

Editorial note: Certain information has been redacted from this judgment in compliance with the law.



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

Reportable

Appeal case no. CA 6/2023

High Court case no. 827/2019

In the matter between:

N[...] K[...] on behalf of

U[...] K[...]

Appellant

and

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

Respondent

JUDGMENT

LAING J

[1] This is an appeal against the whole of the judgment of the court *a quo*, which previously dismissed the appellant's claim for damages in the amount R 28,200,000.

The cause of action arose from the alleged negligence of the medical staff at Dora Nginza Hospital, Gqeberha, during the birth of the plaintiff's son, U, on 8 March 2019.

BACKGROUND

[2] The salient details of the parties' respective cases, the trial proceedings, the findings of the court *a quo*, and the application for leave to appeal are described under the corresponding sub-headings below.

Appellant's case

[3] As plaintiff in the court *a quo*, the appellant pleaded that she was admitted to Dora Nginza Hospital on 3 March 2019 for delivery. She endured five days of prolonged labour before giving birth to U, who suffers from cerebral palsy, mental retardation, and epilepsy.

[4] It was the appellant's case that the defendant's medical staff had been negligent in their care. They had failed to, *inter alia*, properly assess and examine the plaintiff upon her admission, appropriately monitor her labour and the well-being of the foetus, and prevent U from sustaining brain damage at birth, when this could have been avoided by exercising reasonable skill and diligence. More particularly, pleaded the appellant, the medical staff had failed to, *inter alia*, detect and prevent the onset of chorioamnionitis¹ and the health complications associated therewith.

[5] As a result of the above negligence, U has endured pain, suffering, discomfort, the loss of amenities of life, and total and permanent disability. The appellant, in her

¹ Chorioamnionitis is defined as inflammation and infection of the inner and outer fetal membranes, often after pre-term premature rupture of the membranes. See Elizabeth Martin (*et al*), *Concise Medical Dictionary* (Oxford University Press, 10ed 2020), at 146.

personal capacity, pleaded that she has experienced psychological shock and trauma, limitations on her freedom, and the loss of the joys of parenthood.

[6] She claimed damages in relation to U's future medical treatment, loss of earning capacity, and both special and general damages. The claim also made provision for, *inter alia*, the costs for establishing and administering a trust to hold any damages so awarded.

Respondent's case

[7] In her plea, the defendant admitted that the plaintiff had been at the Dora Nginza Hospital but denied that she had endured a prolonged five-day period of labour. If it was found that the plaintiff had proved U's medical condition and injuries, then the defendant pleaded that these could have occurred at an antenatal stage because the plaintiff had contracted an infection or chorioamnionitis. This could have led to placental insufficiency which, in turn, resulted in asphyxiation prior to the onset of labour.

[8] The defendant denied all allegations of negligence. She asserted that the medical staff had, *inter alia*, provided such care to the plaintiff as would have been reasonably expected, including the assessment and monitoring of the plaintiff, and had acted in accordance with accepted nursing and medical practice. The defendant denied that the onset of chorioamnionitis had been foreseeable or that steps could have been taken to prevent it. Moreover, the defendant denied that there had been repeated vaginal examinations or that these had caused the onset of chorioamnionitis. She pleaded that the measurement of the umbilical cord blood gas demonstrated that U had not suffered from any fetal distress; such harm as was caused had not occurred during labour or delivery. The defendant pleaded that the neurological problems suffered by U had arisen despite the reasonable care provided by the medical staff; there was no

evidence that the pathology which had led to U's cerebral palsy had emanated from labour or that it had been preventable.

[9] Consequently, the defendant denied that there had been any cause of action. She denied that she was liable to the plaintiff for the damages claimed.

Trial proceedings

[10] Prior to the commencement of the trial, the parties agreed upon the admission of several joint minutes prepared by the relevant experts. These formed part of the trial bundle.

[11] At the trial, the plaintiff testified on her own behalf. She also relied on the evidence of an obstetrician and gynaecologist, Dr Constant Ndjapa; and a paediatric neurologist, Dr Amith Keshave. The defendant, in turn, led the evidence of an obstetrician and gynaecologist, Dr Krzysztof Janowski; and a paediatric neurologist, Dr Yavini Reddy. She also led the evidence of a clinical manager responsible for medico-legal matters at Dora Nginza Hospital, Dr Ziefred McConney.

Findings of the court *a quo*

[12] The trial court held that there were two issues for determination: the defendant's negligence and whether this had caused the harm suffered. It assumed, without making any finding, that the medical staff had indeed been negligent, and proceeded to determine the issue of causation, based on the medical evidence and the plaintiff's testimony. The trial court focused on the question of whether the cause of U's brain damage had been the onset of chorioamnionitis.

[13] Ultimately, the trial court found that there had been no evidence that sub-standard monitoring had adversely affected the baby. This was because the plaintiff had indicated that, during the evening of 7 March 2019, prior to U's birth on the following day, a nurse had checked the results of a cardiotocographic ('CTG')² reading and declared that everything had been satisfactory and that the plaintiff would shortly be required to start the process of delivery. If there had been any problem at that stage, then the nurse would have mentioned this. The plaintiff's cervix, moreover, would not yet have been fully dilated.

[14] The trial court observed that the pH level for the baby, at birth, had been normal.³ There had also been no evidence to demonstrate that reliance by the medical staff on vacuum extraction and fundal pressure had caused the brain damage. The same could be said for the administration of misoprostol, used to induce labour.

[15] It was undisputed, said the trial court, that the baby's condition had been compromised at the time of delivery. This was in keeping with the diagnosis that his brain had sustained an injury because of a significant lack of oxygen. The question to be determined was when the injury had occurred.

[16] The trial court accepted the evidence of the defendant's experts, Dr Janowski and Dr Reddy; their conclusions were based on logical reasoning and grounded in fact. The learned judge found as follows:⁴

² Cardiotocography is defined as the electronic monitoring of the fetal heart rate and the frequency of uterine contractions. *Op cit*, at 122.

³ The reading was 7.33, which, according to the experts, was within the normal range. A pH reading is a measure of the acidity or alkalinity of a solution; a pH of 7 indicates a neutral solution, below 7 indicates acidity, and above 7 indicates alkalinity. *Op cit*, at 588.

⁴ *NK on behalf of UK v MEC for Department of Health* (unreported, case no. 827/2019, Eastern Cape Division, Bhisho, delivered on 11 October 2022), at paragraphs [44] and [45]. The identities of the plaintiff and her minor child have been concealed.

'...the answer as to when the child's brain was injured lies in the histological report on the evaluation of the placenta... The histological report would not have revealed that the chorioamnionitis was acute and severe, exhibiting a fetal inflammatory response with the presence of funisitis⁵ and vasculitis⁶ unless the mother contracted it a few days or weeks before the onset of labour. That finding excludes a finding that the damage to the child's brain occurred intrapartum. In the circumstances, I find that even if it were proved that the prolonged second stage of labour, induced labour, and sub-standard monitoring may have caused damage to the child's brain, it would have happened at a stage when the child's brain had already been damaged over a few days or weeks by the insufficiency of oxygen and nutrients from the placenta, caused by chorioamnionitis.

...In other words, the probabilities indicate that the child's brain injury predated the prolonged second stage of labour, induced labour, and sub-standard monitoring. The damage to the child's brain might have caused or contributed to the plaintiff not going into labour on time, the prolonged second stage of labour, and the difficulties with the delivery. The above finding means that the plaintiff failed to show that the prolonged second stage of labour, induced labour, and sub-standard monitoring caused the damage to the brain of the plaintiff's child. That being the case, the plaintiff failed to establish that the defendant is delictually liable to her...'

[17] Consequently, the trial court dismissed the plaintiff's claim with costs. She applied for leave to appeal.

Application for leave to appeal

[18] The plaintiff listed numerous grounds upon which she based her application. She asserted that the trial court had erred in its assessment of the evidence and the balancing thereof for purposes of the determination of probabilities, especially in relation to the issue of causation.

⁵ Funisitis means inflammation of the connective tissue of the umbilical cord that occurs with chorioamnionitis. See <https://en.wikipedia.org/wiki/funisitis> (accessed on 31 January 2024).

⁶ Vasculitis is defined as a patchy inflammation of the walls of blood vessels that leads to damage and thrombosis. Martin, *op cit*, at 808.

[19] The trial court held that an appeal against its findings enjoyed reasonable prospects of success. The learned judge granted leave to appeal against its findings that: (a) the plaintiff had failed to prove that the damage done to U's brain had been caused by the negligence of the medical staff prior to and after the onset of labour; and (b) the plaintiff was liable for the defendant's costs.

