



**NOT REPORTABLE**

**IN THE HIGH COURT OF SOUTH AFRICA  
(EASTERN CAPE DIVISION, MAKHANDA)**

CASE NO. CA18/2022

In the matter between:

TY (obo MY)

Appellant

and

MEC for Health, Gauteng Province

Respondent

*(in re the negligence of the staff at the Natalspruit Hospital)*

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**APPEAL JUDGMENT**

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**HARTLE J**

[1] The appellant appeals, with the leave of the Supreme Court of Appeal, against the judgment and order of the East London Circuit Local Division of this court (“*the trial court*”) dated 13 October 2020 dismissing her action for delictual damages which she had sought to claim in her representative capacity as mother and natural guardian of her minor child, M, who was born at the Natalspruit Hospital (“*the hospital*”) on 5 March 2014.

[2] In her claim she contended for negligent intrapartum care on the part of the hospital staff responsible for M’s safe delivery.

[3] In essence her case came down to the fact that the respondent’s employees failed to adequately monitor her and her foetus during her labour which led to a missed opportunity to detect foetal distress and to appropriately act upon it to prevent him from suffering a hypoxic-ischaemic injury to his brain. The latter injury was the fore runner to the mixed spastic cerebral palsy condition that he was diagnosed with in early childhood.

[4] The appellant initially instituted the action in the Gauteng Local Division but the matter was transferred to this court by order of the latter division in 2018.

[5] The respondent’s plea at first read as a bare denial of the claimed negligent treatment of the appellant or that the staff at the hospital were responsible for M’s

cerebral palsy, but was amended almost three years later, ostensibly just before the trial commenced.

[6] To the allegations going to the specific grounds of negligence pleaded by the appellant, the respondent countered that the hospital staff had kept her under constant and meticulous observation and that the treatment and advice given to her was rendered to her with such skill and diligence as could reasonably be expected “*in the circumstances of a pregnant woman presenting in labour with pyrexia and a urinary tract infection and a foetus with fetal tachycardia.*”

[7] Regarding causal negligence, the defendant denied that M’s condition was as a result of the negligent actions of the hospital’s employees. Instead he claimed that the child’s cerebral palsy arose rather as a result of certain factors set out in the amended plea.<sup>1</sup>

[8] These factors, alluded to in the trial court’s judgment “*as the so-called proximal and or distal factors as the probable causes of the cerebral palsy*” (sic) entailed: (1) the appellant’s illness during pregnancy which resulted in her losing weight in the third trimester; (2) the pyrexia and urinary tract infection that she presented with at the hospital upon her admission in labour; (3) the foetal

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<sup>1</sup> Implicit in the respondent’s plea in response to the appellant’s claim of causal negligence, is the suggestion that proper monitoring and detection and acting appropriately on the results would have not made an iota of difference because the cerebral palsy arose by virtue of a different pathway than hypoxic ischaemic encephalopathy.

tachycardia which the foetus presented with; and (4) the “*congenital neonatal encephalopathy*” (sic).<sup>2</sup>

[9] The matter proceeded to trial on the merits only after the trial court ordered a separation of the issues in terms of rule 33 (4) of the Uniform Rules of Court.

[10] The appellant testified that she fell pregnant during the year 2013. At the time she was residing in Engcobo, Eastern Cape Province, and initially accessed antenatal care at the local community clinic (Sinqumeni). She relocated to Gauteng during her pregnancy and also obtained further antenatal care at the Greenfield Clinic.

[11] On the face of it her antenatal care raised no concerns with the clinic staff although it appears that she suffered a weight loss of 9 - 10kg during her pregnancy over a significantly short period. The appellant herself confirmed that she was not ill at all during her pregnancy, but the defendant sought through expert testimony to persuade the trial court that her significant weight loss (the assumption being that it was as a result of illness)<sup>3</sup> had probably redounded or contributed to M’s cerebral palsy.

[12] On 4 March 2014 the appellant experienced labour pains and attended the Phola Park Clinic in Ekurhuleni where she expected to deliver.<sup>4</sup> She was examined

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<sup>2</sup> Gathering from the context the defendant intended to refer to “*congenital microcephaly*”.

<sup>3</sup> The evidence did not establish that the appellant was ill during her pregnancy.

<sup>4</sup> The appellant stated in her evidence that the clinic is “*meant for ... to give birth*” which accords with the respondent’s policy that a patient will first report to a primary healthcare facility that will refer her to a hospital in

and treated there. She was admitted to a bed and a drip was inserted. Her stay was brief and for a few hours only. Ultimately she was conveyed by ambulance to the hospital. She testified that she was told that the referral was necessary because they were “*not able to make me to give birth at the clinic*”. Although there are no records of her treatment at the clinic the surmise (from all the experts who testified) is that the appellant was probably treated for the infection for there to have been an absence of any reference to it again in the hospital records after she was admitted.<sup>5</sup> The appellant herself seemed oblivious to the fact that there was any concern around her having had a fever or her foetus having been in any kind of distress. Indeed according to her testimony the reason she presented herself at the clinic is that she

felt she was in labour which, so it was confirmed by the hospital records, was in fact the case.

[13] She arrived at the hospital in the late afternoon. The maternity case hospital records (these and others admitted into evidence at the trial on the customary basis that they are what they purport to be without either party admitting the correctness of the content thereof), indicate in a note made contemporaneously with her admission at 16h45 that she had presented at the referring clinic “*clinically not well*” with a fever and a temperature of 38.5°C, a urinary tract infection (leucocytes in her urine), and the foetus with foetal tachycardia, vouched for by CTG tracings provided to the hospital by the clinic. Indeed this accords with the premise pleaded by the respondent upon which he avers the appellant presented at the hospital with

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appropriate circumstances. This was such a case, the referral ostensibly necessitated by M’s “*foetal tachycardia*”. This confirms the critical risk factors at play when the appellant presented in labour.

<sup>5</sup> The notes of the Phola Clinic were not adduced at the trial.

her foetus, and which maladies the respondent contended were among the factors that gave rise to M's condition.

[14] I should add that the appellant had volunteered to the hospital staff upon her admission that she had drunk "*church water*" for eight months. This was evidently not of any concern to the staff as nothing further was noted in the records about it. I mention it however as the defendant latched on to this at the trial as a further factor

possibly contributing to M's condition.<sup>6</sup>

[15] The appellant testified that at the hospital she was not initially referred to a bed. She was told (commensurate with the hospital notes that record that she was still in the latent phase of labour upon her admission between 1 - 2cm dilation) that she was not yet to give birth and had to sit on a bench where she remained until

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<sup>6</sup> This was in my view a mere red herring. Whatever its implications, if any, it was not pleaded by the respondent that the church water was toxic, that the appellant's ingestion of it made her ill, or in the end was supposedly a causal factor for M's cerebral palsy. The trial court gave the appellant's mention of the ingestion of this water misplaced significance surprisingly after having agreed with counsel during closing arguments that it was a bit of "nonsense". Further, whilst cautioning itself that it should not enter into speculation as to the contents of the water, it nonetheless took "*judicial notice of the notorious fact that some churches are and have been administering various toxins to congregants as published in recent times*". The consequence of elevating unwarranted concern to the appellant's throw away comment noted by the hospital in its clinic notes as a feature of the causality enquiry at the trial resulted in the appellant being unfairly criticized for failing to make any "*meaningful attempt*" to "*discharge the evidential burden resting upon her*" to "*meaningfully deal with the nature and effect of the water, if any, on her and possibly the foetus*". The inevitable conclusion reached by the trial court along this trajectory is that "(t)his proximal factor too (was) not ... excluded".

