



THE SUPREME COURT OF APPEAL OF SOUTH AFRICA
JUDGMENT

Not Reportable
Case No: 272/2022

In the matter between:

**MEMBER OF THE EXECUTIVE
COUNCIL OF HEALTH AND SOCIAL
DEVELOPMENT, GAUTENG
PROVINCIAL GOVERNMENT**

APPELLANT

and

**F B M
(obo L P M)**

RESPONDENT

Neutral Citation: *MEC of Health and Social Development of the Gauteng
Provincial Government v M (272/2022) [2024] ZASCA 21
(05 March 2024)*

Coram: DAMBUZA AP and SALDULKER, NICHOLLS, MABINDLA-
BOQWANA and GOOSEN JJA

Heard: 07 March 2023

Delivered: 05 March 2024

Summary: Delict – claim for medical negligence damages – minor born with brain injury sustained during birth – whether hospital staff were negligent – if so, whether such negligence caused the brain injury – evidence did not establish that the hospital staff were negligent.

ORDER

On appeal from: Gauteng Division of the High Court, Johannesburg (Wilson AJ, with Weiner and Mudau JJ concurring) sitting as a court of appeal:

1. The appeal is upheld with costs, including the costs of two counsel where so employed.
2. The order of the full court is set aside and substituted with the following:
‘The appeal is dismissed with costs, including the costs of two counsel where so employed.’

JUDGMENT

Dambuza AP and Nicholls JA (Saldulker, Mabindla-Boqwana, and Goosen JJA concurring):

[1] This appeal is against an order of the full court of the Gauteng Division of the High Court, Johannesburg (Wilson AJ, with Weiner and Mudau JJ concurring). That court overturned a judgment of the trial court of the same division (Molahlehi J) which held that the appellant, the Member of Executive Council for Health and Social Welfare for the Gauteng Provincial Government (MEC)¹, was liable for damages suffered by the respondent, Ms M’s child, L, as a result of a brain injury which it found to have been sustained during L’s birth.

¹ In April 2012 this Department was split into - a separate provincial department of health and a department of social development. However, in this judgment we continue to refer to the parties as they were in the pleadings, the trial court, and the full court.

[2] The primary questions in this appeal are whether the medical staff at Tshwane District Hospital were negligent in the care and treatment of Ms M, the mother of L, and whether such negligence caused the consequent medical condition from which L presently suffers. This appeal is with the leave of this Court.

[3] L was born at Tshwane District Hospital on 18 May 2010. His mother, Ms M arrived at the hospital in the early hours of the morning of that day as an ‘unbooked’ patient, with no ante-natal records. She was admitted at 01h45 in the latent stage of labour, with ruptured membranes. L was born at 15h10, on the same day, by natural delivery, with Apgar scores of 1 at one minute, 7 at five minutes (assisted through resuscitation), and 8 at ten minutes (assisted through resuscitation). At birth he was flaccid, acidotic and had to be placed on a ventilator.

[4] On 15 January 2014 Ms M instituted an action in the Gauteng Division of the High Court, claiming damages against the MEC for medical negligence on the part of the hospital’s staff. In the summons she alleged that L suffered a hypoxic-ischaemic encephalopathy (HIE or brain injury)² during birth as a result of negligence by the hospital staff. This resulted in L suffering cerebral palsy which continues to afflict him, she alleged.

[5] She pleaded various acts of negligence on the part of the hospital staff. In the main, her case was that her labour was unduly prolonged and that the hospital medical staff failed to take a decision at 13h10 to deliver the baby by caesarean section. The contention was that L suffered the brain injury due to perinatal

² A type of brain dysfunction (brain injury) that occurs when the brain experiences a decrease in oxygen or blood flow. It can occur before, during labour and delivery or after birth. <https://www.massgeneral.org> as of 4 February 2024.

asphyxia³ which caused him to sustain severe brain damage, resulting in cerebral palsy (CP), mental retardation and epilepsy.