Grounds of appeal

[20] The plaintiff asserts that the trial court erred in failing to find that the negligence of the medical staff in managing the intrapartum and second stage of labour had caused the injury to U's brain. It also erred in its assessment of the probabilities in relation to the impact of chorioamnionitis on the baby. The trial court, says the plaintiff, ought to have accepted her undisputed evidence that the baby had been hypoxic at birth, and that the relevant medical staff had informed her that the injury had been caused by a delay in the delivery process and an insufficient supply of oxygen to U's brain. This contradicted Dr Janowski's views. These had, in turn, been at odds with those of Dr Reddy, who had testified that the injury had been occasioned by asphyxia or hypoxia.

[21] Furthermore, contends the plaintiff, the court erred in failing to recognize the improbability that a baby with such a serious brain injury could have survived *in utero* without any indication of foetal distress. The defendant's experts had incorrectly conflated the presence of chorioamnionitis, accompanied by funisitis, with the occurrence of the injury. The trial court, argues the plaintiff, erred in failing to deal with the MRI⁷ results and the joint minute of the radiologists, who had agreed that there had been no evidence of an infective or inflammatory disease and that such a condition was

⁷ MRI or magnetic resonance imaging is defined as a diagnostic imaging technique based on the emission of electromagnetic waves from the body when the patient is placed in a strong magnetic field and exposed to radiofrequency radiation. *Op cit*, at 456.

unlikely to have been the cause of the injury. The plaintiff asserts that the trial court ought to have found that the injury had been caused by the medical staff's failure to have monitored, properly, the foetal heart rate; the plaintiff's contractions over a period of three days; and the infusion of syntocinon.⁸ It had also been as a result of their unsuccessful attempt at vacuum extraction and application of fundal pressure during a prolonged second stage of labour, contrary to the maternity guidelines.⁹

[22] The plaintiff goes on to assert that the trial court erred in accepting Dr McConney's evidence to the effect that the relevant medical records had been removed from the Dora Nginza Hospital without consent. It was incorrect to have found that the Department of Health could not have been blamed for this or the resulting paucity of information pertaining to the medical staff's care of the plaintiff and her baby.

[23] A further ground of appeal was that the trial court erred in its determination of the issue of causation without first having decided the issue of negligence. It also applied the incorrect test for causation.

[24] The plaintiff contended that the trial court erred in failing to find that the injury could have occurred intrapartum, especially when Dr Reddy had conceded that the Volpe criteria had been met.¹⁰ It ought to have rejected her reliance on the histology report regarding the condition of the placenta, considering the MRI results and the joint minute of the radiologists. The plaintiff argues, too, that the trial court erred in finding that the injury had occurred weeks prior to the birth, because of chorioamnionitis. It erred in placing reliance on the umbilical cord blood gas measurement and erred in accepting Dr Janowski's views to the effect that the CTG reading had indicated that

⁸ Syntocinon is understood to be the trade name for medication that is used to cause the contraction of the uterus to start labour, increase the speed of labour, and to stop bleeding following delivery. See [https://en.wikipedia.org/wiki/oxytocin_\(medication\)](https://en.wikipedia.org/wiki/oxytocin_(medication)) (accessed on 31 January 2024).

⁹ National Department of Health, 'Guidelines for Maternity Care in South Africa' (4ed 2015).

¹⁰ The criteria were developed by the renowned physician, Dr Joseph Volpe, of the Harvard Medical School, Boston, MA, USA. His work is often quoted in matters such as the present.

everything had been satisfactory. It also erred, says the plaintiff, in failing to consider Dr Ndjapa's views regarding the plaintiff's contractions in relation to the supply of oxygen to U's brain.

[25] On a proper weighing of the probabilities, considering the expert evidence, the trial court ought not to have dismissed the plaintiff's claim. It ought to have determined the merits and costs of the matter in her favour.

ISSUES TO BE DECIDED

[26] Inasmuch as the grounds of appeal serve to delineate the issues to be determined by this court, the questions that lie at the core of the dispute are the following: (a) whether the defendant's medical staff were negligent; and (b) whether such negligence was the cause of the injury to U's brain. The court must decide whether the trial court was correct in finding that the plaintiff had failed to prove, on a balance of probabilities, that the above questions had been answered in the affirmative.

[27] Before proceeding further, it may be helpful to reiterate the basic principles involved. This is especially so where the determination of the dispute involves the analysis of a complex matrix of fact- and opinion-based evidence.

LEGAL FRAMEWORK

[28] Regarding appeals on questions of fact, the erstwhile Appellate Division observed, in *R v Dhlumayo*,¹¹ that where there has been no misdirection on fact by a trial court, the presumption is that its conclusions are correct; the appeal court will only

¹¹ 1948 (2) SA 677 (A), at 705-6.

reverse such conclusions where it is convinced that they are wrong. Furthermore, where an appeal court is required to decide a case purely on the record, the satisfaction of the onus becomes all-important. Subsequently, in *Van Aswegen v De Clercq*,¹² the Appellate Division held that:

'[where] the trial court has reached no finding at all on the credibility of witnesses to vitally important incidents... [t]he appellate court has to do its best on such material as it has before it... The onus should not be allowed to operate in such a case unless and until, after all the relevant evidence has been examined to see whether there is a sufficient balance of probabilities on one side or the other, the state of inability to decide is reached.'

[29] The Supreme Court of Appeal, within the context of a medical negligence case such as the present, had this to say in *HAL obo MML v MEC for Health, Free State*,¹³ per Makgoka JA:

'The presumption is that a trial court's factual findings are correct in the absence of demonstrable error. To overcome this presumption, an appellant must convince the appellate court on adequate grounds that the trial court's factual findings were plainly wrong. If the appellate court is merely left in doubt as to the correctness of a factual finding, then it will uphold that finding. It is only in exceptional circumstances that an appellate court will interfere with the trial court's evaluation of oral evidence, in the light of the advantages enjoyed by the trial court of seeing, hearing and appraising the witnesses.'¹⁴

[30] The above principles pertain to appeals on questions of fact.¹⁵ In relation to questions of opinion, the Supreme Court of Appeal dealt with the role of an expert witness in *Pricewaterhousecoopers Incorporated and others v National Potato Co-operative Ltd and another*,¹⁶ where Wallis JA remarked that:

¹² 1960 (4) SA 875 (A), at 881-2.

¹³ 2022 (3) SA 571 (SCA).

¹⁴ At paragraph [72].

¹⁵ See, in general, the discussion in AC Cilliers (*et al*), *Herbstein and Van Winsen: Civil Practice of the High Courts and the Supreme Court of Appeal of South Africa* (5ed, 2009, ch39), at 1251-2.

¹⁶ [2015] 2 All SA 403 (SCA).

'Opinion evidence is admissible "when the court can receive 'appreciable help' from that witness on the particular issue".¹⁷ That will be when:

"...by reason of their special knowledge and skill, they are better qualified to draw inferences than the trier of fact. There are some subjects upon which the court is usually quite incapable of forming an opinion unassisted, and others upon which it could come to some sort of independent conclusion, but the help of an expert would be useful".¹⁸

As to the nature of an expert's opinion, in the same case, Wessels JA said:

"...an expert's opinion represents his reasoned conclusion based on certain facts or data, which are either common cause, or established by his own evidence or that of some other competent witness. Except possibly where it is not controverted, an expert's bald statement of his opinion is not of any real assistance. Proper evaluation of the opinion can only be undertaken if the process of reasoning which led to the conclusion, including the premises from which the reasoning proceeds, are disclosed by the expert."¹⁹

[31] Wallis JA went on to remark, further, that:

'Lastly when dealing with the approach to an expert witness I have found helpful the following passage from the judgment of Justice Marie St-Pierre in *Widdrington*:²⁰

"Legal principles and tools to assess credibility and reliability

[326] 'Before any weight can be given to an expert's opinion, the facts upon which the opinion is based must be found to exist.'

[327] 'As long as there is some admissible evidence on which the expert's testimony is based it cannot be ignored; but it follows that the more an expert relies on facts not in evidence, the weight given to his opinion will diminish.'

[328] An opinion based on facts not in evidence has no value for the court.

[329] With respect to its probative value, the testimony of an expert is considered in the same manner as the testimony of an ordinary witness. The court is not bound by the expert witness's opinion."

¹⁷ *Gentruco AG v Firestone SA (Pty) Ltd* 1972 (1) SA 589 (A), at 616H.

¹⁸ *Coopers (South Africa) (Pty) Ltd v Deutsche Gesellschaft für Schädlingsbekämpfung MBH* 1976 (3) SA 352 (A), at 370G-H.

¹⁹ At paragraph [97].