3am the following morning when she was referred to a ward once her labour pains became severe. An earlier assessment at 12pm that night had forecast that she would be checked again in the labour ward once in active labour.

[16] At 3am on 5 March 2014 a doctor arrived at her bedside and broke her water but still it remained “*hard*” for her to give birth.

[17] She answered positively to various questions posed to her under cross examination that she was continuously checked and examined intermittently by a nurse. According to questions elicited by the trial court, this entailed both vaginal

examinations and foetal heart rate checks.<sup>7</sup>

[18] Eventually at 5.15am she gave birth to M.

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<sup>7</sup> The evidence indicated the lack to be in continuous CTG monitoring despite how the appellant presented on admission (and the results of earlier foetal heart rate tracings that were not assuring), as well as the failure to have monitored her high temperature which by all accounts would have had an impact on the foetus. The joint minute between the obstetricians for example recorded that no temperature checks were done whilst the appellant was in hospital though she had been admitted on this premise by the referring clinic. There was also no specific cause for her pyrexia documented. The records reflected no treatment given for her fever and likely infection. CTG's were only done for short periods after her admission which showed persistent foetal tachycardia and several episodes of reduced variability. Even though the experts agreed that the foetal heart rate recordings, such as were obtained, were not indicative of significant foetal hypoxia, they concluded that they were neither normal and at best were equivocal. There was one check on the fetal heart rate at 8.50 pm and no fetal heart rate monitoring in labour from about 11pm until birth. The experts agreed that in view of their equivocal nature CTG monitoring ought to have been continued in the ward at least on an intermittent basis, which was not done at all.

[19] Significantly, she testified that her baby came out not crying. The staff took him away telling her that he was not breathing well. In fact she formed the belief that he was not even alive but was later apprized that he was in the ward.

[20] She saw him the following morning having gone to find out where he was. (The staff did not bring him to her bed.) He was in the “*ward of the sick children*”, on a drip, and “*needed oxygen*” administered to him by something “*inserted in him* (in his nostrils) *and also he was covered on his head only the lower part of his body .... was seen*”.

[21] She noticed that he was different from how she remembered seeing her first two children in the hospital.<sup>8</sup> He was not breathing well. He was not crying. He was not showing movement in his arms or legs and was foaming at the mouth and having fits. She was also told by the doctor that M was fitting.

[22] Despite how the appellant had presented at the hospital on the morning of 4 March 2014, she was discharged the following day but M remained in hospital for three weeks after his delivery. During this period he was fed through a tube. She did try unsuccessfully to feed him at her breast. Once he was discharged into her care, she fed him by spoon.

[23] At the age of three months she noticed that he was definitely not on par with other children development-wise. He could not sit or engage. He was not using

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<sup>8</sup> Co-incidentally this was her fourth pregnancy, but she miscarried in her third pregnancy. M was her third child.



his hands which both remained clenched in the formation of a fist. His toilet habits were not normal. He was (later) unable to walk or talk.

[24] She returned from Gauteng to Engcobo where she took him back to the Sinqumeni clinic for immunization and in 2015 was referred to the Red Cross Children's Hospital in Cape Town for him to be examined (the records indicate for suspected neonatal encephalopathy based on the history that she had given them).<sup>9</sup>

[25] The appellant's experience of the birth of M was largely confirmed by the medical records of the hospital to the extent that the staff bothered to complete the template documents that comprise all the relevant stages of a patient's confinement and delivery of a baby and their joint care but significantly there was an absence of

clinical notes detailing the treatment administered to him neonatally.<sup>10</sup> The experts advising the parties and seeking to assist the trial court in determining the vital

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<sup>9</sup> The history provided by the appellant was given without any inkling that M suffered from cerebral palsy. She repeated it to the medical experts who consulted with her even before the hospital's maternity records were made available to the plaintiff's attorneys. To the extent that the records were completed, her contemporaneous account of M being neurologically depressed at birth happened to coincide with what the records state.

<sup>10</sup> There were a few notes made at M's birth concerning aspects of his condition but certainly nothing in the ward later on for the 23 days that he remained under the care of the hospital. There was no reference in the evidence as to what the Maternity Guidelines required of the hospital at the time concerning the requisite recordkeeping, but it is fairly common knowledge in matters of this kind that there are Guidelines that prescribe how health records are to be maintained. Section 13 of the National Health Act, No. 61 of 2003 ("NHA") creates the obligation for the person in charge of a health establishment to ensure that a health record "*containing such information as may be prescribed*" is created and maintained at health establishments (including public institutions) for every user of health services. Section 17 (2(d) of the NHA under the caption "*Protection of Health Records*" provides that any person who "*fails to create a record when properly required to do so*" commits a criminal offence.

issues upon trial had therefore to piece together what information they could from the negligible records available and reason inferentially where applicable.

[26] I do not intend to repeat all the salient features of the appellant's labour or the obstetric care rendered to her as recorded in the hospital's maternity records (again very negligibly) save that which is relevant to determine the issue of factual causation, which is at the core of the present appeal.

[27] The trial court found that the appellant had established sub-standard monitoring on the part of the staff who attended to her at the hospital whilst in their care and leading up to the birth of M. The expert obstetricians who reviewed the management of the appellant's labour and testified at the trial indeed conceded that the proper monitoring of the appellant by CTG in all the circumstances, especially having regard to the acknowledged risk factors that she and her unborn foetus had presented with at admission, had been substandard. It was also accepted that M's birth could have been assisted if with proper monitoring his foetal distress (given the prior non-reassuring CTG traces) had been recognized in time and acted upon.

[28] There is no cross-appeal with regard to the aspect of negligence.

[29] What prompted the present appeal however is the trial court's failure to have found that the appellant had established a causal link between the accepted substandard monitoring during her labour and the harm suffered by her in her representative capacity as a result of M developing cerebral palsy. Rather the court's judgment in this respect concluded that the evidence suggested in all

probability that “*the harm could have arisen due to the cumulative effect of the so-called proximal or distal factors*”.

[30] In getting to this conclusion the trial court rejected the appellant’s case, underpinned by the expert testimony adduced on her behalf, that M’s cerebral palsy was more probably related to birth events having a causal connection with the established negligence than the factors referenced by the respondent in his plea.

[31] Alluding to a professional Consensus Statement referenced by the experts concerning the issue whether M had exhibited neonatal encephalopathy supportive of an intrapartum birth event, the trial court observed as if this was the test that “*a comprehensive multidimensional assessment (had) to be performed taking into account all potential contributing factors*” but suggested that such an assessment had not been undertaken *vis-à-vis* M and, more especially couldn’t, because there was a dearth of hospital records concerning his treatment after birth against which the relevant Consensus Statement template could be tested.

[32] With regard to the factors relied upon by the respondent as constituting either distal or proximal factors as the probable cause(s) of the cerebral palsy it found that their existence (each of the pleaded bases found proven)<sup>11</sup> had simply not been excluded. It found further that the opinions of the appellant’s experts relied upon to gainsay or refute that the factors contended for by the respondent

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<sup>11</sup> The trial court added its finding that the appellant had ingested toxic church water as one of the bases.

were causal of M's cerebral palsy rather than negligent intrapartum care, were “*unacceptable*” to it.

