

**IN THE HIGH COURT OF SOUTH AFRICA
EASTERN CAPE DIVISION, BHISHO**

CASE NO: 586/2017

REPORTABLE

In the matter between

A.M obo L.M.

Plaintiff

And

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

Defendant

JUDGMENT

GOOSEN, J.

Introduction

[1] On 9 June 2011, the plaintiff gave birth to a son, L.M., at Saint Barnabas Hospital in the Eastern Cape Province. The plaintiff was 20 years old at the time. It was her second birth. Her first child was born by caesarean section. She was admitted to Saint Barnabas Hospital, in labour, at 03h25. L M was born by vaginal delivery at 09h00. He was born in a compromised state, as will be described below. The plaintiff and LM were discharged on 10 June 2011.

[2] On 21 September 2017, the plaintiff instituted action in her personal and representative capacities against the defendant. She alleged that the defendant's servants, medical personnel employed at Saint Barnabas Hospital, were negligent in the management and treatment of her during labour. She alleged that this negligence

caused LM to suffer a hypoxic ischaemic brain injury (HI injury) at term manifesting as cerebral palsy. In consequence, she claimed damages payable by the defendant.

[3] The defendant denied liability. Two special pleas were raised. The first, concerning the failure to give timeous and proper notice in terms of s3(1) of the Limitation of Legal Proceedings against Certain Organs of State Act, 40 of 2002, was resolved by an order granting condonation in terms of s3(4) on 7 May 2019. The second, concerning the alleged prescription of the claim by the plaintiff in her personal capacity, remained alive on the pleadings at trial.

[4] The case came to trial on 25 April 2022. It was agreed to separate the determination of liability from that of the quantification of damage, if any. An order to this effect was made at the commencement of the trial. In relation to liability, two issues had crystallised. The pre-trial minute which was signed by the parties identified the issues for determination as being:

‘a. What was the factual cause of the hypoxic ischaemic brain injuries suffered by the baby resulting in cerebral palsy and when did the said injuries occur?

b. Did negligence (if any) on the part of the Defendant’s staff cause or materially contribute to the baby’s hypoxic ischaemic brain injuries in the sense that the Defendant by the exercise of reasonable professional care and skill could have prevented it from occurring?’¹

The proceedings

[5] The trial, save for proceedings during which the plaintiff testified, was conducted on a virtual platform. This occurred by agreement between the parties, to accommodate several expert witnesses who were unable to attend the trial in

¹ In a pre-trial checklist submitted in accordance with case management requirements and to facilitate determination of set-down for trial, the issues for determination are framed as follows:

‘Whether the defendant’s employees were negligent in the management of the plaintiff’s labour and whether there was any causal relationship between such [negligent] management and the minor child’s condition.’

person. The trial proceeded over several days. It was then adjourned to allow for the plaintiff to testify in person. It was concluded in December 2022.

[6] The plaintiff presented the evidence of five expert witnesses. They were Prof Davies (a neonatologist); Prof Andronikou (a radiologist); Dr Kara (a paediatrician); Dr Pearce (a paediatric neurologist); and Prof Anthony (an obstetrician). The defendant only presented the evidence of Prof Rothberg (a neonatologist). The defendant qualified several other expert witnesses. Joint minutes reflecting points of agreement and disagreement between the parties' respective experts were prepared and, during the plaintiff's case, reference was made to these joint minutes where relevant.

[7] As far as documentary evidence is concerned, the plaintiff produced the maternity medical kept by the hospital. The plaintiff also produced medical records relating to post-natal treatment of LM and the plaintiff. Extensive reference was made to these medical records by the experts, both in their reports and testimony. At the commencement of the trial Mr McKelvey, who appeared for the plaintiff, pointed out that they were not admitted. Accordingly, the normal evidentiary rules apply. Although it was stated that the evidential value and weight to be attached to the records would be addressed at the conclusion of the trial, Mr McKelvey foreshadowed plaintiff's stance, in his opening address, with reference to the judgment in *HN v MEC for Health, KwaZulu Natal*,² where Koen J set out the approach as follows,

'Both parties made extensive reference during the leading of the evidence to the hospital records which the Defendant had kept, relating to the Plaintiff's confinement at the Greytown Hospital and the birth of M. At the commencement of the trial, it had been agreed that the status to be assigned to these documents inter alia was that they are what they purport to be without being proof of the content thereof.