[6] The MEC denied liability, pleading that there was no negligence on the part of the hospital staff. He pleaded that any duty of care owed to Ms M and the baby she was carrying was circumscribed by, and subject to the reasonable financial, human, and other resources available to the Department of Health to equip staff and maintain the hospital. It was asserted that the hospital rendered the best service it could to Ms M. However, in the event of the court finding that the monitoring of the foetus was insufficient, such deficiency was not causally connected to the cerebral palsy suffered by L.

[7] It was common ground that on her admission in the early hours of the morning, Ms M was connected to a cardiotocography (CTG) machine.⁴ At first, the CTG tracings (partogram) were ‘non-reassuring’. This was resolved by application of Ringers Lactate.⁵ The CTG machine was allowed to run for another 30 minutes and was described as ‘re-assuring with good variability’. Ms M was then monitored with a Doppler at 4h00, 6h00, 8h00 and 10h00. Over this period the foetal heart rate was recorded as being between 142 and 147 beats per minute,

³ ‘Perinatal asphyxia or birth asphyxia, results from an inadequate intake of oxygen by the baby during the birth process – before, during or just after birth. Decreased oxygen intake can result in chemical changes in the baby’s body that include hypoxemia, or low levels of oxygen in the blood, and acidosis, in which too much acid builds up in the blood.’ <https://www.hopkinsmedicine.org> as of 4 February 2024.

⁴ The CTG is a machine used to monitor the heartbeat of a foetus and the mother’s contractions during labour. It generates a printout on which traces depicting the baby’s heartbeat over time are recorded. The three indicators to look out for when reading CTG tracings are: first, the rate of the foetal heartbeat, second, the baseline variability, and third decelerations in the heartbeat. A foetal heartbeat of between 110-160 beats per minute (bpm) is considered normal. Baseline variability refers to the extent of variation between one heartbeat and the next. When the heart rate deviates from the normal baseline variability this can be a sign that the foetus has initiated defence mechanisms as a result of decreased levels of oxygen. The presence of variability suggests that the nervous system of the foetus is still undamaged. Deceleration occurs when the heart rate is reduced to less than 15 beats per minute. This may signal that the foetus is becoming hypoxic, or it may indicate something less sinister.

⁵ This is a ‘type of isotonic, crystalloid fluid further classified as balanced or buffered solution used for fluid replacement. The content of Ringer’s Lactate includes sodium, chloride, potassium, calcium and lactate in the form of sodium mixed into a solution with an osmolality of 273 mOsm/L and pH of about 6.5. See <https://ncbi.nlm.nih.gov>, (the National Library of Medicine of the United States of America) as of 28 February 2024.

thus falling within the normal range of 110 -160 beats per minute. The next CTG assessment was performed at 06h24. The partogram was again described as ‘re-assuring’. A handwritten note by the senior midwife Sister Motshwene recorded that the CTG assessment should be repeated after one hour. This was not done.

[8] Ms M went into the first stage of active labour at 7h30. Sometime thereafter she was seen by a doctor⁶ who referred to a ‘reassuring’ partogram. At 11h30, after a further foetal heart rate assessment, she was transferred to the labour ward. The foetal heart rate was again assessed with a Doppler at 12h00, 12h30 and 13h00. The next CTG assessment commenced at 12h40 and the machine ran continuously until approximately 15h00. A note at 13h30 recorded that the foetal heart rate was 160⁷ beats per minute with slight decelerations. A further written note was made to ‘report CTG to Sr Motshwene’. Foetal distress was diagnosed at 14h30 and Ms M was prepared for a caesarean section. However, she became fully dilated at 14h45, after being wheeled into theatre, and she delivered L naturally at 15h10.