[33] The reasons for this according to the trial court's judgment relate to a perceived lack of cogency in the opinions expressed by them on the contentious issues. Indeed the trial court concluded in this respect that:

*“It would seem to me that the expert evidence led on behalf of the plaintiff, on the triable issues, is argumentative, not authoritative and not persuasive. It simply fails to take account of all the facts and does not exclude the proximal and distal factors as the cause for the harm.”*

[34] What the contentious issues were was not clearly identified in the judgment but the trial court's starting premise cannot be criticized. Remarking upon the fact that there were “*a lot of peripheral issues relating to (the) matter,*” the “*true issue*” delineated by it was “*simply whether or not the cerebral palsy was caused by a hypoxic ischemic injury which was not detected or prevented by the defendant's employees and...that (it) ultimately caused the cerebral palsy.*”<sup>12</sup>

[35] Before advertent to an evaluation of the expert testimony, it is necessary to highlight that the concepts of “*distal*” and “*proximal*” are unique to the application of a professional Consensus Statement developed by the experts in the field of obstetrics and gynaecology that was referenced by the experts in the trial. Although its criteria implicating intrapartum hypoxia in neonatal encephalopathy have utility

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<sup>12</sup> A proper reading of this conclusion suggests that there was no confusion that M suffered a hypoxic ischaemic injury, the injury qualified by the observations that it was one that was missed because of a lack in the monitoring and further not prevented by the respondent's employees.

from an obstetric perspective and can and do assist the court in its determination of the proof of a causal link between a defendant's actions or omissions (read negligent intrapartum care), on the one hand, and the harm suffered by the plaintiff (cerebral palsy in this instance), on the other hand, its application is not a substitute for the court's own legal causation enquiry that it must undertake.

[36] It is a trite principle that a successful delictual claim entails the proof of a causal link between a defendant's actions or omissions, on the one hand, and the harm suffered by the plaintiff, on the other hand.<sup>13</sup> This is in accordance with the "but-for" test.<sup>14</sup> As is indicated by the authorities, in order to apply this test one must make a hypothetical enquiry as to what probably would have happened but for the wrongful conduct of the defendant. Sometimes however this enquiry involves the mental elimination of the wrongful conduct and the substitution of a hypothetical course of lawful conduct (in this instance the proper monitoring of the plaintiff and her then unborn foetus) and the posing of the question as to whether upon such hypothesis the plaintiff's loss would have ensued or not.<sup>15</sup>

[37] Legal causation must be established on a balance of probabilities.<sup>16</sup>

[38] The vital question in this matter was whether, as a matter of probability, M's condition would in any event have ensued even if the Respondent's negligent

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<sup>13</sup> *International Shipping Co (Pty) Ltd v Bentley* 1990 (1) SA 680 (A) at 700F-I; *Siman & Co (Pty) Ltd v Barclays National Bank Ltd* 1984 (2) SA 888 (A) at 915B-H; *Minister of Police v Skosana* 1977 (1) SA 31 (A) at 35C-E; *Lee v Minister of Correctional Services* 2013 (2) SA 144 (CC) at para [37] - 58]; *Oppelt v Head: Health, Department of Health Provincial Administration: Western Cape* 2016 (1) SA 325 (CC) at [35].

<sup>14</sup> *Lee, supra* at para [37] - [58]

<sup>15</sup> *Mashongwa v PRASA* 2016 (3) SA 528 (CC) at [65]; *AN v MEC for Health, Eastern Cape* [2019] ZASCA 102 at [8].

<sup>16</sup> *Lee supra* at [39]

intrapartum care had not occurred,<sup>17</sup> given (this confirmed by the common testimony of the paediatric experts) the accepted premise underscored by the Consensus Statement that cerebral palsy has its pathogenesis in multifactorial pathways and is not necessarily the direct result of an adverse event during labour that could have been prevented.

[39] In evaluating the expert testimony, it would have been necessary to glean if the other concerns raised by the respondent as being supposedly causal of M's cerebral palsy presented non-negligent alternatives to absolve the respondent from directional liability. But even before getting to that inquiry it was also important to establish, with the assistance of the relevant experts, whether there was evidence of neonatal encephalopathy as this, according to the Consensus Statement, if present soon after birth, favours an intrapartum insult compared to an early antenatal insult. (Confusingly the trial court's judgment suggests that there was, yet accepted the respondent's expert's view that neonatal encephalopathy was contraindicated because of M's good Apgar scores in place at 10 minutes.)

[40] For the appellant to have succeeded it was sufficient for her to have established that the inference she sought to have drawn in this respect, namely that the outcome was probably caused by birth events, was the most apparent and acceptable inference from a number of possible inferences.<sup>18</sup> A court is expected

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<sup>17</sup> *Oppelt, supra*, at [35]; *Mashongwa, supra* at [65]

<sup>18</sup> *AA Onderlinge Assuransie Bpk v De Beer* 1982 (2) SA 603 (A); *Cooper v Merchant Trade Finance Limited* 2000 (3) SA 1099 SCA at [7]; *Minister of Safety and Security v Van Duivenboden* 2002 (6) SA 431 (SCA) at para 25; and *Minister of Finance and Others v Gore* NO 2007 (1) SA 111 (SCA) at para 33.

by balance of probability to select a conclusion from amongst several conceivable ones even though that conclusion is not the only reasonable one.<sup>19</sup>

[41] Firstly, on the issue of the non-negligent alternatives contended for by the respondent, distal risk factors, according to the Consensus Statement, entail those that exert a pathogenic effect on foetal brain development starting at a time that is remote from the onset of irreversible brain injury. Examples include genetic abnormalities, environmental and sociodemographic factors, and some placental abnormalities. Proximal risk factors are those that exert pathogenic effect on foetal brain development at a time that closely predates or coincides with the onset of irreversible brain injury. Examples include *abruptio placentae*, chorioamnionitis and twin to twin transfusions.

[42] The template put forward by the Task Team of the American College of Obstetricians and Gynecologists (“ACOG”) on Neonatal Encephalopathy was according to the common expert testimony designed (and evolved over the past fifteen years) to examine the causal relationship between intrapartum hypoxic events and cerebral palsy, particularly so it seems to provide guidelines in forensic settings.

[43] The second edition of the Consensus Statement recognizes that there are multiple causal pathways that lead to cerebral palsy in term infants and that a broader perspective (by the consulting specialists) is now necessary before attributing neonatal encephalopathy to an intrapartum event. Thus, it recommends presently that:

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<sup>19</sup> *Govan v Skidmore* 1952 (1) SA 732 (N) at 734 C-E.

*“a comprehensive multidimensional assessment be performed of neonatal status and all potential contributing factors including maternal medical history, obstetric antecedents, intrapartum factors and placental pathology.”*

[44] The paediatricians who testified (Dr. Kara on behalf of the appellant and Prof. Bolton on behalf of the respondent) deferred to the updated Consensus Statement published by the American College of Obstetricians and Gynecologists (the Second Edition)<sup>20</sup> both regarding what ought to be taken into consideration before ascribing an intrapartum event as the likely cause for neonatal encephalopathy as well as the warning sounded that the specialists need to go beyond the radiological perspective by comprehensively assessing the relevant clinical context.

