THE SUPREME COURT OF APPEAL OF SOUTH AFRICA

JUDGMENT

Reportable

Case No: 585/2018

In the matter between:

AN on behalf of EN **APPELLANT**

and

MEMBER OF THE EXECUTIVE COUNCIL FOR HEALTH, EASTERN CAPE **RESPONDENT**

Neutral citation: *AN v MEC for Health, Eastern Cape* (585/2018) [2019] ZASCA 102 (15 August 2019)

Coram: Cachalia, Tshiqi and Molemela JJA and Gorven and Weiner AJJA

Heard: 22 May 2019

Delivered: 15 August 2019

Summary: Delict – Medical negligence – failure to monitor the appellant and foetus during labour – whether negligence of hospital staff was causally connected to the child's brain damage – factual causation not established – appeal dismissed.

ORDER

On appeal from: Eastern Cape Local Division of the High Court, Mthatha (Dawood J sitting as court of first instance):
The appeal is dismissed.

JUDGMENT

Gorven AJA (Cachalia and Tshiqi JJA and Weiner AJA concurring):

[1] There is one central issue in this appeal. A baby was born with brain damage caused during labour. Did the negligent failure of the staff at All Saints Hospital, Engcobo, (the hospital) to properly monitor the mother and foetus during delivery cause that brain damage? In other words, as a matter of fact, did the harm result from the negligence?

[2] The mother in question is the appellant and represents her minor child, the baby. She claimed damages arising from the brain damage. The respondent is responsible in law if such loss was caused by the negligent conduct of the hospital staff. The matter was tried in the Mthatha High Court before Dawood J. She held that the appellant did not prove that the negligent failure to monitor the mother and foetus had caused the damage. She dismissed the appellant's claim but made no order as to costs. This appeal is with her leave.

[3] It is worth briefly sketching the legal landscape governing such a claim. '[I]n order to be liable for the loss of someone else, the act or omission of the defendant must have been wrongful and negligent and have caused the loss.'[1]

Wrongfulness involves the breach of a legal duty. The legal duty in the present matter arose when the mother was admitted to the hospital in labour. The staff assumed a duty to care for mother and fetus during the birth process without negligence, in other words, as would reasonable staff in their position. More particularly, they had a duty to monitor the condition of mother and foetus and act appropriately on the results. They negligently failed to do so, in breach of that legal duty. Their conduct was thus wrongful. But this, in and of itself, has never been sufficient to found delictual liability. 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.[2]

[4] 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.[3] 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. In heads of argument and initially before us, counsel for the appellant sought to rely on what were termed the majority and minority judgments in *Lee v Minister for Correctional Services*.[4]

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[1] *Telematrix (Pty) Ltd t/a Matrix Vehicle Tracking v ASASA* 2006 (1) SA 461 (SCA); [2006] 1 All SA 6; [2005] ZASCA 73 para 12.
[2] *Mashongwa v Passenger Rail Agency of South Africa* 2016 (3) SA 528 (CC); 2016 (2) BCLR 204; [2015] ZACC 36 para 64.
[3] *Silva's Fishing Corporation (Pty) Ltd v Maweza* 1957 (2) SA 256 (A); [1957] 2 All SA 313 at 264A-B.
[4] *Lee v Minister for Correctional Services* 2013 (2) SA 144 (CC); 2013 (1) SACR 213; 2013 (2) BCLR 129; [2012] ZACC 30.

[5] *Lee* concerned an inadequate system to monitor and isolate offenders who were in the infectious stage of tuberculosis in a correctional facility. It was accepted that the plaintiff probably became infected whilst incarcerated. The causation question was whether this inadequate system could be said to have caused the tuberculosis. This court held that the case had not been made out. In the Constitutional Court, Nkabinde J, who wrote the majority judgment, summarised the approach to factual causation taken in this court in that matter:[5]

'The Supreme Court of Appeal judgment . . . non-suited Mr Lee on the basis that he failed to prove that reasonable systemic adequacy would have "altogether eliminated" the risk of contagion, that he does not know the source of his infection and that had he known the source it is possible that he might have been able to establish a causal link between his infection and the specific negligent conduct on the part of the responsible authorities.'[6]

She criticised this approach:

'The implication of that kind of inexorable logic is that factual causation under our law can never be proved *where the specific incident or source of infection cannot be identified*. This means that even wrongful and negligent conduct of correctional facility authorities can by no means, in those instances, lead to delictual liability.'[7] (my emphasis)

She went on to hold that the 'but-for' test should be more flexibly applied.

[6] There has been some debate as to whether *Lee* changed the law on factual causation. Malcolm Wallis[8] convincingly argues that *Lee* did not do so. Professor Anton Fagan[9] compared what he says are the common law elements with those he says were applied in *Lee* and comes to the opposite conclusion. Such reasoned disagreement suggests that some of the reasoning in *Lee* muddied the waters of factual causation. The majority certainly highlighted difficulties which might

[5] *Minister for Correctional Services v Lee* 2012 (3) SA 617 (SCA); 2012 (1) SACR 492; [2012] 2 All SA 586; [2012] ZASCA 23.
[6] *Lee* para 42.
[7] *Lee* para 63.
[8] MJD Wallis 'Revel without a cause – Delictual liability after Lee?' (2019) *SALJ* 165.
[9] A Fagan *Undoing Delict: The South African Law of Delict under the Constitution* (2017) 216.

arise in the application of the but-for test, in particular where the wrongfulness arose from an omission:

'Although different theories have developed on causation, the one frequently employed by courts in determining factual causation, is the *conditio sine qua non* theory or but-for test. This test is not without problems, especially when determining whether a specific omission caused a certain consequence.'[10]

And later:

'However, in the case of an omission the but-for test requires that a hypothetical positive act be inserted in the particular set of facts, the so-called mental removal of the defendant's omission. This means that reasonable conduct of the defendant would be inserted into the set of facts.'[11]

[7] To the extent that it might be thought that *Lee* changed the test for factual causation, this was subsequently expressly disavowed by the Constitutional Court in *Mashongwa*:

'*Lee* never sought to replace the pre-existing approach to factual causation. It adopted an approach to causation premised on the flexibility that has always been recognised in the traditional approach. It is particularly apt where the harm that has ensued is closely connected to an omission of a defendant that carries the duty to prevent the harm. Regard being had to all the facts, the question is whether the harm would nevertheless have ensued, even if the omission had not occurred. However, where the traditional but-for test is adequate to establish a causal link it may not be necessary, as in the present case, to resort to the *Lee* test.'[12]

Mashongwa went on to apply the traditional but-for test.[13] This was in line with *Lee* which seemed to embrace a flexible approach to causation only 'where the specific incident or source of infection cannot be identified.'[14]

[8] However, I do not believe it is necessary to resolve that debate here. The present matter differs from *Lee*. It relates more closely to *Mashongwa*. The source

[10] *Lee* para 40.
[11] *Lee* para 41.
[12] *Mashongwa* para 65.
[13] *Mashongwa* para 66.
[14] *Lee* para 63.

of harm is known. It was an acute profound hypoxic ischaemic[15] insult caused by complete lack of oxygen to the brain for a sustained period. Based on *Lee*, the approach to be taken is 'that reasonable conduct of the defendant [must] be inserted into the set of facts.'[16] In the present matter, the question must be asked: Would the brain damage have been avoided if the hospital staff had properly monitored the mother and foetus and had acted appropriately on the results? If so, factual causation is established. If not, factual causation has not been established and one is left with only wrongful conduct without proof that it caused the harm suffered. It must be mentioned that, during argument, counsel for the appellant retreated from reliance on *Lee* and accepted that in the present matter the but-for test was applicable in order to determine factual causation.