² *HN v MEC for Health, KwaZulu Natal* [2018] ZAKZPHC 8 (4 April 2018) at para 6 – 7.

To the extent that there are records, the Defendant has inter alia relied on a favourable Apgar score which the nursing staff of the Defendant had determined at one minute and five minutes after M's birth as indicative of him having been born as a healthy baby. The Plaintiff, on the other hand, has relied on references in the official hospital records that there was 'foetal distress' and 'cephalo-pelvic disproportion' present to support her case. The questions arising from relate to the admissibility and the evidentiary value to be given to these entries appearing in these records.

Statements in the medical records that are favourable to the Defendant are hearsay where the author thereof has not been called to testify, and hence not admissible. Accordingly, the apparent high Apgar scores relied upon by Doctor Wildenboer, namely scores of 7 and 8 out of 10, determined at 1 and 5 minutes respectively, remain hearsay as the author determining and recording those scores was not called to testify thereto. No application was made for the admission thereof in evidence in terms of section 3 of the Evidence Law Amendment Act 1998, but even if there was, it would be unlikely to have succeeded as there was no evidence that the author thereof was no longer available to give that evidence. These scores are in any event, in the opinion of all four medical experts who testified, subjective and to that extent open to debate and unreliable. This was particularly, as Doctor Kara testified, that the five-minute score would be inflated by M's resuscitation with oxygen. Doctor Wildenboer agreed. Thus, even if admissible evidence, no valid conclusion can be drawn from these scores.

Recordings favourable to the Plaintiff's case in establishing negligence and liability generally, and accordingly damaging to the Defendant's case, made as part of the records kept by the Defendant's servants, are however on a different footing. They constitute admissions by the servants of the Defendant made in the ordinary course of discharging their duties, which are binding against the Defendant. The Defendant's staff are obliged to make these statements by recording the medical position as it unfolds in the records. They have an obligation to speak on behalf of the Defendant and dispute what is recorded, if indeed incorrect.'

[8] As in that case, no application was made for the admission of the hospital records in terms of s 3 of the Law of Evidence Amendment Act. I shall return to the consequences of this, later in the judgment.

[9] During the trial, reference was also made to a substantial volume of published medical and scientific journal articles and textbooks on relevant subjects. For reasons which will become apparent, this material will only be referred to where strictly necessary, and where the material to which reference was made was adopted by the expert concerned.³ It was held, in *Menday v Protea Assurance Co Ltd*⁴ that:

‘Where .. an expert relies on passages in a text book, it must be shown, firstly, that he can by reason of his own training, affirm (at least in principle) the correctness of the statements in that book; and, secondly that the work to which he refers is reliable in the sense that it has been written by a person of established repute or proved experience in that field. In other words, an expert with purely theoretical knowledge cannot in my view support his opinion in a special field (of which he has no personal experience or knowledge) by referring to passages in a work which has itself not been shown to be authoritative... [T]he dangers of holding the contrary are obvious.’

[10] Finally, in respect of the unchallenged evidence of expert witnesses, it remains to point out that it does not necessarily follow that because the opinion is unchallenged that it must be accepted. The expressed opinion must be founded upon facts admitted or accepted by the parties or found by the court. And such opinions must be based upon sound and logical reasoning. It is incumbent upon the court to determine whether the expressed opinion, despite being unchallenged, is to be accepted.⁵

The facts

³ See *S v Collop* 1981 (1) SA 150 (A); *S v Harris* 1965 (2) SA 340 (A) at 344C-D.

⁴ *Menday v Protea Assurance Co Ltd* 1976 (1) SA 656 (E) at 569H.

⁵ *Michael & another v Linksfield Park Clinic (Pty) Ltd & another* 2001 (3) SA 1188 (SCA); [2002] 1 All SA 384; [2001] ZASCA 12 para 36 – 37. *Buthelezi v Ndaba* 2013 (5) SA 437 (SCA); [2013] ZASCA 72 para 1.

[11] The plaintiff said that she had attended the antenatal clinic on one occasion, when she was already six months pregnant. She testified that she experienced no complications or illnesses during pregnancy. The records from the antenatal clinic indicate that on examination her blood test results were normal and she tested negative for viral infections. The plaintiff was admitted to Saint Barnabas Hospital, in labour, in the early hours of the morning of 9 June 2011.