[45] Neonatal encephalopathy according to the Consensus Statement is a clinically defined syndrome of disturbed neurological function in the earliest days of life in the term infant, manifested by difficulty with initiating and maintaining respiration, depression of tone and reflexes, sub normal level of consciousness and often seizures, usually affecting the full term infant.

[46] The term “*neonatal encephalopathy*” is preferred to hypoxic ischemic encephalopathy (HIE) as it is not always possible to document a significant hypoxic-ischemic insult and also because there are potentially several other causes for it like metabolic disease, infection, drug exposure, nervous system malformation etc. Investigation depends on the clinical presentation.

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<sup>20</sup> ACOG/AAP, Neonatal encephalopathy and neurological outcome, Second Edition, Pediatrics 2014



[47] HIE is a subgroup of neonatal encephalopathy. To consider HIE to have occurred in the intrapartum period, there has to be evidence of neonatal encephalopathy, but before attributing the cause of neonatal encephalopathy, one has also to consider the probability of other conditions that may cause an encephalopathy. Other indicia of HIE relate to Apgar scoring, umbilical artery cord PH, the presence of multisystem organ failure consistent with the condition, fetal heart rate patterns, neuroimaging studies, developmental outcome etc.

[48] In the present matter the experts were divided on the question of whether the Consensus Statement template was a fit for M to implicate intrapartum hypoxia having a causal connection with the harm suffered by him.

[49] The correct approach to the evaluation of conflicting experts opinions offered to a court to assist it in determining an issue does not involve considerations of their credibility but rather entails an examination of the opinions presented and the analysis of their *essential reasoning*, preparatory to the court reaching its own conclusion on the issues raised.<sup>21</sup>

[50] What is further required in such evaluation is to determine whether and to what extent the opinions advanced are founded on logical reasoning.<sup>22</sup>

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<sup>21</sup> *Michael and Another v Linksfeld Park Clinic (Pty) Ltd and Another* 2001 (3) SA 1188 at [34] – [40] and *Oppelt supra* at [36].

<sup>22</sup> *Michael supra* at [36]

[51] The Supreme Court of Appeal in *Oppelt*,<sup>23</sup> with reference to *Micheal*,<sup>24</sup> summarised the general thrust of the approach to be adopted in medical negligence cases (adopted from English law) as follows:

*“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’ (at 241G-242B). 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 (at 242H).*

*A defendant can properly be held liable, despite the support of a body of professional opinion sanctioning the conduct in issue, if that body of opinion is not capable of withstanding logical analysis and is therefore not reasonable. However, it will very seldom be right to conclude that views genuinely held by a competent expert are unreasonable. The assessment of medical risks and benefits is a matter of clinical judgment which the court would not normally be able to make without expert evidence and it would be wrong to decide a case by simple preference where there are conflicting views on either side, both capable of logical support. Only where expert opinion cannot be logically supported at all will it fail to provide ‘the benchmark by reference to which the defendant’s conduct falls to be assessed’ (at 243A-E).*

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*This essential difference between the scientific and the judicial measure of proof was aptly highlighted by the House of Lords in the Scottish case of *Dingley v The Chief Constable, Strathclyde Police* 2000 SC (HL) 77 and the warning given at 89D-E that:*

*‘[O]ne cannot entirely discount the risk that by immersing himself in every detail and by looking deeply into the minds of the experts, a judge may be seduced into a*

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<sup>23</sup> *Supra*

<sup>24</sup> *Supra*

*position where he applies to the expert evidence the standards which the expert himself will apply to the question whether a particular thesis has been proved or disproved – instead of assessing, as a judge must do, where the balance of probabilities lies on a review of the whole of the evidence.”<sup>25</sup>*

[52] The authorities referenced above recognize that there is at play both clinical judgement on issues within the expert’s peculiar domain and the court’s own independent judgement on what must be established for a delictual claim on the appropriate standard of proof. It goes without saying that an opinion of an expert does not necessarily bind a court but its value is in assisting it to come to an informed view, particularly in a specialized field where the court lacks the necessary expertise.

[53] In *VN obo PN v MEC for Health, Eastern Cape*<sup>26</sup> the court in this Division also noted the following warnings regarding the evaluation of expert testimony when there is a conflict of opinions:

*“... the evaluation of expert opinion in determining its probative value and the considerations relevant thereto, are determined by the nature of the conflict in the opinion, and the context provided by all the evidence and the issues which the court is asked to determine. In general, it is important to bear in mind that it is ultimately the task of the court to determine the probative value of expert evidence placed before it and to make its own finding with regards to the issues raised.<sup>27</sup> Faced with a conflict in the expert testimony of the opposing parties, the court is required to justify its preference for one opinion over another by a careful evaluation thereof. Further, the primary function of an expert witness is to guide the court to a correct decision on questions which fall*

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<sup>25</sup> At [36].

<sup>26</sup> [2021] ZAECPHC 50 (31 August 2021) at para [34].

<sup>27</sup> *Van Wyk v Lewis* 1924 AD 438 at 447 and *S v Gouws* 1967 (4) SA 527 (E) at 528D. See also *Schmidt and Rademeyer op cit* at page 17 – 16.

*within that expert's specialised field. To that extent, the expert witness has a duty to provide the court with abstract or general knowledge concerning his or her discipline, and the criteria, necessary to enable the court to form its own independent judgment by the application of the criteria to the facts proved in evidence.<sup>28</sup> Accordingly, the mere "... pitting of one hypothesis against another does not constitute the discharge of the functions of an expert."<sup>29</sup> Finally, it is not the function of the court to develop its own theory or thesis and to introduce on its own accord evidence that is otherwise founded on special knowledge and skill.<sup>30</sup>*

[54] The trial court deviated from the standard approached indicated above when the opinions of the two experts who testified on behalf of the appellant were dismissed by the broad statement that they were not of any value or failed to take account of all the facts or because they "*excluded*" the distal and proximal factors.

[55] There is further an absence in the trial court's judgment for any real justification for its preference for the respondent's experts' opinions over the appellant's regarding the respondent's theory that M's cerebral palsy was more likely the cause of something other than a hypoxic ischemic injury that had occurred perinatally.

[56] There was no real dispute between the experts as to the underlying factual bases forming the premise for their application of the accepted criteria.

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<sup>28</sup> See the authorities referred to in *Stacey supra* at 348 to 359 F. See also *AM and Another v MEC for Health supra* at para [17].

<sup>29</sup> *Stacey supra* at 350 G-H.

<sup>30</sup> *MEC for Health, Eastern Cape v ZM obo LM* (576/2019) ZASCA 160 (14 December 2020) at paras [12] and [13].

[57] Where their variance lay was instead in their views on the impact of the relevant events or happenstances pertaining to the question of the timing of the insult that would inform the question of causal negligence.

[58] It was not in issue, for example, firstly that the appellant suffered a significant weight loss (between 9-10 kgs) in the third trimester of her pregnancy.

[59] Further, even though the appellant seemed to have been nescient of this, it was not in contention that she presented in labour with pyrexia and her foetus with foetal tachycardia. Indeed, this was the pleaded premise on which the respondent acknowledged it became seized of the management of the appellant's labour.