[9] Ms M’s case was, in the main, constructed around two aspects of her baby’s birth. First, she contended that the CTG partogram showed that a decision should have been taken at 13h10 on the day of L’s birth, to have her deliver the baby by caesarean section. Aligned to this, was the contention that L’s foetal heart rate should have been monitored by a CTG machine for the whole period that she was at the hospital, or that Ms M should have been put back on the CTG machine at some stage before 12h40. Had that been done, irregularities in L’s heartbeat would have been observed sooner, and the decision to perform a caesarean section would have been taken prior to brain injury occurring.

⁶ The evidence was that the clinic did not employ specialist doctors but only general practitioners.

⁷ The actual note referred to 60 beats per minute but it is accepted that this was an error and it should have been 160 beats per minute.

[10] This contention was based on the evidence given by Ms M's obstetrician, Dr Murray, that if the CTG had been repeated prior to 12h40, an abnormal pattern would have been diagnosed earlier, and a plan made for a caesareansection to be performed before the foetus became acidotic. According to her, from 12h40 the traces depicted a pathological foetus, even though there was normal variability. From that time two atypical decelerations, and a baseline foetal heart rate of 160 bpm were present on the partogram. When these features persisted for the 30 minutes ending at 13h10, the foetus became pathological. The decision to perform an emergency caesarean-section should have been taken at that time, and the failure by the hospital staff to do so was sub-standard medical care, she opined.

[11] The second aspect on which Ms M relied was the contents of joint minutes prepared by medical experts, including radiologists Dr A Weinstein and Prof. Savvas Andronikou. The Magnetic Resonance Imaging (MRI) scan on which the radiologists based their opinion was done when L was 4 years and 5 months old. The radiologists agreed that the MRI demonstrated features in keeping with acute profound hypoxic ischemic injury. They also agreed that because the findings were isolated, they required clinical correlation. In their minute they recorded that they had observed a bilateral 'T2/flair'⁸ hyperintensity of the child's putamina,⁹ these being features of an acute profound hypoxic ischaemic injury. They agreed that the MRI scan demonstrated an 'acute profound HII [Hypoxic Ischaemic Injury] that occurred in a term brain at 37 weeks or [older] probably in the perinatal time period . . . '. They also agreed that the involvement of the basal ganglia in a HII, in general, indicates an acute profound event. In addition, they

⁸ This stands for 'T2-weighted Fluid-Attenuated Inversion Recovery', an area of high intensity on types of MRI scans of the brain of a human reflecting lesions produced largely by damaging of the myelin sheath surrounding neurons and axonal loss.

⁹ A paired structure that is part of the nuclei that form the basal ganglia – which is responsible for motor control and other functions of the brain.

agreed that the injury to L's brain was limited to the putamina and that the other deep grey structures as well as the hippocampi,¹⁰ the Peri-Rolandic¹¹ regions and cerebellar vermis had been spared. They stated that the neurological outcome should be assessed clinically, by obstetricians, for determination of a more accurate time and cause of injury.

[12] Dr Andronikou, the radiologist who testified for Ms M, was of the opinion that if the baby's heart had been monitored 24 hours a day by CTG, it might have been possible to pinpoint when the sentinel event occurred by observing the deteriorating foetal heart rate on the CTG. He persisted in the view that the assault on L's brain was acute profound, and likely to have been caused by hypoxia. The MRI imaging, he said, indicated that something severe had happened to the foetus, although it did not show the period when the injury occurred.

[13] Paediatric neurologists Drs D Pearce (for Ms M) and J Rademeyer (for the MEC) also prepared a joint minute intended 'to assist the court in respect of causation/origin of the timing of [L's] diagnosis and neurological disability'. They did a neurological examination on L on 13 April 2017 and 8 August 2017 respectively, and also considered the joint minute prepared by the radiologists. They agreed that L suffered from 'mixed cerebral palsy (predominantly dystonic¹²) with a superimposed right hemiplegia and gross motor functional classification scale 1V [which were] indicative [of the fact] that L's physical impairments severely restricted his movements'.