[9] Having surveyed the legal terrain, attention must be directed to the facts of this matter. There is much which is common cause or not seriously contested. The mother was admitted to the hospital on 2 October 2013. This was her first birth and she had slightly exceeded full term. The delivery appeared uncomplicated. The apgar scores were low and alerted the staff to the possibility of damage. The baby sustained a brain injury during labour. The cause was an acute profound hypoxic ischaemic insult. Acute means sudden, as opposed to developing over a period of time. This occurred during labour. Ischaemia is a restriction in blood supply. Blood supplies oxygen to the brain. A continued restriction in blood supply leads to a lack of oxygen supply. Where this takes place, bradycardia occurs. This is a slowing of the foetal heart rate. Hypoxia results from a sustained reduction in the supply of oxygen to the brain. The injury to the baby is described as hypoxic-ischaemic encephalopathy. This is a form of neurological dysfunction. In this case, the baby developed cerebral palsy.

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[15] There are different ways of spelling the medical terms for foetus (fetus) ischaemic (ischemic) etc. I have used the English spelling in the text but have retained the original spelling when used in quotes.
[16] *Lee* para 41.

[10] In the present matter, it is common cause that the hospital staff did not properly monitor the labour. Accepted guidelines require such staff to take and record the foetal heart rate over certain periods. In well-equipped hospitals, the foetal heart rate is monitored with cardiotocographs (CTCs). The hospital was not equipped with these. In such situations, the monitoring is done by auscultation of the foetal heart.

[11] There are two phases of labour; the latent phase progressing to the active phase. The active phase has two stages. It was agreed by the experts that no damage was caused during the latent phase or the first stage of the active phase. It occurred during the second stage of the active phase. This began at 06h00 and lasted until delivery at 06h45. The last recorded foetal heart rate was taken at 06h00. The damage took place during a 45 minute period. The appellant's claim is that the failure to monitor during this period caused the damage.

[12] Doctor Murray, a specialist obstetrician called by the appellant, explained that decelerations of the heart rate occur during contractions. During this stage, the foetal heart rate should be taken before, during and after alternate contractions or every five minutes. This is important because, after each deceleration, the foetal heart rate should return to a normal baseline. Any delay in doing so signals worsening hypoxia. The appellant contended that warning signs of this nature would have been observed if monitoring had taken place. If so observed, the delivery would have been expedited. The case for the appellant was thus that: (a) there would have been warning signs before the sudden, damage inducing, event; and (b) delivery would have taken place prior to 06h45 and damage averted as a result. The case for the appellant was that the damage resulted because no warning signs were detected.

[13] Both parties accepted the report of Professor Andronikou. He interpreted a Magnetic Resonance Imaging (MRI) scan of the baby's brain. He concluded that the '[f]eatures are those in keeping with a global insult to the brain due to hypoxic ischaemic injury, of an acute-profound nature, occurring at term.' All of the experts concurred that this means that only the deep brain structures were damaged. The deep brain structures are also referred to as the grey matter, or core, of the brain. An acute profound hypoxic ischaemic event, such as in the present case, must be distinguished from a partial prolonged hypoxic ischaemic event. An acute profound event means a sudden, not progressive, event. A partial prolonged event causes damage to the white matter, or peripheral structures, of the brain.

[14] The mechanisms giving rise to these two types of brain damage are uncontroversial. Professor Van Toorn, Head of Paediatric Neurology at Tygerberg Childrens' Hospital and Stellenbosch University, was called by the appellant. He gave clear and uncontroverted evidence on this issue. During labour, the blood to the brain is supplied from the placenta along the umbilical cord (the cord). If there is an inadequate supply of oxygen, the brain shunts the limited blood from the peripheries to the deep grey matter. This is designed to protect the deep grey matter which is the most vulnerable matter due to its higher metabolic rate. When shunting takes place, damage occurs to the white matter of the brain. This means that if there is some blood supply, but it is inadequate, damage occurs to the white matter. If there is no blood supply at all, none is available to shunt to the deep grey matter. In that instance, only the grey matter will be damaged. The MRI scan shows only damage to the grey matter in the present case. No damage to white matter was evident.

[15] Based on this reasoning, all of the experts agreed that the damage described by Professor Andronikou was caused by a sudden, total, persistent lack of blood

supply to the brain. It was not caused by a reduced volume of blood supply. If this had been the case, damage to the white matter would have been evident.

[16] The sudden, total, persistent interruption to the blood supply is usually caused by a perinatal sentinel event. Such events include placental abruption, uterine rupture, umbilical cord prolapse, shoulder dystocia or maternal collapse. Each of these can be verified afterwards because they leave what was termed a footprint. Professor Smith, a neonatologist called by the appellant, was initially adamant that those listed above are the only phenomena which can be termed sentinel events. According to a study by Shankaran et al,[17] however, such events also include a compression of the cord which totally interrupts blood supply for a period long enough to cause damage (a total, persistent interruption). In Rennie & Rosenbloom,[18] it is said that the damage to the areas of the brain observed in the present matter is regarded 'as the imaging signature of hypoxic ischaemic sentinel events.' When referred to these studies and to his own previous use of the term for a total cord compression, Professor Smith finally conceded that a cord compression resulting in a sudden, total, persistent interruption of blood supply to the foetus could be referred to as a sentinel event.

[17] Whatever the nomenclature, the experts agreed that a sudden, sustained, total interruption to the blood supply caused by cord compression occurred in the present matter. For the sake of convenience, I shall refer to this as the sentinel event. This caused the damage. Unlike the other sentinel events listed above, a cord compression cannot be detected after the fact. This is because the cord does not remain compressed when the pressure on it is alleviated. It leaves no

[17] S Shankaran, A R Laptook, S A McDonald, S R Hintz, P D Barnes, A Das & R D Higgins 'Acute Perinatal Sentinel Events, Neonatal Brain Injury Pattern, and Outcome of Infants Undergoing a Trial of Hypothermia for Neonatal Hypoxic-Ischemic Encephalopathy' (2017) *The Journal of Pediatrics*180 275 at 276.
[18] J Rennie & L Rosenbloom 'How long have we got to get the baby out? A review of the effects of acute and profound intrapartum hypoxia and ischaemia' (2011) *The Obstetrician and Gynaecologist* 169 at 170.

indication that it was compressed. Any pressure on the cord ceases once a baby has been born.

[18] The experts also agreed that the only way to attempt to reduce the likelihood of damage when the blood supply has been completely disrupted is to expedite delivery of the baby. In the circumstances obtaining at the hospital, the only way this could have been done would have been by a vacuum extraction. This is because a caesarean section takes about 45 minutes to perform and the birth took place 45 minutes after this stage of labour commenced. Professor Buchmann, the respondent's expert obstetrician, testified that in a well-equipped Scottish hospital with well-trained staff, a vacuum extraction took an average of 19 minutes. He suggested that, in the hospital, this would take at least 20 minutes.

[19] The experts differed on two main issues. First, whether there would have been any warnings of an impending sustained total interruption to the blood supply. If so, the argument went, monitoring would have detected the warnings. The birth could then have been expedited and damage averted. Secondly, and if not, whether it would have been possible to prevent any damage to the brain when the sentinel event occurred.

[20] It was contended by the appellant's expert witnesses, and Professor Van Toorn in particular, that there would have been warnings of an impending sentinel event. He opined that intermittent interruption to the blood supply to the brain would have occurred during contractions. Blood supply would have been restored between contractions. He testified that these would have had a cumulative effect which ultimately led to the sentinel event. If monitoring had taken place according to the guidelines, therefore, a change in the foetal condition would have been observed. Such a change would be signalled by decelerations in the foetal

heart rate during contractions. The delivery could then have been expedited and the damage avoided.

[21] There are a number of difficulties with this opinion. First, it was entirely speculative. There was no evidence that there was intermittent interruption to the blood supply during contractions. Secondly, he conceded that such contractions would not in fact have totally shut off blood supply to the brain. He testified that during these periods 'the cord is not completely closed'. The interruptions would then have been partial. They would not have caused a total interruption to blood supply but a reduction. He was constrained to agree that, in those circumstances, the limited blood would have been shunted to the deep grey matter. The necessary corollary to this is that damage to the white matter would then have occurred. But there was none. His opinion that, on that basis, warnings would have been observed if proper monitoring had been carried out does not hold water.