[60] Also, it was not in dispute that the recorded measurement of M's head circumference at birth was 32cm, this being the only perinatal *indicia* in the hospital records referencing his head size.<sup>31</sup>

[61] The significance of M's head measurement ties in with a related aspect which is the last date of the appellant's menstrual cycle before her pregnancy commenced. This speaks to the period of gestation and laterally to the question whether M's head was in fact small in relation to how far along the appellant was in her pregnancy at the time she birthed M. Testifying on behalf of the respondent, Prof. Bolton's view that M's head was microcephalic for a *term* pregnancy which

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<sup>31</sup> The first suggestion of a concern that his head was abnormally small was only noted in M's Road to Health Chart ("RTHC"), months after his birth.

he put at 40 weeks - even though this was inconsistent with her last menstrual period and expected date of delivery noted in the appellant's antenatal records,<sup>32</sup> and could have accounted for a brain injury suffered before his birth, underpinned his opinion that an earlier pathway existed for M's cerebral palsy.<sup>33</sup> Dr. Kara was more inclined to opine that M was born at 36 weeks (based on the same antenatal records that the respondent's experts referenced) and that the size of his head at birth, if it had been measured correctly, was therefore not abnormal, neither indicative of an earlier insult to his brain.

[62] Even though there was some contention about the appellant's expected date of delivery (which would speak to M's gestational age at birth) that might give a more probable indication whether there was congenital microcephaly, I point out that Dr. Kara readily conceded that M might well have been born at term and that his head would in that case then obviously have been smaller than expected. He also readily conceded the possibility of M having suffered an earlier injury (manifest in the possibility of a small brain that had stopped growing) but explained why and how that injury must have been survived by M up to the date of his delivery.

[63] In the light of Dr. Kara's willingness to go along with the respondent's very firm contention that M was born microcephalic, there was no real factual "*dispute*"

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<sup>32</sup> He conceded in his testimony that the antenatal records did not support a 40 week gestation. He was however in court when the appellant testified and heard her say that she had delivered at 9 months. The trial court had questioned the appellant about this when she testified and although she was certain that she must have been at full term in the end she relented that she might have been mistaken in this respect. The appellant's credibility was never an issue at the trial.

<sup>33</sup> Microcephaly is a condition that pertains when a baby's head is significantly smaller than expected, often due to abnormal brain development. Causes of microcephaly include infection, malnutrition, or exposure to toxins.

that fell to be resolved about the date of the appellant's last menstrual cycle (translated to M's gestational age) and whether M's head was smaller than was to be expected when he was born.

[64] The court correctly noted that the weight of the appellant's case concerned the experts' reliance on the existence of neonatal encephalopathy for the contention that there was a perinatal hypoxic ischemic injury. The answer to the question whether such encephalopathy was present in this instance (at least from a scientific perspective) involved an application of the accepted data or information (as provided by the appellant and supplemented by the maternity records such as they spoke to the clinical context) tested against the Consensus Statement template.

[65] The respondent's experts accepted M's ultimate diagnosis of mixed cerebral palsy (spastic with some dyskinesia) and blindness yet resisted the appellant's experts' opinion that his condition could be ascribed to birth related events (that would causally implicate the negligent intrapartum care contended for) rather than the other causes contended for on behalf of the respondent.

[66] In this respect Dr. Kara especially relied on the existence of neonatal encephalopathy for the contention that the hypoxic ischaemic injury had occurred perinatally, this borne out by the appellant's history supplemented by the hospital's maternity case records where they were completed and by reasoning inferentially where the records were negligible or deficient.

[67] It is necessary to recap some of the features of the hospital records that concern our present enquiry. The Summary of Labour confirms that on natural vaginal delivery, M required resuscitation. He was suctioned and given oxygen by Ambu-bag. Afterwards he was admitted to the ward due to “*respiratory distress*”. In the ward he received oxygen, but as indicated above there was an absence of any notes regarding his treatment for the next 23 days. The First Examination of Neonate form amongst the hospital’s maternity records indicates that M was “*well*” but this is evidently contrasted by the assessment at the footnote of the sheet that he “*didn’t cry at birth*” and that he was admitted “*at ward O2 for RD*”, meaning that oxygen was administered to him for respiratory distress.

[68] He was also noted under the category of “*sick*” (in contradiction to the note that he was “*well*”) to have had “*caput ++*” concerning the shape of his head. (Dr Kara explained that the swelling would have been occasioned to M’s head as he descended into the birth canal. This would have been due to pressure and accumulation of fluid on the head. In his view it indicates relative difficulty in the delivery although this is not always abnormal.)

[69] According to the Assessment of Newborn form, at birth M weighed 3.28 kilograms. His length was 49cm and his head circumference 32cms. (It is the later observation and after-the-fact diagnosis of microcephaly that underpins the respondent’s contention that his brain damage was congenital rendering it less likely that the injury had happened perinatally and, by obvious implication, as a result of intrapartum negligence).



[70] M's APGAR scores were recorded as 6/10, 7/10 and 8/10 at one, five and ten minutes respectively.

[71] The related APGAR assessment records that M had a "*a vigorous cry*" at 10 minutes<sup>34</sup> in response to stimulation. Respiration and muscle tone were scored at "1" at ten minutes, indicative of "*slow or irregular*" breathing and "*slight flexion*" as far as his tone was concerned.

[72] The interpretation of these scores in relation to M's birth was contentious as between the experts. I will return to this aspect later as it has a peculiar bearing on the issue of whether M could be said to have exhibited neonatal encephalopathy according to the criteria postulated by the Consensus Statement.

[73] The Summary of Labour also records that the feeding of M was not initiated at birth because he was admitted at the ward, on oxygen, for "*RD*", meaning respiratory distress.

[74] After birth M remained in hospital for three weeks. It is common cause though that the treatment administered to him during this period is not accounted for in the hospital records.

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<sup>34</sup> This is not a factual assertion, namely that he had a vigorous cry, but a prepopulated answer to the question how he responded to stimulation. On the APGAR scoresheet "*no response*" would score a zero point, a "*grimace*" a score of one point, and a "*vigorous cry*" two points.

[75] In M's Road to Health Chart ("RTHC") the Sinqumeni Clinic noted on 16 October 2014 that M was not growing well, that he was not reaching his development milestones and that his head circumference was not normal. The appellant had also reported to the staff a history of him having fits after delivery. For this reason the appellant was referred to the Red Cross Hospital for further management and assessment of M.

[76] On 29 January 2015 the Red Cross Hospital Clinic, with reference to the appellant's history leaning toward neonatal encephalopathy at birth (including seizures, two weeks in hospital, one in the intensive care unit), made a diagnosis that M had features of evolving spastic quadriplegia. A CT scan was scheduled to review the extent of injury noted.

[77] The ensuing CT scan confirmed his injuries to have been "*cystic encephalomalacia with profound volume loss in keeping with a perinatal insult.*"

[78] The nature and extent of M's injuries was professionally assessed at the instance of the respondent in the course of preparing for trial by a radiologist, Dr. Tracy Westgarth-Taylor on whose findings both parties relied. Dr. Westgarth-Taylor confirmed the initial assessment by the appellant's radiologist, Dr. Thina Twetwa, of gross hypoxic ischaemic encephalopathy ("HIE"), although the latter opined that the HIE's sequelae was in keeping with a prolonged partial insult to M's brain.

