic Encephalopathy: Relationship with Early Cerebral Activity and Neurodevelopmental Outcome*, Am J 2009;26:605-612

Neurology of the Newborn[12] is an authoritative publication which is referred to by neonatologists worldwide. The major patterns of neuronal injury are discussed by Volpe and three are identified[13]:

[61.1] The diffuse pattern involving the cerebral cortex, deep nuclear and brain stem. In these instances the insult is severe and prolonged.

[61.2] The cerebral cortex-deep nuclear which is caused by a moderate prolonged insult.[14]

[61.3] Deep nuclear-brain stem, in which the insult is severe and abrupt.

[62] Volpe comments as follows:[15]

"In the more prolonged and less severe insults, the diversion of blood to deep nuclear structures occurs to a degree, and thus the cerebral regions are more likely to be affected. Studies in the near-term fetal lamb indicate that the severe terminal insult that results in injury to deep nuclear structures especially may be likely to occur after brief, repeated hypoxic-ischaemic insults first cause a cumulative deleterious effect on cardiovascular function that presumably then can result in a severe late insult…..

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[12] Volpe, J et al, 2018 *Volpe's Neurology of the Newborn*, 6th Ed
[13] Chapter 19
[14] This is the injury found in the present case.
[15] At page 502 and 503

Term fetal monkeys subjected to a partial rather than total interference with respiratory gas exchange (produced by halothane-induced hypotension) developed physiological derangements over longer periods, so-called prolonged partial asphyxia. The maternal event resulted in more slowly evolving hypoxia and acidosis, followed by decelerations of the fetal heart rate, diminished cardiac output, hypotension and evidence for cerebral ischemia. Brain injury became apparent after several hours, and the topography involved cerebral cortex (especially in paracentral areas), basal ganglia and thalamus, as in human infants (see Table 19.1)"

[63] ACOG supported this view in 2019.[16] The ACOG publication acknowledges that a severe partial insult of prolonged duration or a combined partial with profound terminal insult can cause deep nuclear neuronal injury:

"The second form of selective neuronal injury is the cerebral-deep nuclear neuronal injury, which combines neuronal damage in the deep nuclear gray matter with injury in the cerebral cortex, usually the parasagittal areas of the perirolandic cortex.....In experimental models, the mechanism responsible for this pattern is described as a 'prolonged partial with final total asphyxia" with a moderate to severe insult evolving in a gradual manner leading to a potential complete 'asphyxia'"

[16] American College of Obstetricians and Gynaecologists; *American Academy of Pediatrics: Neonatal Encephalopathy and Neurological Outcome*, 2nd Ed, Chapter 10.

[64] The final study, that of Smith *et al,* was published very recently.[17] The authors reviewed 195 cases of neonatal encephalopathy. After excluding a number of cases, ten were identified for inclusion in the study, all of them displaying term gestation neonatal encephalopathy-cerebral palsy. None of the cases were associated with intrapartum fever, congenital abnormalities, metabolic errors, intracranial infections, septic shock or cranial birth trauma. None had suffered prenatal injury. Cases with genetic disorders were excluded. All cases displayed an acute-profound HI pattern on MRI, had clearly documented evidence of an assessment of the foetus on admission and underwent intermittent adequate foetal monitoring during labour. In none of the cases was a sentinel event identifiable. Injuries that did not involve the BGT areas, or those with a mixed or a watershed injury were also excluded.

[65] The study showed that if a non-reassuring foetal status develops during labour, is prolonged and leads into a pathological CTG tracing, the brain injury pattern may be the same as in the case of an acute-profound BGT HI injury. The authors wrote:

> *"The present case series shows that in the absence of a perinatal sentinel event, subacute or subthreshold intrapartum HI occurs in the human fetus and that warning signs in the form of a NRFS, in some instances hours before delivery, may occur. This paper supports the notion that with appropriate intrapartum care and timeous reaction to FHR abnormalities and action in the form of intrapartum resuscitation*

[17] Smith J, Solomons R, Vollmer L, Langenegger E, Lotz, J, Andronikou, A, Anthony, J, and Van Toorn R: *Intrapartum Basal Banglia-Thalamic Pattern Injury and Radiologically termed "Acute-profound Hypoxic-Ischaemic Brain Injury" are not Synonymous, Am J Perinatol 2020 Dec 15. Doi: 10.1055/s – 0040-1721692*

and expedited delivery, in the majority of cases adverse BGT pattern injury would have been prevented."