[14] The neurologists agreed that a diagnosis of early onset of HIE II (neonatal encephalopathy) could be made based on the history and the limited clinical

¹⁰ Complex brain structure embedded deep into the temporal lobe. <https://www.ncbi.nlm.nih.gov> > pmc

¹¹ Central lobe of the brain. . <https://www.ncbi.nlm.nih.gov> > pmc

¹² Involuntary muscle twitch.

records available. Whilst they agreed that foetal distress was evident in L more than 12 hours prior to delivery, they also resolved to defer to the obstetricians the question of appropriate management of that observation. They could not agree on whether there could be other causes for the cerebral palsy other than the HIE. Dr Pearce opined that because the MRI findings were not consistent with infection, congenital brain abnormalities, genetic and metabolic disorders, or cranial haemorrhage, the most probable cause of the injury to L's brain was intrapartum or peripartum hypoxia.

[15] Dr Rademeyer's opinion was that the mild involvement of basal ganglia observed was not consistent with a severe hypoxic event. She maintained that there was no evidence that other causes of HIE had been investigated and insisted that the MRI findings were not in keeping with peripartum¹³ hypoxia. However, she agreed in the joint minute that a diagnosis of HIE could be made from records showing partially compensated metabolic acidosis in L's blood that was taken more than an hour after delivery. She was also satisfied that there were no other identifiable causes of the injury to Baby L's brain injury. She still referred back to the MRI findings which, she said, were not in keeping with peripartum hypoxia.

[16] Dr Pearce saw L when he was 6 years 11 months. He accepted that the MRI scan showed that the damage to the brain was limited to the putamina only, which meant that the lesion was not extensive. He also admitted that the brain injury could have occurred after birth, but stated that because this was not what Ms M told her, and from all the available records, there was no evidence to suggest this. He agreed that the complete cause of L's cerebral palsy was uncertain.

¹³ 'Peripartum' means 'the period shortly before, during, and immediately after giving birth'. <https://profiles.umassmed.edu> as of 14 February 2024.

[17] The obstetricians, Drs Murray (for Ms M) and Archer (for the MEC) agreed in their joint minute that the cerebral palsy from which L was suffering, was likely caused by intrapartum hypoxia which occurred at an undefined time. They also agreed that although at some stage prior to 14h00 the CTG showed a pattern of recurrent decelerations, variability remained normal. Only from about 14h00 did the foetal heart rate rise and variability fall.

[18] In addition, the obstetricians agreed that although there were decelerations or signs of distress on the earliest partogram generated by the CTG, the foetus was ‘likely, overall in a good condition at that stage’ as indicated by its positive response to the administration of the Ringer’s Lactate.

[19] The trial court dismissed Ms M’s claim, having found that it could not conclude that the injury to L’s brain was sustained intrapartum, or that the hospital staff was negligent. With the leave of this Court the matter was appealed to the full court, which overturned the decision of the trial court and held that the MEC was 100% liable for any proven or agreed damages arising from L’s brain injury. The full court held that the trial court should have found that:

‘Ms M’s foetal condition on admission was good, but that the first CTG gave cause for concern; that continuous CTG – or at least two hourly – monitoring ought to have been implemented, but it was not; that the foetal condition deteriorated further at 13:30, to the extent that the senior midwife on duty would have taken immediate action had she been told about it; that the situation deteriorated rapidly after 14h30; that L was born hypoxic; and that L’s cerebral palsy probably resulted from a brain injury that occurred during labour.’

[20] There are a number reasons why the conclusion reached by the full court that the standard of care afforded to Ms M was sub-standard cannot be sustained. First, it is evident that the conclusion of the full court was based on the understanding that Dr Murray was of the opinion that the foetal condition became pathological at 06h24. Based on this understanding the full court reasoned that

there was ‘deterioration’ in the foetal condition between 07h00 and 12h40 in the early hours of the morning, which ‘rendered L’s brain injury foreseeable’. Therefore, CTG monitoring should have been continuous, and when the partogram was not reassuring at 13h10, there should have been prompt intervention to deliver the baby, the court found. It concluded that the failure to monitor with CTG constituted below par medical care.