²⁰ The reference is to the Canadian case of *Widdrington (Estate of) v Wightman* 2011 QCCS 1788 (CanLII).

[32] The principles described above must serve as a rudimentary framework for the analysis of the fact- and opinion-based evidence that follows. The finer details of such an analysis will attract the need to have regard to the relevant case law that has emerged in the field of medical negligence.

[33] It would be salutary to revisit the test that must be applied, as explained by Corbett JA in *Blyth v Van den Heever*,²¹ where he observed:

'As I see it, this case resolves itself into three main questions: (i) what factually was the cause of the ultimate condition of appellant's arm; (ii) did negligence on the part of the respondent cause or materially contribute to this condition in the sense that respondent by the exercise of reasonable care and skill could have prevented it from developing; and (iii) if liability on the part of respondent be established, what amount should be awarded to appellant by way of damages?'

[34] The above questions are pertinent to the present matter. They provide a useful route map for the determination of the appeal. It is necessary, firstly, to consider how the injury to U's brain occurred factually, entailing an assessment of the evidence regarding the medical reasons for the injury.²² It is necessary, secondly, to confront the questions that lie at the core of the dispute: whether there was negligence on the part of the medical staff; and, if so, then whether this caused or materially contributed to the injury in circumstances where the medical staff could have prevented it by exercising reasonable care and skill.

[35] An analysis of the evidence follows.

²¹ 1980 (1) SA 191 (A), at 196E.

²² The question must be distinguished from the issue of factual causation, being one of the two primary elements of causation in the law of delict, the other being the issue of legal causation. See JR Midgley, 'Delict', in *LAWSA* (Vol 15 3ed, 31 March 2016), at paragraph 175.

AVAILABLE DOCUMENTARY EVIDENCE

[36] It would be best to commence with an acknowledgement that there was very little 'hard' evidence before the trial court. As to how much of it was admissible formed the subject of considerable debate on appeal.

[37] The available documentary evidence consisted of a Road to Health Chart, the MRI results that informed the radiological reports, the histopathology report in relation to the plaintiff's placenta, and the arterial blood gas ('ABG') analysis.²³ During argument, the plaintiff's counsel contended that the defendant, at trial, proved neither the histopathology report nor the ABG analysis because she failed to qualify or lead the experts responsible for the compilation thereof. Consequently, the documents in question amounted to hearsay and ought to have been treated as having been of no evidential value. To that effect, counsel referred to *Twine and another v Naidoo and another*,²⁴ where Vally J restated the principles applicable to the use of experts, including the requirement that his or her evidence must be capable of being tested, it must be verifiable.²⁵ In *HN v MEC for Health, KZN*,²⁶ furthermore, Koen J referred to the academic work of DT Zeffertt and AP Paizes²⁷ to emphasise that:

'[s]tatements in the medical records that are favourable to the defendant are hearsay where the author thereof was not called to testify, and hence not admissible.

...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

²³ This is understood as an assessment of the acid-base ratio or pH level in relation to arterial blood obtained from the umbilical cord.

²⁴ [2018] 1 All SA 297 (GJ).

²⁵ At paragraph 18 (q).

²⁶ (1287/2014) [2018] ZAKZPHC 8 (4 April 2018).

²⁷ DT Zeffertt and AP Paizes, *Hoffman and Zeffertt's The South African Law of Evidence* (4ed), at 183ff.

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.¹²⁸

[38] In contrast, the counsel for the defendant asserted that both the histopathology report and the ABG analysis had been discovered or had formed part of the trial bundle. They had been considered by the various experts involved and had featured extensively, either in evidence or argument, throughout the duration of the trial. From the record, it is not apparent that there has ever been a dispute about the admissibility of either document.

[39] The plaintiff's grounds of appeal made no mention of this. Counsel referred to *S v Waldeck*,²⁹ where Kgomo JP remarked:

'[o]n a conspectus of the evidence that has been dealt with hereinbefore, some lesser aspects not specifically, I have no reservation whatsoever that, although the defence did not expressly agree to the admission of the hearsay evidence, the record is replete with evidence to support the view that the defence has by conduct agreed or acquiesced in the State's procuring the hearsay evidence, and that the State was entitled to conduct its case on that basis.'³⁰

[40] The legislative mechanism by which hearsay evidence can be admitted is section 3 of the Law of Evidence Amendment Act 45 of 1988. The relevant provisions permit admission when a party against whom the evidence is to be adduced agrees thereto. This pertains to both criminal and civil proceedings.³¹

²⁸ *HN*, at paragraphs [8] and [9].

²⁹ 2006 (2) SACR 120 (NC).

³⁰ At paragraph [24].

³¹ Section 3(1)(a) of the Law of Evidence Amendment Act 45 of 1988. See, too, *S v Ndhlovu and others* 2002 (2) SACR 325 (SCA), at paragraph [12].

[41] There can be no doubt that the plaintiff, through her conduct and that of her legal team, never challenged the admissibility of the histopathology report or the ABG analysis during the trial. Her counsel had complete access to the documents well in advance; her experts had been aware of the contents and had freely referred thereto in their reports and their testimonies. The findings were tested under cross-examination. Importantly, as the counsel for the defendant pointed out, the alleged inadmissibility of the documents does not constitute a ground of appeal. It would be difficult not to find that the plaintiff has, by conduct, agreed or acquiesced to the admission of the documents. There is every indication that the provisions of section 3(1)(a) of the Law of Evidence Amendment Act 45 of 1988 find application. If there had been any need to repudiate the contents of the documents, then there would have been a duty on the plaintiff to have done so at the appropriate time and no later.³² For the plaintiff only to have raised an objection in her counsel's heads of argument on appeal, supplemented by notes that were submitted during argument, suggests that this was done simply as an afterthought; it was never an issue during the trial.

[42] There is no basis for the contentions made by the counsel for the plaintiff. Both the histopathology report and the ABG analysis must be considered to have been part of the documentary evidence that was properly available to the trial court. How the injury to U's brain occurred must, at this stage, be the focus of further discussion.

MEDICAL REASONS FOR THE INJURY

[43] The views of the experts involved in the present matter played a decisive role in the determination of the medical reasons for the injury. This, in turn, had a direct bearing on the outcome of the dispute in the trial court.

³² *McWilliams v First Consolidated Holdings (Pty) Ltd* 1982 (2) SA 1 (A), at 10E-G; *Seeff Commercial and Industrial Properties (Pty) Ltd v Silberman* 2001 (3) SA 952 (SCA), at paragraph [19].

[44] In *Pricewaterhousecoopers Incorporated*,³³ Wallis JA remarked that opinion evidence is admissible when an expert can provide ‘appreciable help’ to the court on a particular issue.³⁴ The learned judge went on to emphasize, however, the importance of fact-based reasoning. Similarly, in *AM and another v MEC for Health, Western Cape*,³⁵ Wallis JA discussed the role of an expert as follows:

‘The functions of an expert witness are threefold. First, where they have themselves observed relevant facts that evidence will be evidence of fact and admissible as such. Second, they provide the court with abstract or general knowledge concerning their discipline that is necessary to enable the court to understand the issues arising in the litigation. This includes evidence of the current state of knowledge and generally accepted practice in the field in question. Although such evidence can only be given by an expert qualified in the relevant field, it remains, at the end of the day, essentially evidence of fact on which the court will have to make factual findings. It is necessary to enable the court to assess the validity of opinions that they express. Third, they give evidence concerning their own inferences and opinions on the issues in the case and the grounds for drawing those inferences and expressing those conclusions.’³⁶

[45] The learned judge proceeded further to observe:³⁷

‘The opinions of expert witnesses involve the drawing of inferences from facts. The inferences must be reasonably capable of being drawn from those facts. If they are tenuous, or far-fetched, they cannot form the foundation for the court to make any finding of fact. Furthermore, in any process of reasoning the drawing of inferences from the facts must be based on admitted or proven facts and not matters of speculation. As Lord Wright said in his speech in *Caswell v Powell Duffryn Associated Collieries Ltd*:

“Inference must be carefully distinguished from conjecture or speculation. There can be no inference unless there are objective facts from which to infer the other facts which it is sought to establish... But if there are no positive proved facts from which the inference can be made, the method of inference fails and what is left is mere speculation or conjecture.”³⁸

³³ See n 16, *supra*.

³⁴ *Gentiruco AG*, n 17, *supra*.

³⁵ 2021 (3) SA 337 (SCA).

³⁶ At paragraph [17].

³⁷ At paragraph [21].

³⁸ [1939] 3 All ER 722 (HL), at 733E- F, cited in *Motor Vehicle Assurance Fund v Dubuzane* 1984 (1) SA 700 (A), at 706B- D.