[66] The conclusion was:

"This study shows that if a nonreassuring fetal status develops during labour and is prolonged, a BGT pattern HI injury may result, in the absence of a sentinel event."

[67] There is no substantive evidence from the defendant to refute Prof. Smith's version. I would have expected defendant to put up some evidence as to the cause of the injury. I say so in the full understanding that defendant does not bear an onus of proof. However, when the plaintiff presents a well-reasoned opinion, one would expect the defendant to put up some version of its own.[18] Defendant did not even put up a version during cross-examination. I therefore accept Prof. Smith's evidence, that a series of partial intermittent, subacute/subthreshold hypoxic insults may result in this type of injury to the BGT deep nuclear structures including the perirolandic area.

DID THE FOETUS SUFFER PARTIAL INTERMITTENT HYPOXIC INSULTS, AND DID THIS CAUSE THE INJURY TO THE BGT AREAS?

[18] Goliath v Member of Executive Council for Health, Eastern Cape 2015 (2) SA 97 (SCA) par. 19: "*That being so, the MEC, in failing to adduce any evidence whatsoever, accordingly took the risk of a judgment being given against him. After all, it was open to the MEC to adduce evidence to show that whilst Ms Goliath was undergoing surgery, reasonable care had indeed been exercised by his employees. That he did not do.*"
See also: Ntsele v MEC for Health, Gauteng Provincial Government [2013] 2 ALL SA 356 (GSJ) at par. 42

[68] Prof. Smith and Dr. Murray approached this question by applying inferential reasoning. They considered the observed and admitted facts, eliminated all other possible causes, and then deducted that the most likely cause of the injury was partial intermittent hypoxia. Two undisputed facts are important. Firstly, labour in itself is stressful, and during a contraction the foetus may suffer brief bouts of hypoxia. Some foeteses are inherently better equipped to cope with the stresses of labour. Others cannot cope as well, and may suffer some form of injury. Secondly, it is common cause that this particular foetus suffered from placental insufficiency.

[69] It is also common cause that there was no sentinel event. The plaintiff displayed certain risk factors (HIV and anaemia), but there is no evidence to suggest that these risk factors played any role in the outcome. The plaintiff suffered a brief bout of raised blood pressure at 18h00 for which she received medication. There is no evidence that the hypertension persisted, and it is not of significance to the outcome.

[70] At 1h50 meconium (foetal stool) grade 2 stained liquor was observed. Meconium staining is an indicator of possible foetal stress. If the foetus were to suffer a severe enough bout of hypoxia, the shunting of blood would move the available oxygenated blood to the brain and away from the gut, causing the relaxation of the foetal anal sphincter, thus releasing meconium. It is possible that the foetus was under some stress at that stage already, but it is Dr. Murray and Prof. Smith's view that in all likelihood the serious hypoxic bouts occurred between 02h00 and birth at 03h35.

[71] The "Summary of Labour" form records that a retroplacental clot was observed. This could imply that a placental abruption occurred, which would have resulted in heavy bleeding. In such cases the mother displays signs of shock and a hard and distended uterus. Dr. Murray could not completely exclude the possibility of a placental abruption, but the fact that there was minimal blood observed at delivery (150 ml), and no bleeding reported during labour, causes her to deduct that it is unlikely that there was a placental abruption. It is common for a clot to form after delivery due to the separation of the placenta from the placental bed. The available studies reveal no cases that demonstrate that a mild abruption may cause HIE, or cause the baby to require advanced resuscitation, or result in a low 5-minute Apgar score. Most likely placental abruption did not cause the injury. Prof. Lombaard, the defendant's obstetrician agrees that there is no evidence to suggest that there was a significant abruption, according to the joint minute of 8 February 2021.