[12] During her testimony, the plaintiff admitted that she had presented a different history of antenatal clinic attendance to the expert witnesses with whom she had consulted. When asked to explain why she had dishonestly inflated the number of antenatal attendances, she said that she did not want to appear to be a parent who did not care. She explained her limited attendances on the basis that she was afraid of her employer and did not want to be absent from work. Although the plaintiff did not create a favourable impression in this regard, little turns upon her evidence as to what occurred, in relation to her pregnancy, prior to her admission. Her account of her labour and the birth of LM is, as will become apparent, also not decisive.

[13] The hospital records reflect that the plaintiff was examined on admission at 03h25 on the morning of 9 June 2011. LM was born at 09h00 by normal vaginal delivery. At birth LM weighed 2900g and had a head circumference of 34cm. His length was recorded as 41cm. The experts, including those qualified by the defendant but not called, were agreed that the length was not accurately recorded. No significance was attached to this measurement.

[14] The maternity records indicate that the baby did not cry at birth and that active resuscitation, which involved the use of an 'ambu-bag' to stimulate breathing, and oxygen was used. The plaintiff confirmed this. She said that the baby was taken away after birth and she later saw him in an incubator with tubes in his nostrils. The baby was assigned Apgar scores of 4/10 at 1 minute and 6/10 at 5 minutes. The record indicates that he was hypotonic and that his primal reflexes (Moro, grasp and suck) were absent. He was fed with a nasogastric tube. The placenta, on gross examination, was recorded as normal.

[15] The plea of prescription of the plaintiff's claim may be easily disposed of. The plaintiff explained that she had come to institute action against the defendant after meeting a woman at a clinic where her son received treatment. This was in 2017. She was referred to an attorney who advised her. Shortly thereafter this action was instituted. It was argued that the plaintiff had been aware that LM was diagnosed with cerebral palsy from shortly after his birth, and therefore ought, reasonably, to have known that she had a claim in her personal capacity against the defendant. In my view, there is nothing to gainsay the plaintiff's evidence as to when she acquired knowledge of her rights. Accordingly, the plea of prescription must fail.

The condition at birth and cerebral palsy

[16] Professors Davies and Johnson and Drs Pearce and Kara concur that LM suffered from hypoxic ischaemic encephalopathy (HIE) at birth. They accepted that although all the diagnostic evidence for encephalopathy, as set out in the American College of Obstetrics and Gynaecology (ACOG) guidelines, was not present, LM presented with sufficient features to conclude that he suffered from HIE. These were the low Apgar scores, the need for resuscitation at birth, the absence of primal reflexes and the baby's hypotonic state. They considered that it was of moderate severity. Dr Rothberg, who testified for the defendant, also accepted that LM manifested HIE at birth. It was therefore common cause that LM suffered a hypoxic ischaemic insult which caused HIE, as manifested at birth. The question was gave rise to the HI injury and when it occurred.

[17] It is apposite to highlight the fact that the experts all drew upon the maternity records as the factual basis for their opinions. The plaintiff's experts were entitled to do so, notwithstanding the hearsay nature of the records, for the reasons articulated in *HN v MEC for Health, Kwazulu-Natal*. As in that case, Dr Kara here also expressed the opinion that the Apgar scores as recorded may well have slightly elevated because the baby had been resuscitated. He pointed out that the low Apgar scores even after resuscitation indicated a condition, at birth, worse than suggested by the Apgar scores.

[18] Dr Pearce, a paediatric neurologist, consulted with LM and undertook an assessment of the medical records and expert reports. Her diagnosis of LM's medical condition was that he suffers from a mixed type of cerebral palsy, i.e., both dyskinetic and spastic, which is dystonic. She described this as the most severe form of cerebral palsy in terms of motor function and speech. She confirmed that the clinical picture presented by LM correlates with the brain injury as depicted in the MRI and as described by Prof Andronikou.

[19] Dr Pearce also confirmed that, upon a proper assessment of the maternity records, LM presented at birth with moderate HIE. Her opinion was that the HIE was because of intrapartum hypoxic ischaemia. There was no evidence in the medical records or history to point to an antepartum event or occurrence giving rise to hypoxic ischaemic insult which would explain the HIE outcome at birth. I shall return to this aspect of the matter later in the judgment, since the possibility of an antepartum causal factor was raised by Dr Rothberg, on behalf of the defendant.