[21] However, it was not Dr Murray’s evidence that the foetus became pathological at 06h40 and that its condition deteriorated thereafter. In fact, Dr Murray accepted that although the partogram had been non-reassuring when Ms M was first put on the CTG, it became reassuring for hours after the Ringer’s Lactate was administered. The conclusion by the full court that at 06h24 the decelerations demonstrated such degree of abnormality as to be regarded as requiring closer than standard care ignores the positive response to the administration of the Ringer’s Lactate.

[22] Secondly, the suggestion that CTG monitoring of L’s heart rate would have facilitated the detection and avoidance of a sentinel event is unsubstantiated. The radiologists, having observed the injury to the basal ganglia (albeit confined to the putamina), agreed that L sustained acute profound HII. They were unable to pinpoint when this occurred. Although Dr Rademeyer sought to suggest that that might not be so, she offered no clear or logical basis for such opinion. The evidence to the effect that L suffered an acute profound brain injury intrapartum was overwhelming. The other experts were consistent in their opinion in that regard.

[23] The approach adopted by this Court in determining whether there has been a breach of the legal duty to administer reasonable health care and skill in

circumstances such as these is to distinguish between an acute profound and a partial prolonged HII. In *NSS obo AS v MEC for Health, Eastern Cape Province*¹⁴ this court set out the distinction as follows:

‘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.’

[24] In *Member of the Executive Council for Health, Eastern Cape v Z M*¹⁵ this court said the following:

‘The significance of this conclusion is an important matter. It was explained, in the course of the trial, by the expert obstetrician and gynaecologist, Dr Buchmann, who testified on behalf of the appellant. He testified that there is a distinction between an intrapartum acute profound brain injury (‘an acute profound injury’) and an intrapartum prolonged partial brain injury (‘a prolonged partial injury’). An acute profound injury is severe, with total or near-total asphyxia (deficient supply of oxygen); it is of short duration, and sudden onset, and generally occurs 30 minutes before delivery. A prolonged partial injury is less severe, with partial asphyxia; it develops slowly over several hours; it is often preceded by a deteriorating foetal heart rate that gives a warning of developing hypoxia, that is, lack of oxygen. . . .’

[25] With reference to academic writings, this Court has held that authoritative peer-reviewed literature does not support the view that monitoring of the foetal heart by CTG provides prior warning of a sentinel event. In *AN v MEC for Health, Eastern Cape*¹⁶ this court held that:

‘. . . 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 conducted a study where, in some cases, the origin of the fetal bradycardia could not be determined. Monitoring actually indicated the well-being of these foetuses until sudden fall of the foetal heart rate. No

¹⁴ *NSS obo AS v MEC for Health, Eastern Cape Province* [2023] ZASCA 41 para 6. See further the authorities cited in that judgment.

¹⁵ *Member of the Executive Council for Health, Eastern Cape v Z M* ZASCA (576/2019) [2020] ZA4SCA 169 (14 December 2022) para4.

¹⁶ *AN v MEC for Health, Eastern Cape* [2019] ZASCA 102; [2019] 4 All SA 1 SCA paras 24 and 25.

warning was given. In another study, Murray et al¹⁷ studied three groups of infants where CTGs were available. The third group, with normal CTGs on admission, suffered acute sentinel events without warning. Pasternak & Gorey¹⁸ 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*,¹⁹ 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.’”²⁰

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’.

[26] In this case there was no evidence of a sentinel event. The contention that a sentinel event would have been detected and avoided if reasonable care had been taken is based on the reverse reasoning that because L suffers from CP there must have been a detectable and avoidable sentinel event during his birth. The courts have cautioned against commencing with an unfavourable outcome and working backwards in search of a cause. Hornbuckle J warned that with the benefit of the knowledge that there has been a neurologically unfavourable birth outcome, a plaintiff’s attorney ‘can take any foetal monitor strip and make a malpractice case out of it’.²¹

¹⁷ 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.