[22] The second difficulty is that authoritative, peer-reviewed literature referred to by both parties does not support this opinion. In a number of studies, monitoring of the foetal heart did not support the case that there would probably have been prior warnings of a sentinel event. Okumura et al[19] conducted a study where, in some cases, the origin of the foetal bradycardia could not be determined. Monitoring actually indicated the well-being of these foetuses until sudden fall of the foetal heart rate. No warning was given. In another study, Murray et al[20] studied three groups of infants where CTGs were available. The third group, with normal CTGs on admission, suffered acute sentinel events without warning.

[19] A Okumura, F Hayakawa, T Kato, K Kuno & K Watanabe 'Bilateral basal ganglia-thalamic lesions subsequent to prolonged fetal bradycardia' (2000) 58 *Early Human Development* 111.
[20] D Murray, M N O'Riordan, R Horgan, G Boylan, J R Higgins, C A Ryan 'Fetal Heart Rate Patterns in Neonatal Hypoxic-Ischemic Encephalopathy: Relationship with Early Cerebral Activity and Neurodevelopmental Outcome' (2009) *American Journal of Perinatology* 26:8 605 at 608.

Pasternak & Gorey[21] concluded in their study that in 9 of their 11 patients, 'fetal monitoring was thought to be reassuring until the onset of the terminal bradycardia, supporting the premise that the hypoxic-ischaemic insult occurred at the end of labor and was acute and severe.' Finally, a standard text, *Williams Obstetrics*,[22] warns:

'There are several fallacious assumptions behind expectations of improved perinatal outcome with electronic monitoring. One assumption is that fetal distress is a slowly developing phenomenon and that electronic monitoring permits early detection of the compromised fetus.'

The appellant's witnesses were unable to point to any contrary literature. They appealed to the court a quo to accept what they said had been their experience. But this cannot be said to prevail in the face of compelling peer-reviewed literature.

[23] If there was in all probability no warning, the issue is whether, when the sentinel event occurred, there would have been sufficient time to avoid the damage by expediting the delivery. The obvious first factor in this enquiry is that counsel for the appellant candidly admitted that it could not be proved when the sentinel event occurred. Without being able to do so, it could not be said at what time monitoring would have alerted the staff to this event.

[24] The experts agreed that, where a sentinel event occurs, damage takes place within a short period of time. Professor Van Toorn testified that a total interruption to the blood supply gives rise to an 'insult'. If 'you have a severe insult and if that is sustained you end up with brain damage very rapidly'. Professor Smith agreed that, in the third group in Murray,[23] the average time to delivery after the observation of the sentinel event was 22 minutes. In other

[21] J F Pasternak & M T Gorey 'The Syndrome of Acute Near-Total Intrauterine Asphyxia in the Term Infant' *Pediatric Neurology* 18(5) 391 at 396.
[22] F G Cunningham, K J Lenovo, S L Bloom, C Y Spong, J S Dashe, B L Hoffman, B M Casey & J S Sheffield *Williams Obstetrics* 24 ed (2014) at 496.
[23] D Murray et al op cit.

words, the damage was done during this period. Professor Buchmann, the respondent's expert obstetrician, referred to Rennie[24] where damage began to accrue within 10 minutes of a sentinel event. I have already mentioned that the experts agreed that an expedited delivery in the hospital would have taken at least 20 minutes from when it was commenced. Before commencement, a doctor would have had to be called. The doctor would have had to assess the situation. This would have taken time. It is unrealistic to expect those things to have happened in less than 10 minutes. As already mentioned, the authors concluded that '[m]ore severe encephalopathy was associated with . . . acute sentinel events shortly before delivery'. Professor Buchmann testified without challenge that the sentinel event would probably have occurred in the 30 minutes prior to 06h45. His opinion is borne out by the literature referred to by both parties.

[25] As a result, it was not proved that there would have been sufficient time in which to deliver the baby so as to avoid damage. Counsel for the appellant conceded that, as a result, causation could not be determined. When it was pointed out that this in effect amounted to a concession that the appellant had not discharged the onus, he submitted that it was the negligence of the respondent which gave rise to this situation and the respondent should accordingly be held liable. But, as explained above, this is to conflate wrongfulness and causation. Both must be proved. When this was put to him, he did not persist in that submission. In any event, if the damage would probably have occurred within the median time mentioned in Murray of 22 minutes, no remedial action could have averted it. An expedited delivery could not have been performed inside that period. There is thus no basis on which the court a quo could find that damage could have been prevented if monitoring had taken place.

[24] J Rennie et al op cit.

[26] It bears mention that the second judgment, which I have read, concludes that the negligent failure of the hospital staff created risk along the lines mentioned in *Lee*. As I have said, it was conceded in argument that *Lee* did not apply. The matter was then argued on that basis without demur from any members of the court. In the circumstances I do not believe that it is appropriate to decide it as if *Lee* does apply. In any event, I have no difficulty with the proposition that the negligent conduct of the hospital staff in failing to monitor created a risk. It is, however, important to analyse the risk which was created to evaluate whether it can be said to have caused the brain damage suffered by the baby. The risk created was, first, that warnings of an impending sentinel event would not be detected and, secondly, that if a sentinel event did occur, this would not be detected within a reasonable time. As to the first, I have found that the appellant was correctly held not to have proved that there were any warnings of an impending sentinel event. This risk cannot, therefore, be said to have caused the brain damage. As to the second, for factual causation to result, it must be shown that if the sentinel event had been detected within a reasonable time, intervention within a reasonable time would probably have prevented the brain damage. This, too, I have concluded was not proved by the appellant. In other words, in this matter, it cannot be said that the creation of risk by the negligence of the hospital staff caused the brain damage suffered by the baby.

[27] The appellant sought to rely on the minority judgment of this court in *AM obo KM v MEC for Health, Eastern Cape*.[25] There, the same hospital was involved. A baby suffered cerebral palsy as a result of hypoxia. The majority in this court held that causation had not been established. The dissent was based on a feature of the MRI report, also provided by Professor Andronikou, which stated:

[25] *AM obo KM v MEC for Health, Eastern Cape* [2018] ZASCA 141.

'Features are those of a chronic evolution of a global insult to the brain due to hypoxic ischaemic injury, of the acute profound type, most likely occurring at term'.[26]

The minority held that the words 'chronic evolution', when qualifying an injury of the acute profound type meant that 'the acute, profound hypoxic ischaemia was not a sentinel event'. Instead, there was 'hypoxia and foetal distress which developed, undetected due to the lack of monitoring, over some time.' The minority thus held that there would have been forewarning prior to the onset of the sudden, total, interruption to the blood supply. If steps had been taken when the warnings appeared, damage would probably have been averted. The use of the words 'chronic evolution' are absent from the MRI report in the present matter. Also clear, in the present matter, is that there was no development of hypoxia and foetal distress over a period of time since no white matter was damaged. The case is distinguishable on the facts and the minority judgment does not assist the appellant.

[28] It is appropriate to say something about the prevalence of matters such as these. Far too often this court is confronted with serious and serial negligence in hospitals falling under the respondent. Whether or not the negligence can be said to have caused harm in the delictual sense, it is clear that studied neglect of standards has become pervasive in many such hospitals. Those reliant upon their services are receiving substandard care. During the hearing, this situation was put to counsel for the respondent. The response was that this sad state of affairs and the need for urgent remedial intervention had pertinently been brought to the attention of the relevant authorities. Despite this, such conduct does not appear to have abated significantly, if at all. The situation is to be deprecated. In the light of this, even though the respondent succeeded in resisting the appeal, counsel quite properly did not seek to advance any argument for a costs award against the appellant. No such award shall be made as a mark of displeasure. In addition, it

[26] *AM obo KM* para 8.

is directed that this judgment be forwarded to the respondent, and the National Minister under whom health services fall, in the hope that this situation will be urgently addressed.