[72] There was a further suggestion that the foetus had perhaps suffered a bradycardia of unknown origin. Prof. Smith testified that if the foetus had suffered a total cut-off of blood flow, it would have resulted in a fixed bradycardia that would have persisted until birth. At 1 minute of life the baby's heart rate scored 2 on the Apgar test, in other words, it was normal. It is highly unlikely, according to Prof. Smith, that the foetus could have experienced a fixed bradycardia which persisted until birth, and which resolved itself in the 1 minute until the Apgar test was performed. If there had been a fixed bradycardia, it would have occurred in the 10 to 20 minutes before birth. In this case there is no evidence of a fixed bradycardia.

[73] It was suggested to Prof. Smith in cross-examination that a cord compression might have occurred which would not have left a footprint. He testified that while a cord compression was not strictly speaking a sentinel event, if it were complete and sustained it could have the same effect as a sentinel event. That would have meant that there was a cord compression in the 20 minutes before delivery which was sustained, resulting in a bradycardia (the heart rate falling to 60 to 80 beats per minute). If that were so, in Prof. Smith's opinion the bradycardia could not have resolved itself so quickly that at 1 minute of life, when the first Apgar test was performed, the heart rate was normal.

[74] Mr. Rossouw submitted that there was no evidence that the foetus was in any distress prior to birth. He makes this submission on the premise that the partogram is correct. The problem with this argument is two-fold. Firstly, the partogram is in dispute. It is patently wrong in some respects, even to the point of recording a foetal heartbeat after delivery. Secondly, the condition of the baby at birth leaves no doubt that the foetus must have been in distress during the second stage.

[75] Mr. Rossouw criticized the method used by plaintiff's experts in coming to their conclusion, calling it 'backward' reasoning which, he said, is impermissible. He referred to ***Goliath*** (*supra*) as authority for the proposition. In my view ***Goliath*** is not authority for that proposition. That case concerned the application of the *res ipsa loquitur* principle to establish negligence.

[76] In a minority of cases (because they are rare), the cause of the injury is obvious: A placental abruption, uterine tear etc. In some instances the cause would not be obvious, and would have to be determined, if possible, on the available circumstantial evidence, and considering the probabilities. In ***South African Post Office v De Lacy and another***[19] it was held:

> *"The process of inferential reasoning calls for an evaluation of all the evidence and not merely selected parts. The inference that is sought to be drawn must be 'consistent with all the proved facts: If it is not, then the inference cannot be drawn' and it must be the 'more natural, or plausible, conclusion from amongst several conceivable ones"*

[77] In this case, the fact that there was no sentinel event eliminates the possibility of an acute-profound insult. The latest studies support the plaintiff's experts in their view that an intermittent sub-acute repeating hypoxic event may cause such an injury. All of the admissible evidence points to such an event being the most probable cause in this case. No evidence of any moment was presented by defendant and Prof. Cooper's opinion can be criticized on a number of grounds. Defendant never put forward any case whatsoever, save to say that plaintiff cannot provide direct evidence as to what caused the injury, and that plaintiff was speculating.

[78] Mr. Rossouw argued that there was no evidence as to what happened after 02h00. In this regard he is correct, but the cause of the lack of evidence

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[19] 2009 (5) SA 255 (SCA) [2009] ZASCA 45 at par. 35; See also R v Blom 1939 AD 158 at 202 – 203; Ocean Accident and Guarantee Corporation Ltd v Koch 1963 (4) SA 147 (A); Gordhan and others v Public Protector and others [2020] ZAGPPHC, 777 (17 December 2020), par 83

is the appalling record keeping by the nursing staff. I was urged by Mr. Ströh to draw a negative inference against defendant for not calling any witnesses who were present during the labour. Mr. Rossouw argues that plaintiff is equally to blame for not calling witnesses. This issue was discussed in ***Munster Estates (Pty) Ltd v Killarney Hills (Pty) Ltd***[20]:

> *"See, eg, Elgin Fireclays Ltd v Webb 1947 (4) SA 744 (A) in which WATERMEYER CJ stated (at 749, 750):*
>
> *"It is true that if a party fails to place the evidence of a witness, who is available and able to elucidate the facts, before the trial Court, this failure leads naturally to the inference that he fears that such evidence will expose facts unfavourable to him. (See Wigmore ss 285 and 286.) But the inference is only a proper one if the evidence is available and if it would elucidate the facts."*
>
> *In my opinion, however, it is to be doubted whether WATERMEYER CJ intended laying down a general and inflexible rule to be applied without more in every case where a party fails to call as his witness one "who is available and able to elucidate the facts". Whether the inference, that the party failed to call such a person as a witness because he "fears that such evidence will expose facts unfavourable to him", should be drawn could depend upon the facts peculiar to the case where the question arises. It was pointed out in Webranchek v L K Jacobs & Co Ltd 1948 (4) SA 671 (A) at 682 that it might appear that the person*