[46] The existence or otherwise of objective facts must, in the present matter, determine the weight that the trial court ought to have attached to the inferences made by the various experts involved. This principle must be applied to the opinion evidence that was presented.

[47] The analysis that follows is based primarily on the available documentary evidence, as interpreted by the experts. It is divided accordingly.

The MRI results

[48] The parties accepted the findings of two radiologists, Dr Bates Alheit and Dr Zuzile Zikalala, whose joint minute, prepared on 11 March 2021, was not in dispute and was admitted as evidence. It is useful to replicate the contents thereof in full:

1. This joint minute has been prepared between Dr B Alheit (BA) and Dr Z Zikalala (ZZ). This joint agreement is presented as a constructive attempt to present to the court the imaging features of the MRI brain scan and to advance a diagnosis for the described pattern.
2. BA refers to the body and comment of ZZ's report.
3. BA agrees with ZZ that the MR study displays features of hypoxic ischaemic injury of the brain.
4. BA agrees that the MR findings (as described by ZZ) make the diagnosis, in the appropriate clinical context, of a dominant watershed zone hypoxic ischaemic injury of the brain.
5. BA further submits that the atrophy of central structures and the hyperintensities in these structures suggest additional PBGT/central hypoxic ischaemic injury of the brain.³⁹
6. Thus the findings are in keeping with a mixed pattern of dominant watershed zone hypoxic ischaemic (prolonged partial pattern) and PBGT/central hypoxic ischaemic injury.

³⁹ In a footnote to the joint minute, 'PBG' is equated to 'perirolandic, basal ganglia and thalamus'.

7. The experts agree that the findings of the MRI study suggest that genetic disorders as a cause of the child's brain damage are unlikely but not excluded in light of the signal changes of the dentate nuclei and posterior pons. Further clinical, genetic and metabolic assessment is advised.
8. The experts agree that there is no evidence of current or previous infective or inflammatory disease on the various MRI sequences and agree that inflammatory or infective conditions are unlikely as direct causes of the child's brain damage.
9. The experts agree that a review of the clinical and obstetrical records by appropriate specialists in the field of neonatology and obstetrics to be essential in determining the cause and probable timing of this hypoxic ischaemic injury.'

[49] There was consensus that the MRI results revealed a hypoxic ischaemic injury to the brain. A mixed pattern was evident, but the experts in question were unable to comment on cause or timing. These aspects inform the analysis that continues below.

Histopathology report and ABG analysis

[50] There was, as already noted, a limited amount of real evidence upon which the trial court could make its findings. Both the MRI results and the Road to Health Chart were available but it seems that the appeal must turn, ineluctably, on the relevance and implications of the histopathology report and the ABG analysis. These must be considered within the context of the opinion evidence of the experts involved.

Dr Constant Ndjapa

[51] The plaintiff's obstetrician and gynaecologist, Dr Ndjapa, was adamant that the injury had occurred during the intrapartum period. He said that it had been caused by a

combination of factors: inadequate monitoring, the use of misoprostol for induction; prolonged labour, with repeated vaginal examinations that had led to intrauterine sepsis; and the inappropriate use of fundal pressure and vacuum extraction. These had all been high-risk factors. Dr Ndjapa stated, in relation to the histopathological report, that there had been no evidence that the onset of chorioamnionitis had taken place during the antenatal period. Dr Reddy's views in that regard, he said, had been purely speculative. The factors mentioned above, all non-speculative and based on the plaintiff's evidence, were the most likely causes of the chorioamnionitis, leading in turn to placental insufficiency and associated hypoxia.

[52] It is not apparent from the record, however, that Dr Ndjapa properly dealt with the full set of findings that emerged from the histopathology report. He never addressed the severity of the chorioamnionitis, and the presence of chorionic vasculitis and funisitis, and how these factors would have had a bearing on the timing of the injury. Regarding the ABG analysis, Dr Ndjapa was reluctant to comment and deferred, instead, to the views of the paediatric neurologists. I am left with the impression, ultimately, that his opinion evidence was incomplete and presented gaps in relation to an explanation of the factual cause of the injury.

Dr Krzysztof Janowski

[53] The defendant's obstetrician and gynaecologist, Dr Janowski, challenged the impact of the factors mentioned by Dr Ndjapa. He testified that the plaintiff had not undergone an extended period of induction; there was no evidence that the medical staff had repeated the initial administration of misoprostol, if that was indeed the medication that had been used.⁴⁰ They had correctly carried out vaginal examinations, using a glove with antiseptic cream; this had been done prior to the rupturing of the

⁴⁰ The plaintiff appears to have used the terms 'misoprostol' and 'syntocinon' interchangeably. See n 8, *supra*.

plaintiff's membranes. The application of fundal pressure and attempts at vacuum extraction, moreover, did not explain the condition of the plaintiff's placenta.

[54] The value of Dr Janowski's views lay in the detail and logic of the reasoning that he employed. He relied on the ACOG criteria (2003), based on research conducted by MacLennan, for defining the causal relation between acute intrapartum events and cerebral palsy. This comprised four indicators: evidence of metabolic acidosis in the umbilical cord or very early neonatal blood samples (i.e. a pH level that was less than 7.0), the early onset of moderate to severe neonatal encephalopathy for infants of more than 34 weeks' gestation, cerebral palsy of the spastic quadriplegic or dyskinetic type, and the exclusion of other identifiable aetiologies, such as trauma, infectious conditions, or genetic disorders.

[55] In that regard, Dr Janowski testified that there were two items of evidence that prevented the fulfillment of the ACOG criteria. The first item was the ABG analysis, which he described, with reference to research conducted by Higgins,⁴¹ as the most objective determination of foetal metabolic condition at birth. The analysis demonstrated the absence of metabolic acidosis, the pH level had been 7.33; there was no indication that the injury had occurred during labour.⁴² He rejected Dr Keshave's view that a high lactate level in the analysis had demonstrated intrapartum hypoxia, saying that ACOG had never recognized this as a criterion; it was too unreliable and there was a lack of consensus about the critical value to be used when determining evidence of metabolic acidosis. The second item was the histopathology report, demonstrating an alternative and identifiable aetiology. The severe acute chorioamnionitis, as reflected in the report, had been responsible for placental dysfunction and the deprivation of nutrients and oxygen to U's brain. It had not been detected because it was asymptomatic; there were clinical features or signs of it in only 15% of cases. The presence of chorionic vasculitis and funisitis, entailing both a maternal and a foetal response, underlined the severity of

⁴¹ Chris Higgins, 'Umbilical-cord blood gas analysis' (October 2014, downloaded from acute-care-testing.org).

⁴² Dr Janowski also pointed out that the CTG reading, obtained on the evening of 7 March 2019, just before birth on the following day, had not given reason for concern; no abnormalities in the foetal heart rate had been seen.

the chorioamnionitis and indicated that the process had taken time to develop. The radiologists' findings, including the prolonged partial nature of the injury, supported his views.

[56] The counsel for the plaintiff challenged Dr Janowski's opinion evidence primarily on the basis that it was undermined by that of Dr Reddy, who confirmed that metabolic acidosis had indeed been present. A proper examination of the record suggests that this was not the case. The closest that Dr Reddy came to this is apparent from the relevant portion of the joint minute prepared with Dr Keshave, which reads as follows:

1. AK:⁴³ Based on YR report of March 2021, he agrees that there is spastic quadriplegic cerebral palsy, with global developmental delay. The clinical presentation is in keeping with the ACOG criteria (2017), for the type of cerebral palsy associated with intrapartum hypoxic ischaemic injury.

YR: *Agrees with the above statement* but would like to specify that antepartum and postpartum hypoxic ischaemic insults can also result in the child having spastic quadriplegic cerebral palsy and global developmental delay.

AK: As per the birth anthropometry antenatally the insult is unlikely to have occurred prior to 37 weeks gestation. Also, there were no postnatal insults to account for the current clinical picture. The postnatal insult of seizures were as a result of the neonatal encephalopathy, and forms part of the syndrome- as described by JJ Volpe (2018).⁴⁴

[57] As I understand it, Dr Reddy merely agreed with Dr Keshave's general statement that U's condition was typical for an intrapartum hypoxic ischaemic injury, as envisaged by the ACOG criteria. She immediately qualified her statement by pointing out that the condition could also result from both antepartum and postpartum injuries. Dr Keshave replied in turn. This is supported by the explanation that she gave during evidence-in-chief:

⁴³ The abbreviation is a reference to Dr Amith Keshave; similarly, 'YR' refers to Dr Yavini Reddy.

⁴⁴ Emphasis added.