[29] In the result, the following order is made:
The appeal is dismissed.

T R Gorven
Acting Judge of Appeal

Molemela JA

Introduction

[30] I have read the judgment penned by my brother Gorven AJA (the main judgment). I agree with his finding on wrongfulness, but respectfully disagree with his reasoning and conclusion in relation to causation. In my view the appeal should succeed. The reasons for my disagreement are set out hereunder.

[31] The main judgment alluded to some of the findings of this court in *AM obo KM v MEC for Health, Eastern Cape* for dismissing the appeal. For purposes of a proper perspective, I deem it appropriate to mention the following differences between that case and the present. First, in the former, it was accepted that at a certain critical point during labour, there was a reassuring foetal status, which was soon followed by lowering of the foetal heart rate. This led to the acceptance that the foetal heart rate would also have been normal prior thereto. In this case, the court a quo did not accept that there was a reassuring foetal heart rate at 06h00.

The significance of this finding is that, unlike in *AM obo KM v MEC*, in the present case, there was simply no evidence of a reassuring foetal status.

[32] Second, in *AM obo KM*, the mother of the baby did not testify, nor did any of the hospital staff. In this matter, the appellant, who is the mother of the baby on behalf of whom the claim was made, testified. Her evidence was undisputed. The extent of the negligence of the hospital staff is evident from her unchallenged testimony. Her evidence thus formed a solid factual foundation to the opinions of the expert witnesses who testified on her behalf. Lastly, and perhaps curiously, in *AM obo KM*, despite the insult leading to hypoxic-ischaemic encephalopathy (HIE) having been described as the 'acute profound' type, Prof Buchmann accepted that there was no 'sentinel event' suffered by the woman who was giving birth and further opined that the hypoxic event could have been 'related to uterine contractions'.[27] In the present case, although the brain injury was similar, Prof Buchmann was adamant that it was most likely that there was a sentinel event which happened without forewarning.

[33] Turning to the facts of this case, I am of the view that the evidence adduced on behalf of the appellant, the contents of the article written by the respondent's expert witness, Prof Buchmann, entitled '*Babies who die from labour-related intrapartum hypoxia: a confidential enquiry in South African public hospitals*',[28] as well as the concessions he (Prof Buchmann) made during the trial unquestionably prove both negligence and causation on a balance of probabilities.

Negligence

[27] *AM obo KM* para 63.
[28] E J Buchmann and R C Pattinson 'Babies who die from labour-related intrapartum hypoxia: a confidential enquiry in South African public hospitals' (2006) 36 *Tropical Doctor* 88.

[34] The test for establishing negligence is trite.[29] This test rests on two bases, namely, reasonable foreseeability and the reasonable preventability of damage.[30] What is or is not reasonably foreseeable in a particular case is a fact bound enquiry.[31] Of great significance is that negligence must be assessed in light of all the circumstances.[32]

[35] With specific reference to this matter, the standards that were applicable at the time of the appellant's admission are clearly spelt out in the National Maternal Guidelines (the guidelines) published in 2007. The foreword to the guidelines states that they were reviewed by many experts and were updated following a vast literature review. The guidelines are applicable to clinics, community health centres and district hospitals in South Africa. What is abundantly clear from these guidelines is that all women who are in labour must be monitored closely. As regards the establishment of the guidelines, Prof Buchmann's article states as follows:

'So to standardise care, we produced guidelines as a multidisciplinary group with obstetricians, anaesthetists, midwives, nurses, public health specialists and Department of Health officials *to get a consensus of what is feasible and best in South Africa with all its constraints, given the best evidence available and what we have in terms of the resources*.' (Emphasis added).

[36] Under the heading of 'management of the second stage of labour', the guidelines provide as follows:

'The second stage commences when the cervix reaches full dilatation (10 cm).

From the time that full dilatation of the cervix is first noted, up to 2 hours may pass before the mother starts to bear down. *Time can only be allowed for the head to descend onto the pelvic floor if fetal distress and cephalopelvic disproportion have been ruled out.* The bladder should be emptied, using a catheter if necessary. The observations of the first stage of labour should

[29] *Kruger v Coetzee* 1966 (2) SA 428 (A); [1966] All SA 490 (A).
[30] *Jacobs v Transnet Ltd t/a Metrorail* 2015 (1) SA 139 (SCA); [2014] ZASCA 113 para 6.
[31] *Pitzer v Eskom* [2012] ZASCA 44 para 24.
[32] *Kruger* at 430G.

continue. Efforts at bearing down are only encouraged when the fetal head starts to distend the perineum and the mother has an urge to push.

When the mother is ready to bear down:
• Always communicate clearly with the mother to gain co-operation
• Be supportive and encouraging
• Put the mother in a suitable position: propped up, sitting, squatting, kneeling, semi-Fowler's or wedged supine. Avoid the flat supine position as the uterus will compress the aorta and inferior vena cava
• Encourage pushing only during contractions
• Listen to the fetal heart rate between every second contraction
• Protect the perineum when the head crowns
• Gently suction the baby's mouth and nostrils while awaiting restitution and external rotation
• Record the times of onset of the second stage, onset of bearing down efforts and delivery.' (Emphasis added).

[37] Under the heading of 'emergencies during labour', the following is stated in relation to foetal distress:

'**FETAL DISTRESS**

This is suspected when the following signs are observed:
• Baseline fetal heart rate ≥160 beats per minute
• Baseline fetal heart rate <110 beats per minute
• Variability persistently <5 beats per minute on CTG, in the absence of sedating drugs
• Late decelerations of the fetal heart rate

Management of fetal distress

1. Explain the problem to the mother
2. Lie the mother in a left lateral position
3. Give oxygen by face mask at 6 L/minute
4. Start an intravenous infusion of Ringer-Lactate to run at 240 mL/hour
5. Do a vaginal examination for cervical dilatation and to exclude cord prolapse:

- If vaginal delivery is imminent (cervix fully dilated), deliver immediately, by vacuum extraction if necessary

- If vaginal delivery is not imminent, give hexoprenaline 10 micrograms IV and prepare for immediate caesarean section. Arrange urgent transfer from a community health centre to hospital.'

[38] It is trite that the facts on which expert witnesses express an opinion must be capable of being reconciled with all other evidence of the case.[33] The evidence of the appellant is therefore an important consideration. The salient aspects of the appellant's testimony are set out here and in the paragraphs that follow. She testified that after her admission to the hospital, she was taken to the labour ward. The nurse who attended to her performed a few vaginal examinations but did not monitor the foetal heart rate at any stage. During the night, she experienced excruciating pain that rendered her unable to walk, as a result of which she had to crawl whenever she had to go to the toilet. As there was no staff member at the nurse's station closest to her ward, she received assistance from fellow patients whenever she had to go to the toilet. According to her evidence, at some point she requested that a caesarean section be performed, seemingly because of the severe pain she was experiencing. The attendant nurse dismissed her request. At 06h00, a vaginal examination was done, after which the appellant was moved to the delivery room. This examination, too, was not accompanied by the monitoring of the foetal heart rate.

[39] According to the appellant, after examining her, the nurse told her that the baby was about to make its arrival and instructed her to start pushing. The nurse in question then left the ward and never returned. The appellant was left unattended until a member of the cleaning staff, who happened to be passing by, raised alarm after noticing that the baby's head was partially out but seemed stuck in the vaginal opening. In response to the cleaning lady's call for help, a nurse came to the scene and delivered the baby. It is common cause that the appellant's

[33] *Bee v Road Accident Fund* 2018 (4) SA 366 (SCA); [2018] ZASCA 52.

baby was delivered at 06h45 by the nurse who had just reported for the 07h00-19h00 shift.