¹⁸ 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.

¹⁹ 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)496.

²⁰ Article in BJM Electronic Foetal Monitoring and our epidemic of Caesarean births (SEE CB 218-235)

²¹ Hornbuckle J, Vial A, etc see f/n 21 page 229.

[27] Similarly in *Goliath v MEC Health, Eastern Cape*²² this court cautioned that a doctor should not be held negligent simply because something went wrong. It cited with approval the remarks made by Lord Denning in *Hucks v Cole* [1968] 118 New LJ 469 (1993) that to hold a doctor negligent simply because something went wrong, would be to impermissibly reason backwards from effect to cause. If we accept, as we must, that the evidence overwhelmingly pointed to L having sustained an acute profound hypoxic ischaemic event which occurred intrapartum, then based on the evidence of Ms M'S own experts the injury sustained by L was not one that might have been reasonably foreseeable.

[28] During cross-examination Dr Murray was constrained to acknowledge that the CTG is not a perfect tool with which to monitor the foetal heart during labour because of its 'low specificity'. The substantial body of evidence that was tendered at the trial on academic writings regarding the value of CTG in averting or reducing HIE, is not conclusive on the issue. At best the evidence showed that on an interpretation of the partogram in terms of both the American College of Obstetricians and Gynaecologists (ACOG) and National Institute for Health and Care (NICE) clinical guidelines,²³ acidosis may be deduced in category III (ACOG) or pathological (NICE) partogram.

²² *Goliath v MEC Health Eastern Cape* 2015 (2) SA 102 (SCA) para 9.

²³ Dr Murray preferred the NICE guidelines which are evidence based health and care clinical guidelines recommended in the United Kingdom, while Dr Archer preferred the ACOG guidelines. Each one of them explained how a program is interpreted and managed under their preferred system, with Categories I, II, and III partogram on the ACOG grading comparable to normal, suspicious and pathological under the NICE guidelines. Category 1 traces are characterised by a baseline heartbeat of 110-160 per minute; moderate variability; no variable accelerations or decelerations. These are all strongly predicative of a normal fetal acid base. Category 11 traces are those that don't meet the criteria of Category 1. According to Dr Archer because they are not characterised by a lack of variability, they are not associated with foetal acidosis, and should not be associated with brain damage. They are non-reassuring and require some intervention such as moving the patient onto her side and administering fluids, but not necessarily delivery. Category III traces are characterised by recurrent late decelerations or recurrent variable decelerations with no variability. They are strongly predicative of acidosis. Dr Archer stated that it is accepted internationally that fetal brain damage will not occur until there is an abnormal acid base of a ph of less than 7b and a base excess of more than 12.

Under NICE guidelines Normal is where the fetal heart beat is between 110-160; variability is 5-25bpm; no decelerations; and accelerations are present. A trace is characterised as suspicious when there is one non-reassuring feature. It is pathological when there are two or more non-reassuring features present. (CB298-301)

[29] Dr Archer insisted that the 06h40 traces were only suspicious and not pathological, because variability was good, and that from 14h00 the traces were merely non-reassuring (category II on the ACOG grading system), because there was no total loss of variability. He explained that under ACOG, brain injury is likely to occur in category III CTG traces, which would be indicated in recurrent late decelerations with no variability. However, he readily accepted that if the baby had been born at 14h20 it was not likely to have been acidotic as variability was normal on the partogram until 14h30. But he was unwilling to speculate as to what happened thereafter. Her evidence suggests that the insult occurred within an hour of the delivery. However, the fact that the injury occurred during that period does not mean that it was necessarily attributable to negligence by the hospital staff.