[20] 1979 (1) SA 621 (AD); [1979 3 ALL SA 485 (AD)

concerned was equally available to both parties, and that the inference could then be drawn against both parties. VAN DEN HEEVER JA also stated:

'After all, plaintiff was entitled to rest his case upon evidence which he considered adequate to discharge the onus which lay upon him.'"

[79] The trial was held nearly eleven years after the fact. There is no evidence that any of defendant's potential witnesses were available to testify. In these circumstances I am not inclined to draw any inference against either of the parties for not calling the nurses or the doctor.

[80] Mr. Rossouw submitted that Prof. Smith has a proverbial 'dog in the fight', and that as a forensic expert he stands to gain if his evidence were to be accepted, because the State would then be swamped by claims. Prof. Smith is a medical practitioner and specialist of many years' standing. He is evidently well regarded in his field. He gave evidence which was well reasoned, and he supported his opinions with facts. He made concessions where it was appropriate to do so. As much as the Supreme Court of Appeal has warned against imputing bias to a witness in ***Mpetsheni*** (*supra)*, it is also important not to impute self-interest to a witness of high standing, as a motive for his evidence.[21] I decline to make such a finding.

[21] Mpetsheni par. 39

[81] Plaintiff must establish her case on a preponderance of probability.[22] Plaintiff does not have to establish what caused the injury on any level above what is most probable. In my view plaintiff has established:

[81.1] that the BGT type injury shown on the MRI scan can be caused by a subthreshold/subacute, intermittent series of HI insults; and,

[81.2] that, on the probabilities, such HI events caused the injury to the foetus' BGT and perirolandic areas.

CAUSATION

[82] The final question is whether the negligent omission by the defendant's employees to properly monitor the plaintiff, and to take emergency action by instrument delivery or caesarian section caused the harm.

[83] In ***Minister of Police v Skosana***[23] the Court said the following on causation:

> *"Causation in the law of delict gives rise to two rather distinct problems. The first is a factual one and relates to the question as to whether the negligent act or omission in question caused or materially contributed to (see Silva's Fishing Corporation (Pty.) Ltd. v. Maweza, 1957 (2) S.A. 256 (A.D.) at p.*

[22] Miller v Minister of Pensions 1947 2 ALL ER 372 at 373: *"If the evidence is such that the tribunal can say: 'We think it more probable than not" the burden is discharged, but, if the probabilities are equal, it is not."*
[23] 1977 (1) SA 31 (A); [1977 1 ALL SA 219 (A)

264; Kakamas Bestuursraad v. Louw, 1960 (2) S.A. 202 (A.D.) at p. 222) the harm giving rise to the claim. If it did not, then no legal liability can arise and cadit quaestio. If it did, then the second problem becomes relevant, viz. whether the negligent act or omission is linked to the harm sufficiently closely or directly for legal liability to ensue or whether, as it is said, the harm is too remote. This is basically a juridical problem in which considerations of legal policy may play a part. The distinction between these two enquiries is well explained by Prof. Fleming, The Law of Torts, 4th ed., p. 169, as follows:

"The first involves what may broadly be called the 'factual' question whether the relation between the defendant's breach of duty and the plaintiff's injury is one of cause and effect in accordance with 'scientific' or 'objective' notions of physical sequence. If such a causal relation does not exist, that puts an end to the plaintiff's case, because no policy can be strong enough to warrant the imposition of liability for loss to which the defendant's conduct has not in fact contributed.