[20] Dr Pearce dealt with the post-natal care of LM and the fact that he was discharged within 24 hours of birth. She considered that the early discharge was inappropriate given the condition of LM at birth. She expressed the view that there was a window of opportunity immediately post-delivery for treatment which might have mitigated the progression of the cerebral palsy.

The cause of the brain injury

[21] There is no dispute that LM suffered a profound HI injury to the brain and that he now presents with severe mixed cerebral palsy. A key question, however, was when this injury occurred and what caused it.

[22] On 7 November 2017, a Magnetic Resonance Imaging (MRI) scan was performed on LM. Prof Andronikou testified to the interpretation of the MRI. He concluded that the MRI displays features of 'a global insult to the brain due to hypoxic ischaemic injury of an acute profound nature at term.' There was no dispute about the nature of the injury pattern. Joint minutes concluded between Prof Andronikou and both radiologists qualified by the defendant reflect this concurrence.

[23] In his testimony, however, Prof Andronikou provided an explanation for the description of the nature of the injury and the mechanism by which such injury occurs. He said that at the time that he prepared his report, in 2017, he used terminology which was descriptive of the mechanism rather than the injury pattern. This explained his use of the phrase 'acute profound' which might be contrasted with the term 'partial prolonged.' The MRI images indicate an injury pattern to a region of the brain. In this case the injury is to the basal ganglia thalamus region, i.e., the core or central grey matter of the brain. Such injury typically occurs when there is a global insult which completely shuts off oxygenated blood to the brain. He stated that the use of the term 'acute profound' had in fact been misunderstood to refer only to a sudden and unpredictable event causing the occlusion of blood supply to the brain. He explained that the injury pattern was not always associated with what are termed sentinel events. In the light of this he would not now describe the injury as indicating an acute profound pattern. He would merely describe the injury pattern as being a basal ganglia thalamus (BGT) or central core injury.

[24] He said the following:

'So, in the past, the words partial prolonged do not describe what you are seeing in the brain. What you are seeing in the brain in partial prolonged, is a watershed. It is an injury between the main vascular territories. When we used to talk about acute profound, we are talking about whether the injury has affected the [deep] nuclei, the areas of the brain that have got such a high energy demand at that period of your life. And but we were describing or what we knew from the literature and the time and going, well, that pattern happens when the injury is of a short duration and very severe.

I am glad you called me to court so I can go with the words acute, I mean many hours. Hours, many hours. With the word prolonged I mean like 24 hours, much longer.'

[25] Prof Andronikou's reinterpretation of the word 'acute' to suggest an occurrence that is not sudden or takes place over a short duration, must be seen

against the backdrop of his consistent assertion that the BGT injury pattern occurs when the oxygen supply is completely or near completely shut off. He did not suggest that the word 'acute' is now uniformly taken by radiologists to mean something other than a sudden insult of short duration. Prof Andronikou accepted that the imaging indicates some measure of damage in the perirolandic and corticospinal region. This was recorded in a joint minute concluded with Dr Swartzberg, a radiologist qualified by the defendant. Prof Andronikou, however, said the following:

'...so, both of us definitely say that it is acute profound. And then he has been, he mentions in his report that he thinks there is a watershed injury, and I am not completely agreed with this. What I have done is I cannot disprove considering that there is an injury involved in the surface of the brain, the cortex, and that the perirolandic region which normally fits with the deep nuclei pattern, is also within the watershed zone. I have conceded that it is, there is a potential of a possibility, that there is an additional watershed injury. But in fact, without other watershed injuries, that is unlikely. But I concede that there is, I cannot exclude the watershed injury as of course for perirolandic involvement. But basically, we both agreed on the acute profound components, and he has suggested a watershed injury as well, which is a partial prolonged component and I have excluded that I cannot exclude that.'

[26] Dr Swartzberg did not testify. His analysis of the MRI imaging is not before the court and not explained. The concession, made on the basis that it is not possible to exclude the additional injury features, is not, as the passage above indicates, a positive assertion of a fact-based opinion. On the contrary, Prof Andronikou highlighted the improbability of such additional injury given the absence of other, expected, watershed injuries. I shall return briefly to the effect of this evidence hereunder.