[40] Dr Murray, an expert witness who testified on behalf of the appellant, is a senior specialist in obstetrics and gynaecology at Tygerberg Hospital and a lecturer at the Department of Obstetrics and Gynaecology at Stellenbosch University. She manages the labour ward and is a consultant that covers obstetrics wards and obstetric clinics. Her *curriculum vitae* further states that she is involved in collecting data on the underlying causes of foetal stillbirths and neonatal deaths, HIE audits and babies that may have come to harm during labour. Dr Murray gave crucial evidence relating to the paucity of monitoring during the appellant's latent and active stages of labour. She stated that it has been established that some babies do not tolerate or cope with labour, which is why a woman's labour must be monitored. The foetal heart rate must be monitored throughout labour, regardless of the instrument used. The correct manner of assessing the foetal heart rate is by monitoring and recording its reading before, during and after contractions. If there were merely random measurements of the foetal heart rate that were not timed to contractions, no reliable information would be gained as to whether the foetus was coping with labour. Thus, warning signs would not be detected and it would not be possible to intervene.

[41] Dr Murray further testified that if the uterine contractions were very strong and presented too much hypoxic stress to allow the foetus to stay oxygenated, the foetus would normally show signs of hypoxia, which would manifest themselves in a deceleration and slow recovery of the heart rate. A delay in returning to the normal baseline would signal worsening hypoxia and point to foetal distress. She pointed out that when it comes to labour, every minute counts. Thus, even in the face of a sentinel event, attempts are made to mitigate the risk of harm to both the mother and baby. Where there was a cord compression or cord prolapse, the

delivery of the baby would have to be expedited. Further, the mother would have had to be turned to her side to relieve the pressure on the umbilical cord. Under such circumstances, a caesarean section is recommended. Where a caesarean section was the appropriate intervention, an imminent delivery could be delayed by turning the mother to her side. However, if the foetal head had already engaged in the pelvis, a vacuum extraction or forceps delivery would have to be performed. It is of significance that under cross-examination, Prof Buchmann admitted that Dr Murray's evidence pertaining to the manoeuvres that are performed to try and reduce hypoxic ischemic damage constituted good practice.

[42] I pause to mention that the partogram signed by the night nurse reveals that the appellant was given intravenous medication referred to as Ringers Lactate at 06h00. According to the maternal guidelines, Ringers Lactate is administered as part of the management of foetal distress. Under the discussion on the management of foetal distress, the guidelines state that if the cervix is fully dilated, the baby must be delivered *immediately* by vacuum extraction if necessary. Notably, the clinical notes made by the same nurse who noted that Ringers Lactate was administered also recorded that at 06h00, the appellant was experiencing 'strong contractions' and her uterus was fully dilated. The respondent has not provided any reasons explaining why not even an attempt was made to deliver the baby at 06h00. What is plain is that despite the existence of foetal distress, the appellant was left unattended at the most critical stage of labour and this state of affairs persisted for approximately 45 minutes. The following extract from Prof Buchmann's own article sounds eerily prophetic when consideration is paid to the facts of this case. He stated that '[w]omen who are pushing in the second stage of labour should not be left alone, and fetal heart auscultation must be done after each contraction to confirm return to the baseline.

This will allow early detection of fetal bradycardia, so that appropriate action can be taken.'[34] (Emphasis added.)

[43] Having noted the appellant's evidence pertaining to her labour, Dr Murray's conclusion was that it was highly probable that the hospital staff had, due to inadequate monitoring of the appellant's labour, failed to notice that the foetus was not coping and therefore failed to take the remedial steps set out in the guidelines. This substandard care ultimately led to the brain injury suffered by the baby. Dr Murray's evidence is a very important piece of the puzzle in relation to the appellant's evidence.

[44] The guidelines corroborate Dr Murray's unchallenged evidence regarding the need for proper monitoring. Prof Buchmann's own article underscores the importance of monitoring a woman who is in labour. His conclusion is that HIE-related deaths can be prevented if there 'is close and careful monitoring of all women in labour with particular attention to detail in fetal heart-rate monitoring.'[35] The following extract from the article authored by Prof Buchmann is of significance with regards to the monitoring of a woman in labour:

'*The most striking finding in this study was the failure in most of these deaths, to detect signs of fetal distress*. It is likely that these babies would have shown some evidence of intrapartum hypoxia during labour. Late decelerations, the hallmark of fetal distress, were detected in just over one quarter of these cases…. It appears that there is a serious deficiency in intrapartum fetal monitoring in the hospitals studied, and probably in most other state hospitals in South Africa. It seems likely that insufficient time and care is taken with auscultation, and that the early signs of fetal hypoxia – fetal heart decelerations – are frequently missed.

. . .

Labour-related intrapartum hypoxia is common *and avoidable* cause of perinatal death in South Africa, and the majority of these deaths occur in low-risk situations where labour appears to be

[34] Buchmann and Pattinson at 10.
[35] Ibid.

normal. The overwhelming problem seems to be failure to detect evidence of fetal distress. To prevent these unnecessary deaths, the emphasis in the labour ward care should be close and careful monitoring of all women in labour, *with particular attention to detail in fetal heart rate monitoring.*'[36] (Emphasis added.)

[45] The appellant's unchallenged evidence revealed that the monitoring of her labour was hopelessly inadequate. The most crucial part of the monitoring, namely the assessment of the foetal heart rate against the uterine contractions, fell far short of the applicable standards. On the odd occasion on which the heart rate was recorded in the partogram, it was not done in accordance with the guidelines as it failed to reflect the foetal heart rate before, during and after contractions. The nurse who recorded that the foetal heart rate was normal at 06h00 did not testify, which left the evidence of the appellant relating to the failure to monitor the foetal heart rate, undisputed. Notably, the entry in the partogram did not reflect any measurements of the foetal heart rate taken during, before or after uterine contractions. This, in my view, is a clear indication that the foetal heart rate was not monitored. On this aspect, the court a quo found, correctly in my view, that the recording that the foetal heart rate was normal at 06h00 could not be accepted. As stated before, the court a quo's finding on that aspect was not attacked by the respondent on appeal. The main judgment's finding that the last recorded foetal heart rate was taken at 06h00 unfortunately fails to take that finding into account. In my view, this finding impacts on the respondent's case as it puts paid to Prof Buchmann's theory that it was unlikely that there were any decelerations of the foetal heart rate before 06h00. It follows that once that theory is discarded, his reliance on it as a basis for refuting the probability of prior warnings of foetal distress must follow suit.

[36] Ibid.

[46] The evidence shows that the nurses who attended to the appellant during labour are nursing sisters. Despite that, the manner in which they dispensed medical care to the appellant during the most critical part of labour fell below the standard of reasonably competent nursing sisters. That a nursing sister in their position would have foreseen harm is unquestionable. A nursing sister in their position would undoubtedly have taken steps to prevent harm by expediting the delivery of the appellant's baby. Given that the hospital in question falls under the category of level 1, it can be accepted that a doctor was present in the maternity ward at all material times of the appellant's delivery. Thus, it can be accepted that had there been any difficulties that the attendant nurses could not handle, they would have been escalated to a doctor on duty. It is clear from the evidence that at no stage was the assistance of a doctor sought. Having considered the evidence in totality, I am satisfied that all the grounds of negligence pleaded by the appellant have been proven on a balance of probabilities.

Causation

[47] I now turn to the contentious issue of causation. In the context of this case, the question is whether there was a causal link between the failure of the hospital staff to monitor and manage the appellant's labour in accordance with the stipulations of the guidelines, on the one hand, and the baby's brain damage which led to acute cerebral palsy, on the other.[37] In my view, there is. Indeed, the fact that the medical staff's conduct was wrongful and negligent does not necessarily resolve the question concerning whether liability should be imputed to it, thereby rendering the respondent vicariously liable. A successful delictual claim entails the proof of a causal link between a defendant's actions or omissions and the harm suffered by a plaintiff.[38]

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[37] *Mashongwa* para 63.
[38] *International Shipping Co (Pty) Ltd v Bentley* 1990 (1) SA 680 (A) at 700F-I.