'So I agree with Dr Keshave regarding the clinical condition of the child. We are in full agreement with that and what I wanted to specify is that antepartum and even postpartum... hypoxic ischaemic insults can also result in exactly the same condition in the child. So you can have spastic quadriplegic cerebral palsy and global developmental delay not only from an intrapartum insult. There are many causes of that condition.'⁴⁵

[58] In her report, moreover, Dr Reddy referred to the ACOG criteria (2014) and commented that:

'The following neonatal signs are consistent with an acute peripartum or intrapartum event:

- Foetal umbilical artery pH less than 7.0, or base deficit greater than or equal to 12 mmol/L, or both, increases the probability that neonatal encephalopathy, if present, had an intrapartum hypoxic component; lesser degrees of acidemia decrease that likelihood. (*Not fulfilled.*)
 - o *Blood gas shows a compensated metabolic acidosis with high lactate levels:*
 - pH 7.33/ pCO₂- 24.5/ pO₂- 155/ Glucose- 7.1/ Lactate- 11.8/ HCO₃- 16.1/ BD 13
 - o *If there was acute intrapartum brain injury, the acidosis should not be compensated. This implies that the insult may have been prior to the initiation of labour.*⁴⁶

[59] Dr Reddy went on to emphasize:

'[U] fulfils 3/6 ACOG criteria to determine an intrapartum cause of HIE. The Apgar score and blood gas findings do not support an acute peripartum or intrapartum event.'

[60] During her testimony, she stated as follows:

⁴⁵ Sic.

⁴⁶ Emphasis added.

'So I am happy that this blood gas gives you a reasonable picture of what the baby was at birth, I am not saying it is the most accurate because I do not know exactly how it was taken, but I think it is reasonable and for me the big thing here, is it was a compensated metabolic acidosis. And what do I mean by this? I mean that this baby had time to actually adjust to what was happening so there was an insult at some point but this child had the time to compensate and that is why the pH was not lower.'

[61] The record does not support counsel's contention that Dr Reddy undermined Dr Janowski's opinion evidence. Far from it. If anything, the record demonstrates that the defendant's experts were entirely in agreement that the ABG analysis had revealed a compensated metabolic acidosis with a pH level of 7.33, within the normal range.

Dr Amith Keshave

[62] The plaintiff's paediatric neurologist, Dr Keshave, dealt with the ABG analysis by noting that the lactate level of 11.6 had been almost three times higher than normal. This indicated severe acidosis at birth. There had been anaerobic respiration, meaning that, in the absence of sufficient oxygen, U had depended on lactate to generate energy. Consequently, Dr Keshave was of the view that the ABG sample had not been obtained at birth. The baby's encephalopathic state would have led to urgent attempts to resuscitate and stabilize him, which would have quickly corrected the pH level but not the lactate level, which would have taken considerably longer to recover. The sample could not have been obtained from the umbilical cord because this would have been removed at birth, prior to resuscitation. That was the only way to account for the ABG analysis, said Dr Keshave.

[63] Under cross-examination, however, Dr Keshave admitted that there was no record of the sample having been obtained from the baby, instead of the cord. He partially conceded, too, the correctness of Dr Janowski's assertion that the high lactate

level could have been caused by an infection but asserted, nevertheless, that it failed to account for the high oxygen level; this could only have resulted from resuscitation after an intrapartum hypoxic ischaemic injury.

[64] In his report, Dr Keshave concluded that U's condition at birth had satisfied both the Volpe criteria (2018) and the ACOG criteria (2014) for an intrapartum hypoxic ischaemic injury. Regarding the histopathology report, Dr Keshave addressed the presence of chorioamnionitis in the joint minute prepared with Dr Reddy. He referred to an academic article by Harteman and others⁴⁷ and noted that U's C-reactive protein ('CRP') levels had been normal, suggesting that the presence of chorioamnionitis had played a lesser part than other factors.⁴⁸ He went on to assert, in testimony, that academic studies were inconclusive in relation to the impact of chorioamnionitis on the foetus; none of the cases considered had involved a prolonged second stage of labour and neonatal encephalopathy. Dealing specifically with the presence of chorionic vasculitis and funisitis, Dr Keshave pointed out that there could be inflammation even during a normal pregnancy. There would have been signs of an infection, such as a fever, but the inflammatory markers in this case had all been normal. There could have been changes in the markers between the time of the collection of the placenta and the time of its analysis at the laboratory. U's neonatal encephalopathy had to be considered against the background of a prolonged second stage of labour and the complications that had followed, not just chorioamnionitis.

[65] The record indicates that Dr Keshave addressed, to a limited extent, the implications of chorionic vasculitis and funisitis. From the opinion evidence of both Dr Janowski and Dr Reddy, these factors had a significant bearing on the severity of the chorioamnionitis and the timing of the hypoxic ischaemic injury. It is not apparent from Dr Keshave's report, the joint minute, or his testimony, that sufficient attention was given

⁴⁷ Johanna Harteman (et al), 'Placental Pathology in Full-Term Infants with Hypoxic-Ischemic Neonatal Encephalopathy and Association with Magnetic Resonance Imaging Pattern of Brain Injury', *Journal of Pediatrics* (October 2013), at 968-75.

⁴⁸ C-reactive protein is a protein with plasma concentrations that are raised in infections and inflammatory states and in the presence of tissue damage or necrosis. Martin, *op cit*, at 184.

to them to avoid the impression that his views, specifically in this regard, tended to be superficial in nature and unsupported by any clear reference to studies or research.

Dr Yavini Reddy

[66] The defendant's paediatric neurologist, Dr Reddy, expressed the view that severe acute chorioamnionitis with a maternal and foetal inflammatory response, as reflected in the histopathological report, had introduced the possibility of placental foetal vascular malperfusion ('FVM'). This meant impaired foetal blood flow and oxygenation, thereby priming the baby's brain for injury. In her opinion, chorioamnionitis and the resulting foetal inflammatory response had led to placental insufficiency, resulting in the hypoxic ischaemic injury that had caused U's cerebral palsy. It had not been preventable. The injury, said Dr Reddy, was likely to have occurred after 36 weeks' gestation because there had been no intrauterine growth restriction, but prior to the onset of labour. In her view, the injury would have occurred even if the second stage of labour had not been prolonged.

[67] At trial, Dr Reddy commented on the joint minute that she had prepared with Dr Keshave. She testified that the ACOG criteria were merely guidelines; each case had to be considered thoroughly and in its entirety. The International Cerebral Palsy Task Force, comprising a global affiliation of obstetric associations, had emphasized that there must be an absence of all other proximal factors, i.e. factors that were present prior to the onset of labour, before attributing neonatal encephalopathy to an intrapartum hypoxic ischaemic injury. She testified that placental FVM was a new medical concept, only emerging in the past ten to 15 years. There was, furthermore, no better way to understand what had happened at the time of delivery than by studying the condition of the placenta; the histopathology report in the present case was decisive.

[68] Dr Reddy asserted that numerous studies demonstrated that placental hypoperfusion, i.e. a reduced amount of blood flow through the mother's placenta to the foetus, can cause injury to the brain. The process takes place over weeks. The reduced blood flow places stress on the foetal brain such that it cannot endure the normal rigours of labour; there are no energy reserves to allow the brain to cope. The damage has already been inflicted. A reduced blood flow via the umbilical cord has the implication that less oxygen and fewer nutrients reach the brain. Dr Reddy referred to an academic article by Volpe⁴⁹ in which he considered studies carried out on the role of placental FVM in relation to neonatal HIE; the learned writer stated that, in most cases, it evolved over a sub-acute to chronic period prior to delivery but not closer than approximately 48 hours. Volpe went on to observe that the studies also indicated that a state of impaired foetal blood flow and oxygenation existed for many days to weeks prior to delivery. Consequently, said Dr Reddy, there was a basis upon which to say that the onset of chorioamnionitis in the present case had not occurred over a period of hours or a day. The injury to U's brain had taken place during a four-week period, after 36 weeks of gestation but prior to the onset of labour; it had not occurred any earlier because there had been no evidence of growth retardation. She also referred to an article by MacLennan and others⁵⁰ where the learned writers used the findings of multiple epidemiological⁵¹ studies to contend that chorioamnionitis and funisitis were evidence of infection that predated labour and were associated with an increased risk of cerebral palsy.

[69] In relation to Dr Keshave's reference to the Harteman study, Dr Reddy stated that it supported her views. Studies have shown that milder degrees of chorioamnionitis, with only a maternal inflammatory response, create a lower risk of injury to the foetal brain; in contrast, more severe degrees of chorioamnionitis, with thrombosis and a

⁴⁹ Joseph Volpe, 'Placental assessment provides insight into mechanisms and timing of neonatal hypoxic-ischemic encephalopathy', *Journal of Neonatal-Perinatal Medicine* 12 (2019), at 113-6.