[27] Dr Kara testified about the cause or causes of the HI injury and whether the injury arose, intrapartum. He examined LM and diagnosed him as suffering from dyskinetic cerebral palsy on a gross motor function scale of 5, which accords with the diagnosis of Dr Pearce. Regarding the timing of the HI injury, Dr Kara's opinion was that it occurred intrapartum. In support of this conclusion, he stated that the only

known antepartum factor was the birth of the plaintiff's first child by caesarean section because of preeclampsia. The measurements of LM taken at birth, particularly his normal weight and head size, do not point to any growth restriction in utero.

[28] Dr Kara stated that there is nothing to suggest that intrauterine infection or maternal chorioamnionitis, played any role in the development of the cerebral palsy. The presentation of HIE at birth suggests an HI injury having occurred intrapartum. He said that the MRI scan establishes that the injury occurred at term. This excludes an early antenatal injury, i.e., prior to 35 weeks gestation. The fact that the baby required resuscitation at birth; was hypotonic, unable to feed and the absence of reflexes all point, on the probabilities to an intrapartum insult. Dr Kara referred to a study by *Rennie et al*⁶ which suggested that 80% of cases resulting in dyskinetic cerebral palsy are because of intrapartum hypoxic ischaemia. This probability is supported by the 2014 Revision of the ACOG criteria.

[29] Dr Kara's opinion was supported by Prof Davies and Prof Anthony. Dr Rothberg expressed the view that a pathway to cerebral palsy that involved antepartum factors could not be excluded. It was accepted by all the expert witnesses that there was, in this case, no evidence to suggest the occurrence of a sentinel event, such as maternal haemorrhaging, placental abruption, prolapse or occlusion of the umbilical cord or some other unexpected and catastrophic event during labour or birth. A joint minute between Prof Davies and Dr Rothberg was presented in evidence. The minute records the following points of agreement:

(a) That LM suffers from permanent brain damage in the form of a mixed cerebral palsy with severe impairment because of a hypoxic ischaemic brain injury.

(b) That the MRI is in keeping with a hypoxic ischaemic brain injury.

⁶ Rennie JM, Hagmann CF, Robertson NJ. *Outcome after Intrapartum Hypoxic Ischaemia at Term*, Semin Fetal Neonatal Medicine, 2007 Oct; 12(5):397-407 Epub 2007 Sep 7.

(c) That the neonatal encephalopathy presented at birth was most probably due to hypoxic ischaemia.

(d) That the recorded growth parameters are appropriate for gestational age.

(e) That chronic hypertension, intrauterine congenital factors or genetic or other syndromes can be excluded as possible causes of the pathology; and

(f) That the monitoring of the plaintiff during labour was not according to the published VBAC (vaginal birth after caesarean section) guidelines.

[30] It was Prof Davies' evidence that the HI injury occurred intrapartum. He stated that the intrapartum period poses the greatest risk to the foetus. The BGT injury pattern is not only associated with what is described as an acute or sudden and profound hypoxic ischaemic event. It can also be observed where the hypoxic ischaemia is partial and occurs over a period. He referred, in this regard, to the results of a study conducted by *Smith et al*⁷ in support of his opinion. I shall touch upon this study hereunder.

[31] Both Prof Davies and Anthony described the mechanisms of hypoxic ischaemic injury as follows: a decrease in the level of oxygen in blood supplied to tissue gives rise to a neurological response in the foetal brain. Oxygenated blood is shunted away from less vital or critical, peripheral areas of the brain to the deeper tissues and core centres of the brain. This is an autonomic response which serves to protect those core structures from damage. Brain tissue in the peripheral and surface areas may suffer damage because of the absence of an oxygenated blood supply. The compensatory shunting of blood to protect core tissue can result in damage observed in the 'watershed' areas, i.e., at the furthest extremities of blood vessels. It is in this context that reference was made to 'partial prolonged' insults where the

⁷ Smith J, Solomons R, Vollmer L, Langenegger EJ, Lotz JW, Andronikou S, Anthony J. *Intrapartum Basal Ganglia-Thalamic Pattern Injury and Radiologically Termed 'Acute Profound Hypoxic Ischemic Brain Injury' Are Not Synonymous* American Journal of Perinatology (Am J Perinatol), 4 Nov 2020.

blood supply is constrained in successive events over a period. In such instances the damage is seen on an MRI in the peripheral areas. In the case of a sudden ‘profound’ event, where little or no oxygenated blood is available the damage occurs in the basal ganglia-thalamic region since it is, in the foetus, the most metabolically active region of the brain.