[48] In *Minister of Safety and Security v Van Duivenboden*,[39] this court stressed that a plaintiff is not required to establish the causal link with certainty, but only to establish that the wrongful conduct was probably a cause of the loss, which calls for a sensible retrospective analysis of what would probably have occurred, based upon the evidence and what can be expected to occur in the ordinary course of human experience. In *Minister of Finance & others v Gore NO*[40] this court aptly held that the application of the 'but-for' test is not based on mathematics, pure science or philosophy. Rather, it is a matter of common sense, based on the practical way in which the ordinary person's mind works against the background of everyday life experiences. The flexible approach reflected in the above judgments was adopted by the Constitutional Court in *Lee*.

[49] The issue of causation recently received attention in the case of *Mashongwa v PRASA*. In *Mashongwa*, the Constitutional Court pointed out that *Lee* never sought to replace the pre-existing approach to factual causation, rather, it adopted an approach to causation premised on the flexibility that has always been recognised in the traditional approach[41] as reflected in the authorities referred to above. In re-stating the 'but-for' test in *Mashongwa*,[42] the Constitutional Court settled the law on this aspect. It pointed out that the imputation of liability to the wrongdoer depends on whether the harmful conduct is either too remotely or sufficiently closely connected to the harm caused. It emphasised that where the traditional but-for test is adequate to establish a causal link, it may not be necessary to resort to the *Lee* test. It is the facts of the case that will dictate which test is more appropriate. That being the case, it follows that a

[39] *Minister of Safety and Security v Van Duivenboden* 2002 (6) SA 431 (SCA) para 25.

[40] *Minister of Finance & others v Gore NO* 2007 (1) SA 111 (SCA); [2007] 1 All SA 309; [2006] ZASCA 98 para 33.

[41] *Mashongwa* para 65.

[42] *Mashongwa* para 68.

wrong concession by counsel regarding the test that is to be applied will not be binding on the court.[43]

[50] Before canvassing all the evidence showing that the nurses' negligence probably caused the brain injury sustained by the baby, I deem it convenient to first address myself to the court a quo's evaluation of expert evidence. It is necessary to do so because I am of the view that its approach was flawed and thus led to a wrong conclusion regarding causation. I will therefore briefly discuss the principles applicable to the evaluation of expert evidence.

[51] The correct approach to the evaluation of expert evidence was authoritatively laid down by this court in *Michael & another v Linksfield Park Clinic (Pty) Ltd & another*,[44] which endorsed the approach followed by the House of Lords in *Bolitho v City and Hackney Health Authority*.[45] This court pointed out that is required in the evaluation of expert evidence is to determine whether the opinions advanced by the experts are founded on logical reasoning and, if so, to what extent. If the court concludes that the opinion is one that can reasonably be held on the basis of the facts and the chain of reasoning of the expert, the threshold will be satisfied.[46]

[52] Prof van Toorn, a paediatric neurologist, supports Dr Murray's conclusion as to the probable cause of the baby's injury. The court a quo expressed several misgivings about Prof van Toorn's evidence in relation to the proximate cause of the baby's injury. It rejected some of his evidence on the basis that it was not

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[43] See *Matatiele Municipality v President of RSA* 2006 (5) SA 47 (CC); 2006 (5) BCLR 622 (CC); [2006] ZACC 2 para 66.
[44] *Michael & another v Linksfield Park Clinic (Pty) Ltd & another* 2001 (3) SA 1188 (SCA); [2002] 1 All SA 384; [2001] ZASCA 12 para 36.
[45] *Bolitho v City and Hackney Health Authority* [1998] AC 232; [1997] UKHL 46; [1997] 4 All ER 771; [1997] 3 WLR 1151 at 241-242. Also see *Daubert v Merrell Dow Pharmaceuticals Inc* 509 US 579 (1993).
[46] See *Imperial Marine Company v Motor Vessel Pasquale della Gatta & another; Imperial Marine Company v Motor Vessel Filippo Lembo & another* 2012 (1) SA 58 (SCA); [2012] 1 All SA 491; [2011] ZASCA 131 para 26.

documented in peer-reviewed publications. Further criticism of his evidence is canvassed in the main judgment. As I demonstrate below, that criticism is, in my view, unwarranted.

[53] Logically, there was nothing wrong with the evidence of Prof van Toorn pertaining to the pattern of brain injuries and the impact of intermittent interruption of the supply of oxygenated blood to the brain resulting in hypoxia. Nor were there any flaws relating to his reliance on the studies on animal models as a basis for his thesis. That evidence is corroborated by the Rennie & Rosenbloom article,[47] alluded to in the main judgment, where it is stated, in relation to animal models of acute profound damage, that '[r]epeated short umbilical occlusions can also cause damage to the striatum (equivalent to the deep grey matter) in lambs and may be closer to the insult that often affects the human fetus'.

[54] In relation to humans, the same article states the following: '[t]hese data support the evidence provided by the primate studies regarding a worse outcome if an acute near-total insult is superimposed on a previous episode of partial hypoxia'.[48] In the article authored by Pasternak, which has already been alluded to in the main judgment, the following statement, made in relation to term infants who had sustained a brain injury from an acute near-total intrauterine hypoxic insult, lends credence to Prof van Toorn's evidence:

'. . . therefore we were unable to determine the precise duration of the bradycardia *or even be confident that these patients had not experienced a significant subacute insult before the terminal bradycardia* . . . We believe that the similarity of their clinical features and imaging studies to those of the other nine patients [who had sustained an acute and severe hypoxic

[47] Rennie and Rosenbloom at 171.
[48] Rennie and Rosenbloom at 173.

brain insult at the end of labour] supports the assumption that all 11 patients represent a homogenous population'. [49] (Emphasis added.)

[55] The excerpts from these published articles not only give a backdrop to Prof van Toorn's logical process of reasoning, but also serve to show that his reasoning was scientifically valid. They fortify the view he expressed in relation to intermittent occlusions and their impact. The undisputed evidence about the sequence of events during the appellant's labour fits in neatly with the conclusion reached by Prof van Toorn. This obviously has a bearing on the probabilities.

[56] There was a difference of opinion between Prof Buchmann about the occurrence of intermittent episodes of a diminished supply of oxygenated blood to the foetus and the impact thereof on the foetus. It is well-established that where there are conflicting opinions of experts in the field, the court's determination of negligence must depend on an analysis of the cogency of the underlying reasoning which led the experts to their conflicting opinions.[50] Prof van Toorn testified that a foetus is normally able to adjust or compensate for the periods of hypoxia caused by the normal contractions. This is known as the auto-regulation of the foetus. According to him, an extended exposure to such hypoxia could compromise the ability of a foetus to recover from those intermittent episodes. He emphasised the importance of making a distinction between an insult and an injury. According to him, not every insult equates to an injury. The reaction to the insults differs from baby to baby. Foetal reserve, which relates to the ability of the foetus to withstand insults, plays a significant role. He testified that if the foetus was exposed to prolonged periods of intermittent hypoxia, the auto-regulation of the foetus could fail due to the depletion of foetal reserves. If a woman was not properly monitored, the intermittent decelerations of the foetal

[49] Pasternak and Gorey at 396.
[50] *Buthelezi v Ndaba* 2013 (5) SA 437 (SCA); [2013] ZASCA 72 para 1.

heart rate would not be observed and consequently, no intervention would be made. In the absence of any interventions, those intermittent decelerations of the foetal heart rate would culminate in a final insult to the brain of the foetus. The extent of the insult would depend on its duration, extent and foetal reserves.