The second problem involves the question whether, or to what extent, the defendant should have to answer for the consequences which his conduct has actually helped to produce. There must be a reasonable connection between the harm threatened and the harm done. As a matter of practical politics, some limitation must be placed upon legal responsibility,

because [24]*the consequences of an act theoretically stretch into infinity. The task is to select those factors which are of sufficient significance to justify the imposition of liability and to draw a boundary along the line of consequences beyond which the injured party must either shoulder the loss himself or seek reparation from another source."*

[84] In the colloquial, the first leg of the test is called the "but for" test.[25] The plaintiff has to prove on a balance of probabilities[26] that the harm would not have ensued but for the act or omission. The second part of the test is whether the act or omission is so remote from the harm that legal liability should not follow.

[85] When considering whether an act is causally linked to the harm, the act is eliminated and one then considers whether the harm would still have ensued. However, an omission cannot be thought away, and a positive act must be inserted into the facts.[27] It was held in ***Lee*** *(supra)* that the application of the test should not be inflexible. In some instances the inflexible application of the rule would result in an injustice. Each case, the Constitutional Court said, had to be decided on its own facts. In ***Minister of Safety and Security v Van Duivenboden***[28] the court said:

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[25] De Klerk v ABSA Bank Ltd 2003 (4) SA 315 (SCA); [2003] 1 ALL SA 651 (SCA)
[26] International Shipping Co (Pty) Ltd v Bentley 1990 (1) SA 680 (A) at 700 E – 701 F; [1990] 1 ALL SA 498 (A)
[27] Lee v Minister of Correctional Services 2013 (2) SA 144 (CC); [2012] ZACC 30
[28] 2002 (6) 431 (SCA)

There are conceptual hurdles to be crossed when reasoning along those lines for once the conduct that actually occurred is mentally eliminated and replaced by hypothetical conduct questions will immediately arise as to the extent to which consequential events would have been influenced by the changed circumstances. Inherent in that form of reasoning is thus considerable scope for speculation which can only broaden as the distance between the wrongful conduct and its alleged effect increases."

[86] It is in these kind of cases therefore necessary to import into the equation the conduct that one would reasonably have expected from trained medical personnel, and then to determine whether the outcome would have been different.

[87] Dr Murray testified that a hypoxic event would in all likelihood be triggered by a contraction and would result in a sudden deceleration of the heart rate. The labour protocols require the use of a CGT monitor, or proper auscultation if a CTG is not available, to monitor the heart rate of the foetus and the contractions simultaneously, more especially in high risk cases (which this was from 1h20 onwards). If a CTG is not employed, auscultation must be done before, during and after each contraction, to confirm that the heart rate has returned to the baseline.

[88] If the foetus suffered an HI event, and such event was repeated intermittently, it would inevitably have had an effect on the foetal heart rate in the form of a deceleration, and there would have been indications of distress

which would have been revealed by proper monitoring. Considering that the injury was not sudden, and was likely sustained over a long period of time, proper monitoring would most likely have resulted in emergency intervention. Dr. Murray and Prof. Lombaard agree that the foetal condition changed between 02h00 and 03h35. They agree that had the foetus become distressed during the second stage of labour (after 02h00) then proper management would have included a prompt recourse to an instrument delivery. In a multiparous patient, with the foetus' head being engaged, such an intervention would probably have been successful. Prof. Lombaard specifically recorded that the insufficient monitoring of the second stage could have caused the foetal distress to be missed.

[89] If one were to insert proper medical care and monitoring of the foetus and the plaintiff into the scenario, it is probable that the foetal distress would have been recognised, and appropriate action would have been taken which probably would have prevented the harm that eventually ensued. Factual causation has thus been established. In this case the harm is not remote from the omission, and there is no question that it was foreseeable that improper care and monitoring might result in the foetus suffering an injury. Legal causation is also established.

[90] Mr. Rossouw argued that if the plaintiff were to succeed not only would it have the effect of the State being held strictly liable in cases where no discernible sentinel event was present, but it would also bankrupt the State.

[91] As for the first proposition, this case must be decided on the facts before the Court. In this particular case the evidence points to defendant being liable for the damages suffered. In the cases referred to above, ***AN*** and ***Mpetsheni*** other facts were presented, and the claims were dismissed. If a plaintiff can produce sufficient evidence to prove her claim, she is entitled to a judgment. Conversely, if the plaintiff cannot produce evidence to support her claim, she will not succeed. I do not believe that his case would open the floodgates of litigation.