[32] This description accords with that provided by Prof Andronikou. The *Smith* study, to which both Prof Andronikou and Anthony were contributing authors, involved a retrospective analysis of 10 medico-legal cases of neonatal encephalopathy-cerebral palsy survivors who sustained intrapartum hypoxic ischaemic basal ganglia-thalamic pattern injury in the absence of a sentinel event. The results of the study suggest that a BGT pattern injury may occur in the absence of a sentinel event where there is evidence of foetal distress or a non-reassuring foetal heart rate in the form of abnormal cardiotocography (CTG) readings during labour.

[33] The assertion made by *Smith et al*, is that the term ‘acute profound’ has come to be associated with obstetric events which occur in the period immediately prior to birth and which result in a BGT injury pattern. Such description of the pattern as ‘acute profound’ should be avoided in radiological descriptions, the article suggests, since a BGT pattern may arise from sub-acute insults over a prolonged period.

[34] The association of the term ‘acute profound’ with sudden short duration events, no doubt arises from the plain meaning of the words themselves and the descriptions of events as presented in evidence before the courts. This can be gleaned from several judgments dealing with cases such as the present. In *AN obo EN v MEC for Health, Eastern Cape*⁸, Gorven AJA (as he then was) said:

⁸ *AN obo EN v MEC for Health, Eastern Cape* (585/2018) [2019] ZASCA 102 (15 August 2019) par 13. See

M v MEC for Health, Eastern Cape (699/17) [2018] ZASCA 141 (1 October 2018) at par 58 -60; *The Member of the Executive Council for Health, Eastern Cape v DL obo AL* (Case no 117/2020) [2021] ZASCA 68 (03 June 2021) par 21.

‘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.’

[35] This was based upon acceptance of the evidence of Prof Van Toorn (another contributing author to the *Smith* paper), as stated by the learned judge;⁹

‘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.’

[36] The evidence in that matter established that there had been compression of the cord and that it had constituted a sentinel event which was not predictable nor possible to detect timeously by way of foetal monitoring. The apparent concern of the *Smith* article with the radiological description employed does not alter the fact that a particular injury pattern arises from each of the mechanisms by which such injury occurs. In each case the injury pattern as established on MRI will provide insight into the mechanism by which it occurred. Prof Davies and Prof Anthony confirmed this.

[37] Prof Andronikou’s suggestion that ‘acute’ may also relate to events that take many hours, does not accord with the description of the pathogenesis of injury given

⁹ Ibid par 14.

by Prof Davies and Anthony. The pattern of brain injury noted by Prof Andronikou did not include features of injury caused by partial and prolonged hypoxic insult, or as suggested by Prof Anthony with reference to the *Smith* study, a series of sub-acute hypoxic insults over a period.

[38] Prof Anthony said that even in the absence of a sentinel event, a BGT injury pattern could occur because of repeated and prolonged sub-acute ischaemic events which overwhelm the foetus' capacity to compensate for the hypoxia. He described the process as the straw that breaks the camel's back. There was, however, no radiological evidence to suggest partial restriction of blood flow to the foetal brain on multiple occasions during the plaintiff's labour. There was no evidence to suggest multiple occlusions of blood flow to the foetal brain during labour. What remains in the evidence, is that the brain injury arose because of a global insult which shut off the supply of oxygenated blood to the foetal brain completely or almost completely, for a sufficient duration to give rise to the BGT pattern injury.

[39] Dr Rothberg, as indicated, postulated the possibility of an antepartum component to the HIE presented at birth. He accepted that LM suffers from a mixed cerebral palsy following neonatal encephalopathy. This, he suggested is associated with late antepartum or intrapartum hypoxic-ischaemic insult. He referenced an article published by *Bhorat et al*¹⁰ which contends that causation of cerebral palsy should not simply be based upon an intrapartum perspective with radiological confirmation. Dr Rothberg's opinion regarding a possible antepartum hypoxic ischaemic event was, however, not based upon any facts. There is no evidence to support a finding that it is probable that the birth outcome arose because of some antepartum event. Dr Rothberg also suggested that there is a growing awareness that placental malfunction or pathology plays a role in the development of HIE and resultant cerebral palsy. In this case, the gross examination of the placenta indicated that there was no abnormality. The placenta was not sent for histology. The fact that there may be a body of scientific opinion which favours histological investigation of placental function to determine its role in HIE-cerebral palsy outcomes, does not assist the defendant. There is no evidence to suggest that placental function was

¹⁰ Bhorat et al, S Afr Med J 2021; 111 (Suppl 1): 280-288.

relevant. It is, in my view, not for the plaintiff to prove that the birth outcome was not the result of placental malfunction where there is no indication that the placenta was not normal.