[57] Prof van Toorn's evidence on this aspect is bolstered by the study of animal models that is alluded to in the Rennie and Rosenbloom article.[51] This article acknowledges that in human cases of acute profound hypoxic ischaemia 'there is clear variability both in the fetal reserve and in the duration and degree of the insult'. In the book entitled *Neonatal Encephalopathy and Neurological Outcome*[52] it is acknowledged that '[d]ifferences exist in the fetal effects of hypoxic stresses associated with late and variable decelerations, and different fetuses may have differing thresholds for damaging degrees of metabolic academia'. Given the views expressed in these articles, the cogency of Prof van Toorn on this aspect survives scrutiny and is unassailable. The court a quo therefore erred in finding that there was no basis for Prof van Toorn's thesis. The following remarks made by this court in *Linksfield* are apposite:

'The court is not bound to absolve a defendant from liability for allegedly negligent medical treatment or diagnosis just because evidence of expert opinion, albeit genuinely held, is that the treatment or diagnosis in issue accorded with sound medical practice. The court must be satisfied that such opinion has a logical basis, in other words that the expert has considered comparative risks and benefits and has reached "a defensible conclusion". If a body of professional opinion overlooks an obvious risk which could have been guarded against it will not be reasonable, even if almost universally held.'[53]

[58] The court a quo similarly erred by disregarding Prof van Toorn's evidence pertaining to the 15 cases which supported his conclusions on the features of

[51] Rennie and Rosenbloom at 173.
[52] American College of Obstetrics and Gynecologists & American Academy of Pediatrics *Neonatal Encephalopathy and Neurological Outcome* 2ed (2014) at 90.
[53] *Linksfield* para 37.

injuries sustained as a result of intermittent episodes of hypoxia culminating in an acute profound injury. The court disregarded that evidence on the basis that his thesis had not been documented in peer-reviewed articles. The court a quo failed to appreciate that expert witnesses are not confined to express views based on literature alone. Experts are required to lay a factual basis for their conclusions.[54] Prof van Toorn's evidence on those 15 cases is part of his own practical as a paediatric neurologist and ought to have been considered.[55] Inexplicably, while disregarding Prof van Toorn's evidence as to his practical experience, the court a quo seemingly accepted Prof Buchmann's. I could not find any basis for this disparity.

[59] It is trite is that a court may not rely on the opinion of an expert whose evidence is stretching beyond the limits of his or her own field of expertise. Prof Buchmann conceded that he had no expertise on certain fields of radiology that fell within Prof van Toorn's expertise as a paediatric neurosurgeon. Despite this concession, the court a quo remarked that '[p]rofessor Buchman quite eloquently satisfied this court that he was sufficiently qualified to give expert opinion on areas that partially infringed upon the expertise of the paediatric neurosurgeon'. This remark, is in my view misplaced.

[60] Moreover, the court a quo failed to take note of a clear inaccuracy in Prof Buchmann's evidence when he erroneously stated that Dr Andronikou's report made reference to injuries to the basal ganglia. The court a quo thus erred when it found that the MRI depiction and the radiologist's report and description of the injury supported Prof Buchmann's views. The following exchange between the

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[54] *Bee v RAF* supra para 22.
[55] See *Schneider N.O. & others v AA & others* 2010 (5) SA 203 (WCC); [2010] 3 All SA 332; [2010] ZAWCHC 3 at 203 & 211.

appellant's counsel and Prof Buchmann, relating to the MRI features canvassed in Pasternak's article is apposite:

'MR WESSELS: Yes, well let's look at what the imaging shows. The first one CT and that's all. Then we have MRI … (intervention).

WITNESS: No, the … (intervention).

MR WESSELS: Defuse(?) T2 abnormality basal ganglia and thalamus.

WITNESS: That's the CT on days 2 and 4, normal, which one often sees because the CT doesn't – isn't spec – sensitive to the changes. The MRI is better. *But this is radiology territory, I grant you that.* But the MRI is there, it's got abnormality in the basal ganglia and thalamus. That's the typical acute profound picture. The baby did badly, died on day 12.

. . .

MR WESSELS: Not all basal ganglia and thalamus are acute profound.

WITNESS: That's – no, that's the pattern. *Basal ganglia thalamus injuries are the signature of an acute profound insult and Prof Andronikou's report states that, gives the description and says these are in keeping with an acute profound. That's the MRI signature of an acute profound, basal ganglia thalamus, or also known as deep grey matter.'* (Emphasis added.)

[61] Despite having conceded that he had no expertise in radiology, Prof Buchmann persisted in expressing an opinion on the features of the injuries that would have resulted and the patterns that would be depicted on the MRI scan if Prof van Toorn's thesis were to be accepted as correct. During cross-examination, when it was pointed out to Prof Buchmann that Dr Andrinokou had not used the term 'basal ganglia' anywhere in his report, Prof Buchmann was adamant that the images depicted in the scan showed an injury to the basal ganglia. This is borne out by the following testimony with specific reference to the brain injuries sustained by the appellant's baby:

'MR WESSELS: *But that is not necessarily exclusive, if you have those two, then you have acute profound, you can have those injuries with other conditions as well.*

WITNESS: No, no, it's the watershed injuries that can go with other conditions such as infections and metabolic disorders. The acute profound is the signature – I mean, the basal ganglia thalamus injury, deep grey matter, is the signature of the acute profound insult. And

Prof Andronikou's report states it like that. *And there are really no other causes as far as I know. But yes, I'm now going to radiology territory.*

MR WESSELS: *Which is not your field.* Professor…"

MR WESSELS: Yes, M'Lady. (Pause). Bilateral symmetric abnormal high signal on T2/FLAIR the caudate nuclei, putamina, thalami, posterior limbs of the internal capsules focally, corona radiata and periorolantic regions, hippocampi, abnormal high signal … (indistinct) flare and volume loss noted focally involving the left cerebral hemisphere, the corpus collasum is thin around the … (indistinct) no significant ventricum(?) megelli(?). *That is what he found, there's no basal ganglia reference here.*

WITNESS: *No, the basal ganglia involved there, I will defer to radiologists on that,* but that is – *this is the basal ganglia thalamus injury.* That's the deep grey matter that he's referred to, but I'm not going to go and look in a textbook now and say to you this is a basal ganglia or that's not a basal ganglia.' (Emphasis added.)

[62] It is clear from the above exchange that even though Dr Andronikou did not find that there was an injury to the basal ganglia, Prof Buchmann remained adamant that the patterns depicted in the MRI scan showed that the basal ganglia had indeed been injured. It bears emphasis that even though Dr Andronikou categorised the baby's injury as one of an acute profound type, he did not find that a sentinel event had occurred. The injuries referred to in the article authored by Rennie & Rosenbloom, which were considered to be the 'imaging signature of hypoxic ischaemic sentinel event' included injuries to the basal ganglia. Given the fact that the basal ganglia are not listed in Dr Andronikou's report, the Rennie & Rosenbloom article cannot be considered as a basis for Prof Buchmann's conclusion that there was a sentinel event. Similarly, that article cannot be considered as a basis for disregarding Prof van Toorn's thesis that intermittent episodes of hypoxia can culminate in an injury of an acute profound type. As Prof Buchman is not an expert in radiology and paediatric neurology, his evidence regarding the features of the injuries and the neuroimaging that would be depicted on the MRI scans if Prof van Toorn's theory were to be accepted, amounted to speculation and was therefore of no value. There was no basis for accepting Prof

Buchmann's evidence as a justification for rejecting Prof van Toorn's thesis. The court a quo therefore erred in rejecting Prof van Toorn's plausible evidence.