⁵⁰ Alastair MacLennan (et al), 'Cerebral palsy: causes, pathways, and the role of genetic variants', *American Journal of Obstetrics & Gynecology* (December 2015), at 779-88.

⁵¹ The term, 'epidemiological', is used here as an adjective; it is derived from the noun, 'epidemiology', which is defined as the study of the distribution of diseases and determinants of disease in populations. Martin, *op cit*, at 262.

foetal inflammatory response, create a much higher risk. The severe acute chorioamnionitis of the placenta, as identified in the histopathological report, created a considerably higher risk of abnormalities in the foetus than a prolonged second stage of labour. Dr Reddy strongly asserted that the presence of such chorioamnionitis had led to the impairment of blood flow, meaning that less oxygen and fewer nutrients had reached the foetus, thereby causing damage to U's brain. This occurred prior to the commencement of labour.

[70] Whereas Dr Reddy agreed with Dr Keshave about the importance of foetal monitoring, this did not contradict the findings of the academic studies that she had mentioned. A prolonged second stage of labour led to foetal abnormalities in a much smaller percentage of babies when contrasted with outcomes involving the presence of severe acute chorioamnionitis, such as that in the present case.

[71] Dr Reddy's opinion evidence was influenced primarily by the histopathology report. Her fact-based approach, clear reasoning, and references to studies or research to substantiate her conclusions were indeed persuasive, as the trial court found.

Discussion

[72] The trial court agreed with Dr Janowski and Dr Reddy that the answer to the question of when U's brain was injured lay in the histopathological report. It found that the conclusions reached in relation thereto by the above experts were based on logical reasoning that was grounded in fact. I am unable to fault the trial court's findings in this regard.

[73] To a greater or lesser extent, Dr Reddy's opinion evidence was the fulcrum upon which the proceedings in the trial court balanced. Unsurprisingly, the plaintiff's counsel

directed his attack accordingly on appeal. The main elements of his attack must be considered further.

Volpe and ACOG criteria

[74] It was argued that Dr Reddy effectively conceded that both the Volpe and the ACOG criteria had been met. A close examination of the record, however, reveals this not to have been the case; she asserted that there was no indication from either the available neonatal records or the plaintiff's evidence that there had been foetal distress; the results of the ABG analysis were not typical for intrapartum asphyxia. The following exchange during cross-examination is pertinent:

'MR MALUNGA: Dr Reddy, if we then look at the criteria of Volpe, which we all agree is almost the Bible for this.

DR REDDY: No, absolutely not. Not the Bible in any way, shape, or form. We use it because unfortunately most people do put it in their reports so we do use it. Volpe is a very respected neonatal specialist... but his criteria... it is actually not criteria, it is just his- the things that he recommends, it is not like [the] ACOG criteria which have been adopted by societies all over the world.'

[75] Counsel continued, referring to U's need for resuscitation, the low Apgar scores, compromise at birth, and seizures within 48 hours:

'MR MALUNGA: And that was the evidence of Dr Keshave that all these factors, if giving a timing to this particular injury..., were the factors that pointed to an intrapartum injury.

DR REDDY: What I would put to you, is if Volpe had to- remember this book, I think it came out, these criteria, in 2016, around there, and like I mentioned earlier, placental studies have been recent and it is evolving. So Volpe has not included it yet in his criteria but every

obstetric association in the world has now included placental histology into their assessment of a child with compromise. So yes, counsel, I do agree that the Volpe criteria, it is met in a very loose way but the ACOG criteria were not met. So there was a discrepancy between the Volpe criteria and the ACOG criteria and I think it is too simplistic to use three criteria when we know that there is a lot more happening with this case.⁵²

[76] Insofar as Dr Reddy conceded that the Volpe criteria had been met, in a 'very loose way', she unequivocally rejected the assertion that the ACOG criteria had been satisfied. Importantly, she attached significantly more weight to the authority of the latter, for reasons apparent from the above exchange.

Hypoxia at birth

[77] The assertion was made by counsel that Dr Reddy conceded that U had been born with hypoxia. Further examination of the record again indicates otherwise. She agreed that the baby had been compromised at birth and that this had been because of a hypoxic injury to the brain; she also conceded that a hypoxic event may occur during a period of sub-standard monitoring. Dr Reddy went on, however, to emphasize that the injury in this case took place in the days or weeks before birth.

Thrombosis

⁵² The reference was to the three Volpe criteria that Dr Keshave identified in his report, viz. evidence of foetal distress or risk of hypoxia or ischaemia, a need for resuscitation and low Apgar scores, and an overt neurological syndrome in the first 24 hours of life. Emphasis added.

[78] The plaintiff's counsel argued that Dr Reddy's evidence was far from consistent or entirely cogent. She withdrew, crucially, her submissions regarding thrombosis. The following extract from the record is relevant:

'DR REDDY: So with the placenta, over weeks this is happening, where the blood flow from the mother to the child is less than it should be, it is not optimal... it is basically the blood flow through the umbilical cord from the placenta, going into the brain that is affected. It is less oxygen, less nutrients and less flow and we know that happened for a fact, because when you look at the placenta, the vessels are thrombosed and what that means when the vessels are thrombosed, means that there is blockages. So there is actually physical blockages where the blood needs to flow in, it has been blocked. And you know, that is how the damage to the brain happens. There are other mechanisms, the inflammation for example, causes certain signals to go to the brain that also makes it more likely to be damaged but the main effect is actually lack of oxygen and lack of blood flow.'

[79] Dr Reddy consequently referred to the Volpe article,⁵³ pointing out that it was published after his book and that it addressed placental hypoperfusion directly. The extract from the record continues:

'DR REDDY: ...So Volpe did an editorial...and in this he was looking at a study [on]... placental [hypo]perfusion...:

"The placental findings of foetal vascular malperfusion..."

And that is what I explained earlier, so not enough blood flow from the mother to the child:

"are considered to be secondary to chronic, partial or recurrent intermittent obstruction of umbilical blood flow, thereby leading to umbilical venous obstruction, and, as a consequence, venous congestion, stasis, thrombosis in severe cases."

⁵³ See n 49, *supra*.

So we have thrombosis in this placenta, so we know that it was a severe case.

“The consequences for the foetus would be expected to include impaired foetal blood flow and oxygenation, and ultimately cardiac insufficiency...”

So if it goes on for a certain time, the heart is also affected.

“[a]nd compromised cerebral blood flow and oxygenation...”

That is impaired blood flow to the brain and what is very important is the last four lines:

“Based on histological features, foetal vascular malperfusion is considered to evolve in most cases over a sub-acute to chronic period prior to delivery and not closer to delivery than approximately 48 hours prior.”

[80] It subsequently became apparent that Dr Reddy had misinterpreted the histopathology report in relation to the presence of thrombosis. She eventually conceded that it had been absent. She remained adamant, nevertheless, that the risk of injury to the foetus remained significant. The following extract pertains:

‘DR REDDY: Absent. Okay, so sorry, that was my- because I understood it as congested or thrombosed in terms of the foetal surface. So sorry, so the thrombosis is not there, *but it is still a severe foetal inflammatory response. So the risk may go from 99 percent to 70 percent in terms of probability.*⁵⁴

[81] She stated further:

‘DR REDDY: ...So there was severe acute chorioamnionitis with a maternal and foetal inflammatory response; so we know that the foetal inflammatory response gives you a much higher risk of damage to

⁵⁴ Emphasis added.

the placenta. So I will withdraw my statement about the foetal placental thrombosis from the previous report. But like I said, *severe acute chorioamnionitis with a foetal inflammatory response still gives you a high probability of damage to the foetus...*⁵⁵

[82] It cannot be held, as the plaintiff's counsel suggested, that Dr Reddy's concession deprived her views of consistency or cogency. The only material effect was to reduce the risk factor. What cannot be ignored is Dr Reddy's opinion evidence that severe acute chorioamnionitis in the placenta, with a maternal and a foetal inflammatory response, posed a considerably higher risk of injury to the foetus; she put this as high as a 70% probability. The concession had no impact whatsoever on her views regarding the severity and timing of the injury.