[40] I am satisfied that the evidence established that LM suffered a profound global hypoxic ischaemic insult, intrapartum, which shut off oxygenated blood flow to the foetal brain completely or almost completely, causing the BGT pattern injury. It is not possible on the evidence to determine when the insult causing injury occurred. Nor, in the absence of a defined or recognised sentinel event, why the insult occurred.

The management of plaintiff's labour

[41] I turn now to the second issue, namely whether the medical staff at the hospital were negligent in the management of the plaintiff's labour and whether such negligence caused, or materially contributed to the injury suffered by the baby.

[42] Prof Anthony expressed the view that the maternity records reflected a clear failure to monitor the plaintiff's progress of labour in accordance with the published guidelines for vaginal birth after caesarean section (VBAC). In this case the plaintiff's prior history presented as a risk factor which necessitated careful monitoring of the foetus' condition during labour. The monitoring, he said, was substandard. Prof Anthony's view was supported by all the expert witnesses, including Dr Rothberg.

[43] I accept that the failure to employ a CTG or to auscultate the foetal heart rate and record the results in accordance with the VBAC guidelines, constitutes a breach of the duty of care owed by the medical staff to the plaintiff and her unborn foetus. It was known that the plaintiff's prior caesarean section and preeclampsia presented elevated risk during labour. The failure to ensure proper and effective monitoring of the foetal condition during labour created a risk that the medical personnel would not be able to act with reasonable expedition as required to prevent injury to the plaintiff or foetus.

[44] The defendant presented no evidence to gainsay the inference of inadequate monitoring to be drawn from the maternity records. Prof Anthony suggested that the failure to monitor the foetal heart rate appropriately caused the baby to suffer the HIE with ensuing cerebral palsy outcome. He described the foetal response to successive partial hypoxic events. At each event variations in the heart rate would occur. These would be detectable as accelerations and decelerations measurable in relation to the contractions of the uterus during labour. Where the foetal heart is compromised in its ability to normalise after a contraction or a hypoxic ischaemic event, bradycardia, a deceleration of the heart rate when compared to the base heart rate, would be evident. This would indicate hypoxic foetal distress. Since the process of labour necessarily places the foetus in some hypoxic distress, effective and careful monitoring of the foetal heart rate during labour is required. The monitoring, by CTG or by auscultation, would enable foetal distress to be detected so that appropriate interventions may be made to protect the mother and foetus.

[45] In this instance, he said that the effect of the inadequate monitoring of the foetal heart rate was that foetal distress was not detected and the medical staff were not able to act to mitigate foetal distress or expedite the delivery of the baby. It was this failure which, in the opinion of Prof Anthony, caused LM to suffer the HI injury which manifested as HIE at birth and resultant mixed cerebral palsy.

[46] Dr Rothberg, while accepting that the monitoring was sub-standard, did not support the conclusion that the failure to monitor properly, caused the baby to suffer a brain injury which it otherwise would not have suffered. He placed considerable reliance upon entries on the partogram contained in the maternity records. The entries involve 12 foetal heart rates recorded between admission and the delivery of the baby. These entries were all 'normal' or fell within a 'normal range' for the foetus.

[47] Dr Rothberg relied upon these recorded heart rates to support two propositions. The first was that the recordings indicated normal heart rates throughout the labour. There was, therefore, no indication of any abnormality or foetal distress. The record therefore does not support the proposition that there was a non-reassuring foetal condition during labour evidencing hypoxic ischaemia which might explain the outcome. The second, concerned the proposition that proper foetal

heart rate monitoring would have placed the staff in a position to prevent the injury suffered by the foetus. He said that the fact that the recorded heart rates were single rates, rather than rates recorded both before and after contractions, was inconsequential. He stated that it was highly improbable that the heart rates that were recorded, were recorded at a time when there was no sign of distress or bradycardia, which on Prof Anthony's testimony would have been present. This suggests that the monitoring would not have had any impact upon the result.