[63] In this matter, it is undisputed that the appellant's ante-natal records showed that she was generally in good health and that the health of the foetus was not at risk. It is undisputed that she was in good health when she was admitted to All Saints hospital. Prof Smith's unchallenged evidence served to exclude the possibility of any congenital factors or genetic abnormalities that could have predisposed the baby to HIE. With regard to the possible interventions that could have been resorted to and whether such interventions were likely to avert the harm suffered by the baby, there is simply no evidence suggesting that on the day in question, the appellant's baby could not have been delivered within the agreed upon median of 22 minutes. The hospital falls into the category of level 1 hospitals that have properly trained and qualified staff (doctors and nurses), medical equipment, 24-hour labour and delivery service and a theatre to provide proper obstetric care. Prof Buchmann's evidence that there was insufficient time available for a vacuum-extraction delivery was not supported by any evidence. Similarly, his evidence that a forceps delivery was probably not possible because of lack of expertise by the nurses was not substantiated. There is no factual foundation supporting his conclusion that the injury was not preventable.

[64] A conclusion that the baby's injury was not preventable would perhaps have been tenable if there was evidence that some attempts were made, albeit unsuccessfully, to deliver the baby in accordance with the procedures stipulated in the guidelines. No such evidence was adduced. On the contrary, the evidence shows that, despite the nurse who delivered the baby being an advanced midwife who, in terms of the guidelines, could do a vacuum-extraction delivery, there was no attempt to expedite the baby's delivery in that fashion. No reasons were advanced for not expediting delivery. Clearly, the appellant and the foetus did not

receive appropriate medical care. It is a crying shame that a woman who was admitted to a hospital more than 12 hours before the delivery of her baby was neglected to the extent that the appellant and her baby were. I echo the following sentiments expressed by this court in *Premier of the Province of KwaZulu-Natal v Sonny and another*:[56]

'In our country poverty and a lack of literacy abound. Masses of our people attend public health facilities. Their lack of sophistication and the vulnerability that accompanies poverty are factors that cannot be ignored. ... What is required is a public health delivery system that recognises the dignity and rights of those who are compelled to use its facilities. It is that basic sensitivity that the Constitution demands.'

[65] Prof Buchmann postulated that despite the substandard medical care dispensed to the appellant, the baby's injury was not preventable because the MRI scan showed that the injury was of an acute profound pattern. This seems to suggest that (1) there can be no intervention during obstetric emergencies and (2) that substandard management of a patient's labour automatically becomes irrelevant once a hypoxic ischemic insult of an acute profound nature has been identified as a cause of the HIE. Taken to its logical conclusion, it in essence, exempts the hospital staff from exercising the requisite reasonable care and skill and absolves them from liability once an obstetric emergency occurs during labour. I find this proposition perplexing. None of the articles relied upon by the expert witnesses suggested that there is a stage when intervention becomes irrelevant because of an obstetric emergency. The fact that the guidelines lay down management procedures (interventions) in respect of 'emergencies during labour' renders that proposition nugatory. That proposition is, in any event, at odds with Prof Buchmann's own written article in which he concludes that HIE-related death *can be prevented* through close and careful monitoring. If that is accepted, then by parity of reason, brain injury must also be preventable. This

[56] *Premier of the Province of KwaZulu-Natal v Sonny and another* 2011 (3) SA 424 (SCA); [2011] ZASCA 6 para 33.

accords with the following proposition made in *Neonatal Encephalopathy and Neurological Outcome*: 'All women in labour should be monitored in an attempt *to prevent "asphyxial" injury* and intrapartum death'.[57] (Emphasis added).

[66] Even on the acceptance that there was a sentinel event in the form of a cord compression, this did not exempt the respondent's medical staff from intervening. Dr Murray testified that a cord compression shows itself by bearing down efforts which can be observed if there is adequate monitoring. All experts agreed that a severe cord compression is normally accompanied by marked decelerations of the foetal heart rate. The upshot is that if the appellant's labour had been properly monitored, these decelerations would have been observed and the intervention of expedited delivery would probably have been executed. Dr Murray emphasised that in an emergency situation, the promptness of hospital staff is key and every minute counts. She testified that even in catastrophic sentinel events such as the rupturing of the uterus or the placenta, babies have been saved by prompt intervention of hospital staff. That evidence was undisputed.

[67] As correctly stated in the court a quo's judgment, Prof Buchmann conceded that adequate monitoring would have been able to detect when the change in the foetal heart rate occurred. When the evidence of the appellant is juxtaposed with the evidence of all the experts, the most plausible inference is that there were indeed forewarnings, in the form of foetal decelerations, throughout the active phase of labour but these decelerations were not detected and recorded due to the inadequate monitoring. On the basis of the undisputed evidence, it is highly probable that a caesarean section would have been performed successfully before 06h00 if the appellant's labour had been properly monitored and managed.

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[57] American College of Obstetrics and Gynecologists & American Academy of Pediatrics *Neonatal Encephalopathy and Neurological Outcome* at 88.

[68] Further, the guidelines provide that 'time can only be allowed for the head to descend onto the pelvic floor if fetal distress and cephalopelvic disproportion have been ruled out'.[58] As stated before, the partogram noted that Ringers Lactate, which according to the guidelines is one of the interventions made when foetal distress is suspected, was being intravenously administered to the appellant. The evidence shows that the other procedures for the management of foetal distress were not followed and no attempt, whatsoever, was made to expedite delivery of the baby despite the attendant nurse having noted strong contractions and a full dilation of the cervix at 06h00. In my view, the acute profound insult that, according to Prof Buchmann, occurred in the last thirty minutes of labour would most probably have been averted if the provisions of the guidelines quoted in the afore-going paragraphs of this judgment had been followed. In other words, the injury would probably not have resulted, but for the negligence of the hospital staff.

[69] Nevertheless, I must say that it is of significance that Dr Buchmann conceded, albeit with some qualification, that the risk of sub-standard monitoring was that 'you will miss something which you could act upon and prevent a bad outcome'. On probabilities, this is exactly what happened in this case. By failing to properly monitor the appellant's labour in accordance with the provisions of the guidelines (the importance of which has been emphasized by all the expert witnesses including Prof Buchmann), the hospital staff created a situation that placed the foetus at a serious risk of HIE and its attendant *sequelae*. This risk precluded the timeous execution of any measures designed to avert injuries consequent upon suffering HIE. If that had been done, the acute profound injury that, according to Prof Buchmann, occurred in the last 30 minutes of the appellant's labour would have been averted. If a caesarean section was no longer

[58] See excerpt of the guidelines at para 36 of this judgment.

an option due to the foetal head having already been engaged, an expedited vacuum extraction delivery would probably have been successfully performed at 06h00.

[70] The study done by Yamada[59] and Prof Buchmann's article are both relevant to probabilities in relation to causation. Both studies conclude that sub-optimal intrapartum care was the probable cause of HIE in the majority of the studied cases. On probabilities, the brain injury sustained by the appellant's baby would not have occurred had the appellant and the foetus been properly monitored and managed by the hospital staff. I am satisfied that the evidence adduced on behalf of the appellant, bolstered by Prof Buchmann's concessions, points to a causal connection between the substandard care the appellant received from the hospital staff during labour and the acute profound hypoxic injury sustained by her baby. Put in the language used by the court in *Mashongwa*, there is a 'sufficiently close connection' between the negligence of the nurses that attended to the appellant and the damage that resulted. In the circumstances, causation has been established on a balance of probabilities.

Conclusion

[71] For all the reasons mentioned above, I would uphold the appeal with costs.

MB Molemela

Judge of Appeal

[59] T Yamada, K Cho, M Morikawa, T Yamada and H Minakami 'Intrapartum risk factors for neonatal encephalopathy leading to cerebral palsy in women without apparent sentinel events' (2015) 41(10) *Journal of Obstetrics and Gynaecology Research* 1 at 3.

APPEARANCES:

For the Appellant:	JJ Wessels SC Instructed by: Nonxuba Incorporated Attorneys, Rivonia Webbers, Bloemfontein
For the Respondent	PJ De Bruyn SC (with him M Rili) Instructed by: State Attorney, Mthatha State Attorney, Bloemfontein