Causative effect of chorioamnionitis

[83] The plaintiff's counsel went on to contend that Dr Reddy had failed to explain, clearly, the causative effect of chorioamnionitis on U's brain. She had not described how or when this had occurred. A close examination of the record, however, reveals that Dr Reddy described in detail how the severe nature of the chorioamnionitis, with both a maternal and a foetal inflammatory response, had given rise to placental FVM; the compromised flow of blood and oxygen from the placenta had created the hypoxic ischaemic event that had caused the damage to U's brain, manifesting, ultimately, as cerebral palsy. This had occurred over a period of days, if not weeks, before birth, based on Volpe's recent article.⁵⁶ She relied, too, on the article by MacLennan and others to assert that the presence of funisitis meant that the infection had pre-dated the commencement of labour.⁵⁷ The radiologists' findings did not undermine her views.

Infective or inflammatory disease

⁵⁵ Emphasis added.

⁵⁶ See n 49, *supra*.

⁵⁷ See n 50, *supra*.

[84] It was the argument of the plaintiff's counsel that Dr Reddy's evidence conflicted with that of the radiologists since they found that there had been no sign of any injury or condition of an inflammatory nature. Their joint minute stated that there was no evidence of 'current or previous infective or inflammatory disease on the various MRI sequences'; they went on to agree that 'inflammatory or infective conditions are unlikely as direct causes of the child's brain damage'.⁵⁸ Dr Reddy dealt with the argument as follows:

'DR REDDY: ...there is direct causes, so infection is an unlikely a direct cause of this injury; I agree wholeheartedly with that but it needs to be qualified and they [the radiologists] have not explained themselves well, unfortunately, in those joint minutes. What they are referring [to] is direct infections of the brain. *I have never said the baby has a direct infection of the brain, the infection is of the placenta, that caused impaired blood flow to the brain.* So I am not arguing with that but what they are referring to is a meningitis, so infection of the covering of the brain that can give you certain characteristics on MRI. They would be referring to an abscess; so if you get infection going directly to the brain, it can cause a brain abscess. There are other viral infections that we know can occur in newborn babies: CMV, toxoplasmosis... that can give you [a] typical picture on MRI. *So when they say that it is not due to direct infection, it has nothing to do with chorioamnionitis. They are talking about something else.*

COURT: Could chorioamnionitis not- your evidence is that is that is the cause.

DR REDDY: Yes, but *it is an indirect cause.*

COURT: Albeit over a long time, but it is the cause, on your evidence, of the injury.

⁵⁸ At paragraph 8 of the joint minute.

DR REDDY: But you could never see it on the MRI. So the MRI of the baby's brain can never give you a window into what the cause was. So that is why they would have no way of excluding chorioamnionitis as a cause. *They would only be able to exclude direct infection to the brain which we have never said that this child has.*⁵⁹

[85] The defendant's counsel pointed out that Dr Keshave had admitted, at trial, that a severe infection could lead to a compromised supply of oxygen to the brain. It was only a direct infection of the brain, such as meningitis, that had been ruled out.

Acute profound and partial prolonged injury

[86] Regarding the contention of the plaintiff's counsel that Dr Reddy had only addressed the question of causation in relation to an acute profound injury, instead of a prolonged partial injury, this is simply not supported by the record. At best, she indicated that chorioamnionitis could have primed U's brain for a subsequent acute insult. It had, however, already damaged the brain over a period of days if not weeks beforehand, corresponding with the prolonged partial pattern that was identified by the radiologists. There was no indication, moreover, of any intrapartum hypoxic ischaemic event, either of a prolonged partial or an acute profound nature, based on the histopathological report and the ABG analysis.

Opinion evidence

[87] Finally, mention must be made of the assertion by the plaintiff's counsel that Dr Reddy was unable to provide opinion evidence on possible factual causation, including supplementary causation, during labour. The underlying premise for this seems to have

⁵⁹ Sic. Emphasis added.

been that a paediatric neurologist was qualified to express a view, as an expert, on events occurring only from the moment of birth onwards; prior to this, i.e. during labour, the relevant expert was an obstetrician such as Dr Janowski.

[88] The *in utero*-birth divide serves as a useful distinction between the respective roles of the experts involved. It must, however, not be treated as a heavily guarded border between sovereign states, preventing movement between the two territories. There will be a transition zone where the expertise and experience of the experts overlap, allowing a court to consider, at the very least, the opinion evidence placed before it. The court must then decide, in accordance with the usual principles of fact-based reasoning, whether such evidence offers ‘appreciable help’ in the adjudication of the dispute, as Wallis JA remarked in *Pricewaterhousecoopers Incorporated*.⁶⁰ Experts in the medical field do not operate in hermetically sealed compartments, as the learned judge went on to observe in *HAL*,⁶¹ the court is entitled to the full picture.

[89] In the present matter, the defendant’s counsel requested Dr Reddy to respond to Dr Keshave’s view that the management and effect of chorioamnionitis was best commented upon by an obstetrician or a pathologist. She stated:

‘DR REDDY: So that is where I have to disagree with Dr Keshave very vehemently. You know, as a paediatric neurologist, it is our job to look -at anything that can affect the brain and this is one of the big factors that recently have been identified as affecting perfusion to the brain. So I would not defer comment on this to anybody, I am happy that I have sufficient expertise to comment on the chorioamnionitis...

MR NABELA: So, in other words, what you are telling this court, is that Dr Keshave was wrong that it is only [an] obstetrician who can comment only [sic] on chorioamnionitis?

⁶⁰ See n 16, *supra*.

⁶¹ See n 13, *supra*, at paragraph [222].

DR REDDY: Yes, so in terms of the chorioamnionitis, obstetrician, yes, in terms of the cause I would say, you know, they are probably the experts on the cause of the chorioamnionitis, but in terms of the effect of the chorioamnionitis, I would argue that I am also an expert on that.'

[90] Notwithstanding the above response, it is vitally important not to overlook Dr Reddy's view, based on the article by MacLennan and others, that the presence of funisitis indicated that the onset of chorioamnionitis had, in fact, pre-dated the commencement of labour. This assertion was never properly disputed or challenged.

What, factually, was the cause of the injury?

[91] Returning to the test described by Corbett JA in *Blyth*,⁶² the determination of the factual cause of the injury to U's brain, i.e. the medical reasons, must be decided before addressing the question of the negligence of the medical staff involved. This is no easy task when confronted with the specialized nature of the subject, the lack of consensus amongst the experts, and the almost complete absence of proper medical records. Counsel for the defendant referred to *Buthelezi v Ndaba*,⁶³ where Brand JA observed:

'The human body and its reaction to surgical intervention are far too complex for it to be said that, because there was a complication, the surgeon must have been negligent in some respect.'⁶⁴

[92] This is, with respect, a particularly apt observation. It is unnecessary to strive, at one extreme, for absolute clarity and unwavering certainty about the reasons for an injury and whether the medical practitioners involved must be held accountable. The

⁶² See n 21, *supra*.

⁶³ 2013 (5) SA 437 (SCA).

⁶⁴ At paragraph [16].

courtroom is not a scientific laboratory. At the other extreme, causation and delictual liability cannot be decided merely on a balance of possibilities. The role of the court, reduced to its essence, is to evaluate the available evidence and to adjudicate the dispute based on whether the plaintiff has on a balance of probabilities proved his or her case. Within the context of an appeal, the court must, of course, decide whether the trial court successfully performed such a role and whether its findings were indeed correct.

[93] In the present matter, the plaintiff failed to produce sufficiently compelling factual or opinion evidence to persuade the trial court that the views of Dr Reddy and Dr Janowski were wrong. No real alternative was presented to deal, effectively, with the pleaded defence in relation to how the injury to U's brain happened.

[94] Having assessed, on appeal, the views of the experts in light of the principles of fact-based reasoning, I am satisfied that the probable medical reason for the injury was a hypoxic ischaemic event or events that resulted from placental FVM to which the severe acute chorioamnionitis, identified in terms of the histopathological report, gave rise. This (or these) had taken place prior to the commencement of labour. It is also clear from the opinion evidence that no investigation of the possible causes of a hypoxic ischaemic injury is complete, in circumstances such as these, without properly considering the placental histopathology and arterial blood gas.⁶⁵

[95] It is necessary to proceed to the next stage of the enquiry. The question to be answered is whether any negligence on the part of the medical staff involved caused or materially contributed to the injury when this could have been prevented.

NEGLIGENCE AND CAUSATION

⁶⁵ The significance of the placental histopathology was acknowledged in *Magqeya v Member of the Executive Council for Health, Eastern Cape* 2018 JDR 1667 (SCA).

[96] Negligence is the immediate issue to be considered. If there had been negligence on the part of the staff, then it must yet be decided whether the requirements for causation were met in the trial court.