[48] The difficulty with this evidence is that it relied upon entries in the maternity record which were not proved. The person who made the entries and who took the heart rates did not testify. The record of heart rates, upon which the defendant relied was hearsay evidence. The consequence is that there is no admissible evidence to support the propositions advanced by Dr Rothberg. Dr Kara's view was that the rates were of little value in determining the condition of the foetus during labour since they are only single rates taken at the indicated times.

[49] Dr Rothberg's further evidence was that CTG monitoring is, in any event, an imperfect tool for determining the condition of a foetus during labour and there is no evidence to suggest that foetal heart rate monitoring necessarily reduces the probability of HIE and cerebral palsy. It is, in my view, unnecessary to engage in the debate about the efficacy or otherwise of CTG and foetal heart rate monitoring. The determination of causation in relation to a negligent act or omission is a matter which falls to the court, having regard to the facts established before it.

[50] Prof Anthony explained his reasoning with reference to an analogy of a child standing alongside a busy road. No one is watching the child, who runs into the road and is struck by a car. If the child is found lying in the middle of the road with traumatic injuries, it is reasonable to infer that he was struck by a car. It is also reasonable to infer that the outcome occurred because no-one was watching. If someone was watching the outcome could have been prevented by appropriate intervention.

[51] The analogy, in my view, was flawed. It does not contain sufficient fact-based premises to allow the final inference to be drawn. To find that the failure to properly

monitor the foetal heart rate and condition during labour caused, in the sense that it materially contributed to, the brain injury, would require additional facts to be inferred since there is no evidence to establish the existence of those necessary facts. It would need to be inferred that the monitoring would have indicated that the foetal heart rate was abnormal, and that the foetus was in distress. It would need to be inferred that the indication of foetal distress would occurred at a time when successful intervention could have taken place.

[52] Regarding the first requirement, namely that the monitoring would have indicated foetal distress, the only evidence to support this was that which described the mechanism of a partial prolonged hypoxic ischaemic insult. In such instances, abnormal foetal heart rates are likely to occur. But the evidence does not point to such mechanism of injury. Even if it was accepted that there would at some stage have been an indication of foetal distress, there is no basis to determine when that might have occurred, given the acute profound mechanism of injury, nor whether the intervention would have averted the injury.

[53] In *AN obo EN v MEC for Health, Kwazulu-Natal Molemela* JA¹¹ usefully set out the approach to establishing causal negligence, as follows:

‘In *Minister of Safety and Security v Van Duivenboden*, 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* 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*.

¹¹ Fn 8 above para 48 – 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 as reflected in the authorities referred to above. In re-stating the ‘but-for’ test in *Mashongwa*, the Constitutional Court settled the law on this aspect. It pointed out that the imputation of liability to the wrongdoer depends on whether the harmful conduct is either too remotely or sufficiently closely connected to the harm caused. It emphasised that where the traditional but-for test is adequate to establish a causal link, it may not be necessary to resort to the *Lee* test.’

(Footnotes omitted)

[54] In my view, the application of the traditional ‘but-for’ test is adequate in this case. The extended inferential reasoning set out above is not supported by the evidence. It comes close to reasoning from the result to the cause. I am accordingly unable to find that ‘but-for’ the failure to monitor the foetal heart rate, the injury would not have occurred.

[55] It follows therefore that I am unable to find that the plaintiff has established that the defendant is liable for the loss suffered. Regarding costs, I am not prepared to impose the usual costs order. I come to this conclusion on the basis that the circumstances of the matter are tragic. LM suffered a catastrophic injury during birth, the consequences of which are stark. This case is yet another matter in which evidence of systemic failure and neglect has emerged about the care and treatment provided in a public health facility under the control of the defendant. Our courts have lamented this situation on several occasions. In *AN obo EN v MEC for Health, Kwazulu-Natal*¹², Gorven JA said,

‘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

¹² Fn 8 above par 28.

has become pervasive in many such hospitals. Those reliant upon their services are receiving substandard care.'

[56] In this case, as in that matter, the medical staff were found to be negligent, although it could not be found that the negligence caused the harm suffered. The ongoing and persistent failure to ensure that persons who are dependent upon public health facilities in the province, receive proper care and treatment deserves censure. For this reason, I would deprive the defendant of the costs which would ordinarily follow the result.

[57] In the result, the plaintiff's claims are dismissed.

G. GOOSEN
JUDGE OF THE HIGH COURT

Dates heard: 25, 26, 28 & 29 April 2022
5, 6, 7 & 14 December 2022

Date delivered: 25 April 2023

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