[79] Dr. Westgarth-Taylor recorded the following comment with reference to the radiological picture upon analysis of the MRI scan of M's brain:

*“Finding suggests previous hypoxic ischemic injury in a term infant, most likely a combination of partial prolonged hypoxic ischemic injury and acute profound hypoxic ischemic injury. The associated occipital and extensive thalamic involvement may indicate super added neonatal hypoglycaemia.”*

[80] Thus, the starting premise at the trial was that the picture of the brain injury as indicated on the MRI scan was in keeping with an injury sustained during the labour process. It also by necessary implication suggested a known pathway for cerebral palsy. As for the mechanism of the injury the scan shows that there was first a gradual reduction in oxygen and a terminal sudden drop that happened after a partial injury occurred.

[81] Further, and quite significantly, the MRI scan, which was accepted by the parties, contra-indicates any other cause for M's cerebral palsy. It notes that there is no evidence of previous infection in the radiological picture of M's brain and, significantly, no congenital abnormality is identified. (Indeed, the expert paediatricians confirmed in a joint minute that the MRI scan did not record any neurocutaneous feature or features of dysmorphology.)

[82] The next question concerns the presence of neonatal encephalopathy.

[83] Prof. Bolton reluctantly conceded ultimately that there were signs of neonatal encephalopathy yet when he testified, he maintained that the Apgar scores were to be interpreted in favour of an argument against a compromised baby at birth. He also referenced an absence of any clinical notes underscoring the appellant's case that M was unwell after being birthed.

[84] Dr. Kara questioned whether the “good” Apgar scoring in the hospital's maternity properly reflected the true situation given their appearance that there was neurological impairment at birth noting that M was resuscitated, had slow and irregular respiration and abnormal tone at 10 minutes after birth following resuscitation by manual ventilation. (That M was neurologically depressed at birth was also drawn from the history given by the appellant.) Prof Bolton conversely insisted that the Apgar score at 10 minutes “*speak for itself*”. That is, the score was 8/10 (1/2 for respiration and tone) which he considered to be a satisfactory one customarily predictive of a favourable outcome in 85% of babies.

[85] Dr. Kara was careful to note that the Apgar scoring is not a reliable basis upon which to confirm the presence or absence of neonatal encephalopathy because the scoring is subjective. This is confirmed by the Consensus Statement itself that warns that:

*“The Apgar scores are known to be subjective, are affected by resuscitation and are in itself not to be used to confirm or refute the presence or absence of intrapartum hypoxic ischemic injury. When a Category I (normal) or Category II (indeterminate) fetal heart rate tracing is associated with Apgar scores of 7 or higher at 5 minutes, a normal*

*umbilical cord arterial blood pH (+\_1 standard deviation), or both, it is not consistent with an acute hypoxic-ischemic event. (REF 3).*

[86] Dr. Kara opined that M's Apgar score of <7 did not predict that an acute hypoxic ischemic event was unlikely without evidence of normal foetal heart rate traces and without evidence of normal blood gases. (It is common cause that these were not available but the evidence suggested that M must have been in foetal distress to have come out as he did and if blood gases were done given all the other indiciae it would probably have been reflective of the fact that M was not breathing at birth and had respiratory distress.)

[87] Prof. Bolton disagreed, relying for his opinion that M was relatively well after birth on an assertion in the Consensus Statement that "*if the Apgar score at 5 minutes is greater than or equal to 7, it is unlikely that **peri-partum** hypoxia-ischemia played a major role in causing neonatal encephalopathy*".

[88] Notwithstanding Prof. Bolton's reservation aforesaid, he ultimately conceded that there was reason to suspect that a moderately severe neonatal encephalopathy had existed with reference at least to the appellant's history of M having suffered convulsions at birth, poor suck, poor cry, and a twenty-three day admission to the hospital. Prof. Bolton's only qualification in this respect is that no contemporaneous neonatal records were available to speak to the clinical context, a feature of the case perfectly known to all concerned.

[89] I digress briefly to deal with the issue of M's microcephaly which the respondent's experts sought to persuade the trial court pointed away from the hypoxic ischemic insult having been sustained intrapartum.

[90] The evidence of Prof. Christianson, a pediatrician with a sub-specialty in genetics who testified on behalf of the respondent, was somewhat of a damp squib. The expectation was that he would say that M's abnormal head circumference (first commented on 16 October 2014 when M was examined at the Sinqumeni Clinic seven months after his birth) warranted a diagnosis of "*congenital microcephaly*" (based on the premise that he was born at full term) but even before giving his testimony, he had foreshadowed that coming to a comprehensive opinion "*was frustrated by the absence of a medico-legal report on the MRI*". He noted in his expert summary that the lack of such a report meant that it could not be confirmed that M had suffered a hypoxic ischemic injury, its nature or if there are other cerebral abnormalities.

[91] However, a look at the report of Dr. Westgarth-Taylor, which the parties accepted at the trial, indeed confirms the radiologist's view that the injury seen on the MRI scan was one probably sustained during the labour process.

[92] Prof. Christianson in his testimony ultimately was obliged to defer to the opinion of the radiologists and at best could suggest that the cause for M's small head premised on a birth at term was that microcephaly was present during the applicant's pregnancy. But even accepting this as a premise, he agreed that this

could have been a cause of some of M's clinical problems or a distal risk factor for him developing hypoxic ischemic injury in the labour and delivery, in other words that such earlier compromise contended for would have rendered him more susceptible to intrapartum asphyxia. Thus, the congenital microcephaly, assuming it to have established itself at a time before M's birth was less probably a *cause* (sic) for the cerebral palsy, although it could have been a contributing factor in priming M's brain for the ultimate insult.

[93] With reference to the history of encephalopathy after birth, M's prolonged hospital admission, his spastic quadriplegia, the reasonable exclusion of other causes of encephalopathy at birth and the lack of evidence of intrauterine growth restriction, Dr. Kara noted that it is probable that the injury recorded on the scan occurred during the intrapartum period. Prof. Bolton disagreed that these features reasonably timed the brain injury to have occurred at birth.

[94] Regarding the so-called antenatal or maternity factors, Dr. Kara noted that the weight loss of the appellant in pregnancy did not concern those treating her at the clinic. Prof. Bolton agreed that they treated her routinely despite her obvious decrease in body mass. Further, not only did they not note any concern about it but they made no onward referral because of it. The foetal growth rate was also recorded as normal on 12 February 2014 when the appellant's 9kg weight loss was recorded. I add that although the appellant testified that it worried her that she was losing weight, she stated that she experienced a normal pregnancy and was not unwell.

[95] Dr. Kara reasoned that although there had been a significant weight loss, he was satisfied that by taking a measurement of the appellant's upper arm circumference, the clinic had ruled out that she was malnourished. In any event, so he assured the trial court, M was born at a normal weight of 3.2kg.

[96] Concerning the appellant's presentation on admission in labour, both experts noted that there is no record of any treatment given by the hospital for the pyrexia or infection first diagnosed at the Phola Clinic (the ostensible reason for the referral of the appellant to the hospital), but both assumed that the appellant had probably been treated for the infection and the high temperature she had initially presented with. Dr. Kara acknowledged that both these features posed significant risk factors in the birth process but contended that they were not likely to have been a *causal* factor for the cerebral palsy. Prof. Bolton on the other hand contended that fever in labour is *associated* with a variety of poor outcomes for a foetus including poor neurological outcomes, but again the MRI picture excluded any infective cause for M's cerebral palsy.