[92] As for the second proposition, that the State would be bankrupted if this claim succeeds, I must emphasize that I fully realise the importance of this matter for the State. However, the effect of a case on the fiscus is not a consideration in a delictual matter.

[93] I consequently find that the omission by the nursing staff of the hospital to properly monitor the plaintiff and the foetus, to recognise that the foetus was in distress, and to take immediate and urgent steps to deliver the foetus, caused the subacute intermittent Hypoxic-Ischaemic insults to build up to a point where the BGT and perirolandic areas of the foetus' brain were injured. That injury resulted in D now suffering from severe asymmetrical mixed type cerebral palsy, predominantly dystonic.

COSTS OF 27 JULY 2020

[94] The matter was previously set down for hearing on 27 July 2020 but was postponed by agreement between the parties, costs being reserved for determination during the trial.

[95] Plaintiff argued that defendant had caused the postponement, and in support of this contention it presented a letter dated 3 August 2020 in which plaintiff's attorney wrote that;

> [95.1] Defendant's expert report in respect of its paediatric neurologist and the signed joint minute of the paediatric neurologists were only delivered to plaintiff on the afternoon of 22 July 2020, five days before the trial;
>
> [95.2] Prof. Lombaard, defendant's obstetrician and gynaecologist, raised the issue of the positioning of the foetus being inconsistent with an assisted delivery at the last minute, only to concede in a joint minute which only became available on 24 July 2020 that assisted delivery could have succeeded within 20 minutes of the detection of distress.

[96] In an email dated 6 August 2020 defendant's counsel, Ms. Pretorius, denied that defendant's actions had caused the postponement, and she emphasized that defendant had been ready to proceed. She alleged that plaintiff had sought the postponement. Plaintiff's allegations regarding late delivery of the expert summary, and the raising of contentious issues shortly before trial were not answered in the email.

[97] I do not know what was discussed between the parties prior to them agreeing to the postponement, nor does the joint practice note cast any light on the events. What is undisputed is that defendant delivered an expert summary that was substantially out of time, which may well

have caused the plaintiff to agree to postpone the matter. Therefore it is, in my view, proper to follow Mr. Ströh's suggestion and to order that the wasted costs occasioned by the postponement on 27 July 2020 be costs in the cause.

CONCLUSION

[98] Mr. Ströh presented me with a lengthy draft order which details the exact costs that are to be paid. Most of the suggested terms of the order are matters that are resolved on taxation. I will therefore make the customary order in respect of costs.

[99] I consequently make the following order:

[99.1] Defendant is declared to be liable for payment of 100% of the proven or agreed damages resulting from the brain injury sustained by D on 18 July 2010 and the consequent cerebral palsy from which she suffers.

[99.2] Defendant shall pay the taxed or agreed costs of the action thus far on the High Court scale, including the costs of senior and junior counsel and the reasonable taxable preparation, qualification, travelling and reservation fees, if any, of the following experts:

[99.2.1] Dr. L Murray (obstetrician and gynaecologist);

[99.2.2] Prof. J Smith (neonatologist);

[99.2.3] **Dr. B Alheit (radiologist);**

[99.2.4] **Dr D Pearce (paediatric neurologist).**

[99.3] **The wasted costs occasioned by the postponement of the matter on 27 July 2020 are costs in the cause.**

[99.4] **In the event that the costs are not paid within 30 days of taxation or settlement, then interest may be levied thereon at the applicable *mora* rate.**

[99.4] The question of quantum is postponed *sine die.*

JJC Swanepoel

ACTING JUDGE OF THE HIGH COURT

GAUTENG DIVISION OF THE HIGH COURT, PRETORIA

Electronically submitted therefore unsigned

Delivered: This judgement was prepared and authored by the Judge whose name is reflected and is handed down electronically by circulation to the Parties/their legal representatives by email and by uploading it to the electronic file of this matter on CaseLines. The date for hand-down is deemed to be 8 March 2021.

PLAINTIFF'S COUNSEL:	**Adv. J. Ströh SC**
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DEFENDANT'S ATTORNEYS:	**The State Attorney**
	Mr. D. Olwage
HEARD ON:	**15 – 18 February 2021**
	25 February 2021
JUDGMENT ON:	**8 March 2021**