[30] Dr Murray had initially asserted that under the NICE guidelines, at 13h10 the partogram traces signalled a pathological foetus. However, this stance changed once she saw the original partogram. She then sent a text message (SMS) to Dr Archer on 3 September 2017 advising that:

‘. . . based on the fact that we have seen the original ctgs, I need to amend some of my points where I comment on there being no rate. Also I am happy and raise no comment about the fact that they are category 2 traces until the last hour. The management thereof we need to discuss.’

[31] Having found that CTG monitoring would not have made a difference, it is not necessary to consider the contention by Ms M that the hospital staff were negligent in failing to adhere to the guidelines stipulated by the Department of Health when attending to her. In any event it was accepted that the decision to perform a caesarean section (14h30), and L’s delivery happened within one hour of each other as stipulated by the guidelines. Ms M’s case on negligence can

This is when the fetal heart is more than 180 beats per minute or less than 100; the variability is less than 5 or more than 25 for less than 90 minutes and that there are variable decelerations present for more than 50% of the contractions for less than 30 minutes or a single prolonged deceleration lasting more than 3 minutes.

therefore only be limited to the alleged failure to monitor the foetal heart beat continuously by CTG or to taking the decision to perform a caesarean section later than 13h10, which has been discussed already. Ultimately Ms M failed to demonstrate that the hospital staff acted negligently in attending to her during L's birth. This also means that she failed to prove that some wrongful conduct on the part of the hospital staff caused the injury to L's brain.

[32] Much was made of the admission into the record, of the evidence of Prof Izelle Smuts, also a paediatric neurologist, on the first day of the trial at the MEC's instance. The full court found that Prof Smuts' evidence should not have been admitted because 'it sought impermissibly to undo agreements previously reached by the parties' experts'. In addition, the court found that, 'it was never made clear which of the expert agreements the evidence sought to undo, and because there [was] no indication on the record that there was good cause for the introduction of the evidence in these circumstances'.

[33] The circumstances which led to the admission of the expert evidence of a further paediatric neurologist are not apparent from the record. And, as the full court remarked, there is no indication in the judgment of the trial court whether there was consideration, by that court, of the effect of the departure from the previous agreements; particularly on the fact that L suffers from CP as a result of the brain injury. In addition, this Court has discouraged departure from agreements previously reached by experts.²⁴

[34] Prof Smuts' evidence led to revised joint minutes of the other experts. Her evidence indeed impacted on issues which had been agreed on between the experts, the most significant of which was the condition in which L presented, when he was born. She cast doubt on the previously uncontested evidence that L

²⁴ *Bee v Road Accident Fund* [2018] ZASCA 52; 2018 (4) SA 366 (SCA) para 65.

was born flaccid, that his low Apgar score at birth was indicative of HIE, and that L suffered from CP. She highlighted his large brain size (megalencephaly), a large head (macrocephaly), both of which, according to her, are inconsistent with CP, which generally presents with microcephaly (a small head). She emphasised the unexpected divergence between the MRI imaging and the clinical findings and remarked on the absence of multi-organ failure in L's case.

[35] We agree that the trial court should not have allowed Prof Smuts' evidence without a substantive application setting out factors on which it could properly exercise its discretion. However, we are satisfied that, even without reference to Prof Smuts' evidence and the events pursuant thereto, Ms M did not establish negligence on the part of the hospital staff and the MEC.

[36] In the result the following order is made:

1. The appeal is upheld with costs, including the costs of two counsel where so employed.
2. The order of the full court is set aside and substituted with the following: 'The appeal is dismissed with costs, including the costs of two counsel where so employed'.

N DAMBUZA
ACTING PRESIDENT

C HEATON NICHOLLS
JUDGE OF APPEAL

Appearances

For the appellant: P Pauw with him U R D Mansingh

Instructed by: State Attorney, Johannesburg
State Attorney, Bloemfontein

For the respondents: D T V R du Plessis SC with him P Uys

Instructed by: Wim Krynauw Attorneys Inc, Johannesburg
Martins Attorneys, Bloemfontein