[97] The trial court appeared to fallaciously equate the absence of the neonatal records with there have been no comprehensive assessment undertaken as if that were the end of the matter for the appellant, missing the obvious implication that the informed views of the experts purported to stand in substitution of that lack. Further, whilst noting the appreciable help that was to be expected of the opinion from a skilled witness, the trial court instead resorted to its own speculation concerning the imponderables and boundless possibilities as to what could have caused M's cerebral palsy including the ingestion of the appellant of church water



during her pregnancy, in the process also in my view losing sight of the fact that there was no obligation on the appellant to prove the exact cause of the cerebral palsy but rather to establish the element of causal negligence on a balance of probabilities.

[98] Dr. Kara, although himself lamenting the pithy state of the hospital records (which the National Health Act, No. 61 of 2003 behooves its staff to have kept) and qualifying that one generally cannot time a hypoxic ischemic injury to a specific period in labour except in the event of a sentinel event (a principle often repeated in our courts in these matters) yet went on to reason, in my view to the contrary quite cogently, why he believed that the insult to M's brain (accounted for on the MRI scan) was probably causally connected to the hospital's staff failure to have monitored the appellant by CTG on a continuous basis until M was born.

[99] I find myself constrained to mention the main features of his opinion which, far from falling to be rejected as lacking in reason or logic, in my view presents coherently, comprehensively and takes account of the array of concerns that were pleaded by the respondent as non-negligent alternatives for M's cerebral palsy.

[100] In acquitting himself of his mandate to identify what caused M's injury and its timing in the context of all the evidence, Dr. Kara's essential reasoning is that M was born in a compromised condition and required significant support. Firstly the resuscitation was not routine, but a vigorous one. Secondly, M was admitted into the neonatal unit for respiratory distress rather than given to his mother and breastfeeding was not initiated as it would with a baby that was well.

[101] He did not consider the Apgar score of eight at 10 minutes to be an accurate reflection of M's condition because he had slow or irregular respiration at 10 minutes that was inconsistent with him having had a "*vigorous cry*". He also questioned whether the first examination of neonate form indicating that M was "well" could be accurate. This is because M had Caput ++ and was admitted to the ward for respiratory distress consistent with the single entry in the notes 12 hours after he was born that he was in the ward on oxygen. As for M's long admission in hospital until 28 March 2014 he opined that this strongly supported the probability of some serious event having occurred in the neonatal period.

[102] He pointed out that M's CT scan confirmed a finding in keeping with a perinatal assault. He added his view that the commonest cause by far for cystic encephalomalacia noted on the scan is a hypoxic injury. He explained that these cysts in this picture arise in the soft fluid areas in the brain where the tissue is dead because of a lack of oxygen or blood supply to those tissues. He reasoned that although the causes for a lack of oxygen to a foetal brain is multifactorial, the commonest cause is hypoxic ischaemia given that labour is a hypoxic-centric event.

[103] He confirmed that M's earlier diagnosis of spastic quadriplegia CP together with the results of the CT scan was in line with his own clinical diagnosis of mixed cerebral palsy that followed a natural evolution of M's condition. This, together with the CT scan findings and the history of events at birth, all tied in to link the injury to one sustained during the labour process.

[104] On the issue of other causes of encephalopathy at birth or altered neurological status after birth, he ruled out meningitis because this is not consistent with the radiological picture shown on the MRI scan. He also confirmed the absence of any brain abnormality as in a congenital brain abnormality. He further alluded to the absence of any evidence that M had a severe overwhelming infection that could cause shock (sepsis) and have damaged the brain. He pointed out additionally that there was no evidence that M had a chromosomal abnormality or a syndrome or metabolic condition that could have caused his brain to be damaged. There was also an absence of any record of obstetric events affecting the appellant's placenta because M was not born growth restricted.

[105] He dismissed the chance that the appellant's consumption of church water could have entailed the ingestion of toxins because M was normally grown at birth at a weight of 3.2 kgs. In his view a severe maternal sepsis was unlikely given that the appellant was discharged from hospital the day after M was born.

[106] He exhaustively dealt with every other probable cause for the cerebral palsy as contended for by the respondent. He pointed out that the appellant had no known genetic or predisposing risk factors for cerebral palsy. As for her fever upon admission he noted that this was not going to cause fetal brain injury of the hypoxic ischemic variety.

[107] He readily acknowledged however that it might obviously have increased the risk of asphyxia during labour but was not the cause for M's cerebral palsy. This is exactly why in his view the obstetrician had to adequately manage her labour in the light of this risk factor.

[108] As for the possibility that M was born with microcephaly, Dr. Kara opined that it was unlikely that his brain had been injured prior to delivery. He explained firstly that a newborn with a head size of about 32 centimetres is the average head size for a baby of about 34 to 35 weeks gestation. Therefore, assuming a birth at 38 to 40 weeks, this would hypothetically mean that the brain injury would have happened prior to 36 or 37 weeks, thus two or three weeks earlier than M's delivery. If this had happened, he opined that M would not have come out in a condition of encephalopathy. Further, if he had survived the brain injury (meaning that certain brain areas would not have been compromised at the time of delivery) there was no reason why he would not have been born in a reasonable, normal condition. Instead, all the indications were of a neonatal encephalopathy which is the commonest injury occurring during labour.

[109] As for the extent of M's neonatal encephalopathy he graded it as moderately severe that lasted several days noting poor feeding and poor cry on history.

[110] Adverting to the MRI scan he emphasised that both experts had found that it shows features of a hypoxic ischemic brain injury, making it fair to argue that M's cerebral palsy was caused by such injury rather than any other.

[111] He added that the further finding of Dr. Westgarth-Taylor that there was a hypoglycaemic injury to the brain (which was a probability that he had pre-empted upon examining M even before seeing the MRI report in this regard) accorded with the more probable scenario that M had suffered the injury during labour. He explained that when a baby is born with hypoxic ischemia, the stores of the baby's glucose is exceptionally low. If after birth that glucose is not rapidly replenished, there is ongoing brain damage from HIE together with further brain damage from low sugar. You would not get the radiological picture on M's MRI scan, so he pointed out, in a baby whose brain was damaged a few weeks earlier. Indeed, this feature present in his MRI scan helped further in timing the injury.

[112] Asked by the trial court to venture a time when the injury occurred, he put this at a juncture between the appellant's admission and before delivery of M, based on the fact that the foetal condition was satisfactory on admission to the hospital. Earlier he had explained that the CTG tracings taken at the time, although suggestive of a non-reassuring foetal heart rate, had not prompted an immediate delivery of M by caesarean section. For the rest, he deferred to the obstetricians who might further be able to narrow down the period.

[113] On the issue of the existence of encephalopathy, Dr. Kara's opinion that the hospital notes are not a fair reflection of M's condition based on the appellant's history and the contra indications that he was well when admitted to the ward where he was hospitalized for an extensive period thereafter is preferable to one that rigidly assumes a position based on a subjective Apgar score. In any event the

Consensus Statement postulates a wholistic view of all the clinical features rather than a formalistic approach.

[114] Prof. Bolton remained adamant when he testified that M had been birthed at full term and that his head circumference was below the 3<sup>rd</sup> percentile for a full term infant, assessed as a microcephalic head. He adverted to a chart and an elaborate process to assess gestational age to demonstrate how he got to this complicated conclusion, in the process eschewing the plain old way of accepting the entries in the appellant's antenatal records concerning when she had her last menstrual period and what her expected date of delivery was.

[115] This informed his view that M's head growth was abnormal at birth and presumably before birth.

[116] Put to him that the appellant's last menstrual period was in fact recorded in her antenatal records he conceded as much:

*“Yes, that was true and I looked at that again and thought why would I have said that and I realised that I said it because I could not read what was written there, it has been crossed out, so it is incorrect, it was recorded, but it was illegible, i am sorry.”*

[117] Expecting that he would then concede that M was “before term” at birth he latched on to the appellant's testimony that he had overheard her give in court that her last menstrual period was in June 2014. Asked by the trial court to instead

focus on what the medical records provide and put to him that Dr Ebrahim's opinion was that the appellant was not full term when she gave birth to M, he said; "yes, I presume that is true" but added that he could not now work it out in the absence of his "wheel" that he uses for these purposes. Finally referred to the appellant's gestation window provided for in her antenatal records, he relented and deferred to the obstetricians. He conceded, as was put to him in conclusion by counsel on this issue that his evidence of the gestation was "on a different plane."

[118] He was equivocal too about the impact of the appellant's weight loss on her pregnancy:

*"What concerned me here was that it got less. And all I think this was indicative of was that the weight loss that was recorded by the scales was probably correct, something was going on, but I think beyond that I would not make anything of it. I'm not going to say but that told me that the mother was malnourished, or she was not. She went from 28 to 24 and I think as I recall both of those, 24 is just above the normal limit where they diagnose malnutrition. If I'm right it is 23 where they say that it is the indicator of malnutrition, what concerned me and I tried to put it in the report was that it dropped and so that it indicated to me that perhaps something had happened, but beyond that I would not be able to comment."*

[119] His rigid approach to identifying distal and proximal events on a trajectory towards cerebral palsy is not of any real assistance to a court engaging with a factual causation enquiry on a balance of probabilities. Ultimately, he appeared to accept that fetal priming (read both distal and proximal risk factors according to the Consensus Statement) can predispose a fetus in labour to become hypoxic

because the foetus is not set up to be able to tolerate (the stresses of labour) properly:

*“But there were also significant proximal and distal risk factors which played a role in the causation of the brain damage and I thought B was the more likely thing, that there were distal factors and proximal risk factors. We haven't talked about her weight loss in pregnancy. That was very unusual period that possibly played a role, was probably a distal risk factor. Then the infection during the labour, the fever during the labour, that was a proximal risk factor. Then there may well have been an intrapartum event, i.e. that a normal baby going through labour became hypoxic. It's absolutely normal, but this baby was set up to not be able to tolerate it properly. So from that other risk factors and that's what happened. So that's why I said there would be these various possible- and it's just possible and probable ways and I thought the probability that they were multiple risk factors, distal and proximal, which then set the baby up for hypoxic event- or not an event, but that the hypoxemia during the labour- the normal hypoxia during labour, was working on a sensitised brain that had these things as risk factors. And that's the multi-dimensional factors of cerebral palsy.*

...

*But if you've got a baby who's got- who set up by perhaps a damage to the developing brain during the pregnancy, when she has a weight loss, I'm postulating, plus an infection, plus an inflammatory- a temperature, now a normal contraction, not something that was abnormal, it's got to happen to push the baby out, might have affected the baby whose brain was set up and that's the complex issues.”*

[120] Having regard to the factors that he perceived played a role in M's condition he watered down some of his starting premises. Regarding the appellant's weight loss during pregnancy, for example, he acknowledged that she had not been sick and then equivocated:



*“...that 10 kg’ worries me, so I looked at it and thought was this an event, whatever it was which may have affected fetal growth including fetal brain growth. And I speculated and it is speculation but that is a possible cause for the microcephaly.”*

[121] Asked under cross examination about the findings of Dr. Westgarth-Taylor’s findings he agreed that her additional comment about the hypoglycemic damage was not in line with the features of microcephaly. He appeared to concede though that the cause of the microcephaly could be represented in the picture seen on the MRI scan, adding that the radiologist was “*probably right*” that it looks more like the damage of hypoxic ischemia than anything else as the cause for it. He then went on to suggest that the injury could be the effect of an acute profound injury on an abnormal brain that is vulnerable but turned the order of things around, suggesting that it was unlikely that this (the already injured brain manifest in the small head) was “*not contributed to by hypoxic ischemia in the perinatal period*”. This is simply another way of saying that a supposed prior injury would render an already compromised foetus susceptible to a hypoxic ischemic injury during labour. It also presupposes that appropriate monitoring may prevent further injury from happening.

[122] I mention finally that the respondent’s obstetrician expert, Dr. Marishana, accepted that M had presented as one in respiratory distress and also that his cerebral palsy was caused by a hypoxic ischaemic event one way or another.

[123] It is ironic in my view that the very risk factors contended for by the respondent would have elevated the need for closer monitoring of the appellant

during her labour given the after-the-fact MRI finding of M having sustained a combination partial prolonged and acute profound injury. This radiological picture suggests that there was some damage before the acute injury occurred and the experts were all in agreement that an infection in the mother does make the foetus more susceptible to hypoxia.

[124] It is exactly the cumulative consequence of the factors at play in this matter as foetal priming as it were that would have rendered M more susceptible to asphyxia during labour. There was no pushback from the appellant's experts that all of these contenders (at least those that she presented with upon admission) were significantly at play during her labour.

[125] In my view the evidence establishes on the requisite standard of proof that the history of M was one of being neurologically depressed at birth with all the related indications suitably pointing to a neonatal encephalopathy (including the findings of the MRI scan showing features of a partial prolonged and acute injury resulting from a cumulative deprivation of oxygen over time) and that the intrapartum negligence is causally connected with his cerebral palsy.

[126] This outcome, so it appears to have been agreed between the expert obstetricians, was foreseeable because it all came down to monitoring and preventable in the sense that the early signs of foetal distress could have been detected and acted upon appropriately by expediting the vaginal delivery.

[127] In the premises the appeal should succeed and the appellant should be entitled to her costs of the action as well.

[128] In the result I make the following order:

1. The appeal succeeds with costs including the costs of second counsel.
2. The order of the trial court is set aside and replaced with the following order:
  - “1. *It is declared that the negligent conduct of the defendant’s employees was the cause of the minor child’s condition and the defendant is ordered to pay to the plaintiff in her representative capacity her agreed or proven damages.*
  2. *The defendant is ordered to pay the plaintiff’s costs of suit, such costs to include the fees of the experts employed for purposes of the trial together with the costs of second counsel.”*

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B HARTLE

JUDGE OF THE HIGH COURT

I AGREE,

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N W GQAMANA  
JUDGE OF THE HIGH COURT

I AGREE,

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I BANDS  
JUDGE OF THE HIGH COURT

DATE OF APPEAL : 9 October 2023

DATE OF JUDGMENT : 1 March 2024

Appearances:

*For the Appellant: Mr. H van der Linde SC together with Mr. M James instructed by  
Mgweshe Ngqeleni Inc, East London (ref. MN/Y06/16)*

*For the Respondent: Mr. P L Mokoena SC together with Mr. Y Malunga instructed by State Attorney, Makhanda (ref. Mr. Maqambayi)*