Negligence

[97] At the outset, it must be noted that the trial court made no finding regarding negligence. The relevant extract from the judgment reads as follows:

‘...To succeed in her delictual claim, the plaintiff was required to prove that the treating staff wrongfully and negligently caused the damage to her child’s brain. What was in issue were the elements of negligence and causation. I will examine the issue of causation *on the assumption, without finding, that the treating staff were negligent* by causing the plaintiff to endure a prolonged and protracted labour, subjecting her to sub-standard care by not monitoring her and the child at regular intervals, attempting to vacuum extract the child and applying fundal pressure and failing to intervene after misoprostol had been given to her. In the determination of causation, the medical evidence will be considered against the background of the plaintiff’s evidence.’⁶⁶

[98] The focus moves away at this stage from the expert’s views to the evidence of the plaintiff herself, necessitated by the paucity of available documentary evidence as already discussed. The extent to which the plaintiff’s evidence is still relevant, after the findings made in relation to factual causation, must be explored further.

[99] The defendant’s counsel argued that the plaintiff was completely unreliable as a witness and provided examples of why this was so. To this, the plaintiff’s counsel pointed out that the trial court had never been required to determine her reliability; the defendant’s counsel had indicated that the dispute pertained to issues of causation, not negligence. Consequently, asserted the plaintiff’s counsel, an appeal court cannot readily interfere with the trial court’s acceptance of the plaintiff’s evidence where it had

⁶⁶ At paragraph [18]. Emphasis added.

been undisputed. The authority upon which he relied, however, viz. *Santam Bpk v Biddulph*,⁶⁷ does not appear to prevent this entirely. To that effect, Zulman JA held as follows:

'...Whilst a court of appeal is generally reluctant to disturb findings which depend on credibility it is trite that it will do so where such findings are plainly wrong... This is especially so where the reasons given for the findings are seriously flawed. Over-emphasis of the advantages which a trial court enjoys is to be avoided lest an appellant's right of appeal "becomes illusory" ... It is equally true that findings of credibility cannot be judged in isolation but require to be considered in the light of proven facts and the probabilities of the matter under consideration.'⁶⁸

[100] The learned judge went on to hold that the proper test was not whether a witness was truthful or indeed reliable in all that he or she says but whether, on a balance of probabilities, the essential features of his or her story were true.⁶⁹ The same principles potentially find application in the present matter.

[101] Both Dr Ndjapa and Dr Keshave pointed out multiple high-risk factors that were present during the labour process. These included the administration of misoprostol, inadequate monitoring, repeated vaginal examinations, a prolonged second stage of labour, the application of fundal pressure, and several failed vacuum attempts. The key factor that emerged from the trial proceedings, however, was whether there had been negligence in the monitoring of the plaintiff. The remaining factors, whether considered individually or collectively, did not seem to have played as important a role. There was no conclusive evidence that the 'mixture' taken by the plaintiff had indeed been misoprostol;⁷⁰ Dr Janowski mentioned that the plaintiff had testified that the staff had used gloves and antiseptic cream when conducting vaginal examinations; he also observed that she had testified that the baby's head had started to protrude, prompting

⁶⁷ 2004 (5) SA 586 (SCA).

⁶⁸ At paragraph [5].

⁶⁹ At paragraph [10].

⁷⁰ The plaintiff mentioned, in her testimony, the administration of 'maiso'; there was no documentary or opinion evidence presented during trial proceedings to corroborate or clarify this.

the use of fundal pressure and vacuum extraction, which were the most practical options available when preparation for a caesarean section delivery would have taken too long; and Dr Reddy referred to academic studies to contend that a prolonged second stage of labour was a low-risk factor, between 0.4% and 1.29%, for a hypoxic ischaemic event.⁷¹

[102] The plaintiff's testimony that a medical practitioner had informed her that insufficient oxygen and a delay in giving birth had caused the brain damage does not take the matter much further. The plaintiff's counsel suggested that the trial court ought to have applied the principles set out in *HN*,⁷² discussed earlier, to find that the above evidence was an admission of negligence on the part of the defendant. Considering the histopathology report and ABG analysis, however, the medical practitioner had no proper factual basis upon which to have made such an assertion at the time.

[103] Returning to the monitoring of the plaintiff, it is apparent from her testimony that CTG readings were indeed taken. She testified that a 'machine' had been placed on her stomach twice in a day but whether this was done in accordance with the benchmark was highly contested.⁷³ Significantly, Dr Janowski conceded that the monitoring had indeed been sub-standard. What is clear from the record, however, is that at no stage was there any indication of foetal distress. The plaintiff stated that, as late as the evening of 7 March 2019, when she had already been admitted to the labour ward, the 'machine for heartbeat' was applied and the following ensued, as indicated in the record:

⁷¹ Both Dr Reddy and Dr Keshave referred to Sandström (et al), 'Durations of second stage of labor and pushing, and adverse neonatal outcomes: A population-based cohort study', *Journal of Perinatology* (2017). The article was, however, excluded from the appeal record.

⁷² See n 26, *supra*.

⁷³ The 'Guidelines for Maternity Care in South Africa' (n 9, *supra*) is generally recognized as the benchmark for the subject in question. See, too, *Nkamela v Member of the Executive Council for Health: Eastern Cape Province* 2022 JDR 1522 (ECB), at paragraphs [9] to [11].

'MS K[...]: After some time, she [the nurse] came up and looked at a print-out that was coming out of that machine; then she said everything was becoming alright [sic] and I was going to start pushing.

[104] There is simply no evidence that the condition of the baby, at any stage prior to birth, gave cause for concern, notwithstanding possible sub-standard monitoring. The chief contention made by the plaintiff's counsel was that proper monitoring, including the conducting of regular CTG readings, would have alerted the staff to signs of foetal distress during the intrapartum period, allowing necessary action to be taken in good time. This never happened. They failed, argued counsel, to prevent injury that occurred.

[105] Dr Janowski and Dr Reddy both expressed the view, however, that the injury had not occurred during the intrapartum period. The facts and reasoning upon which their view was based were canvassed at some length in the investigation of the medical reasons for the injury.

[106] Each of the defendant's experts, moreover, expressed the view that the nature of the chorioamnionitis had been asymptomatic. The following extract from the cross-examination of Dr Janowski pertains:

'MR MALUNGA: Doc, how long would it take for the onset of acute chorioamnionitis?

DR JANOWSKI: To develop?

MR MALUNGA: Yes.

DR JANOWSKI: We do not know, because there are no symptoms, but definitely before the onset of labour.

MR MALUNGA: I think the first part of your answer, Doc, is you do not know, because it does not have symptoms?

DR JANOWSKI: Yes, it is asymptomatic. Patient did not have any symptoms. And as I have said yesterday only in 15 percent of acute chorioamnionitis there are symptoms of- symptoms of clinical chorioamnionitis like a tenderness, temperature is high- high temperature and a high pulse rate.'

[107] He went on to deal with the timing of the onset of chorioamnionitis before returning to the question of whether proper CTG monitoring would have detected it. The relevant extract from the record reads:

'MR MALUNGA: Whilst... you would exclude the hypoxic event occurring between the sub-standard monitoring... do you agree... you could not time when the chorioamnionitis happened?

DR JANOWSKI: Yes, I will repeat myself. We do not know *because there were no signs*; that is why I will repeat again, that is why we keep the placenta as a black box which can tell us what was happening.⁷⁴ We do not know, but taking into consideration that that result, the presence of maternal response, foetal response, the severity of that acute chorioamnionitis and I will say it clear, M'Lord, it did not happen with that short period of time when she was admitted or when she was induced or when she was five times examined with unsterile gloves and... when somebody tried to rupture the membranes, no, no. That is not, it cannot happen like this, not that severity of acute chorioamnionitis, it takes time, it has to develop over certain- how long before delivery? Maybe two weeks, maybe week, maybe three weeks, we do not know. And even more... is just to add, M'Lord... *the problem is that there are no tools right now, even doing CTGs, to predict that maybe there is an infection.* There are no tools because it is like the typical pattern that something is happening... *Unfortunately that is the problem, that is why it is silent, there are no warning signals, it is happening*

⁷⁴ The reference to a 'black box' is understood as the flight data recorder that facilitates the investigation of aviation incidents. See https://en.wikipedia.org/wiki/Flight_recorder accessed on 22 March 2024.

*without any warning, that is why it is so important to check the placenta...*⁷⁵

[108] From Dr Janowski's evidence, CTG monitoring was an inadequate tool for the proper detection of an infection and the onset of chorioamnionitis in circumstances such as these. The examination of the placenta, after delivery, was imperative.

[109] The limitations of CTG monitoring were emphasized, too, by Dr Reddy. She was asked, under cross-examination, whether the CTG reading would have been normal in a situation where, upon admission of the mother for labour management, the foetus had already been compromised. She stated:

'DR REDDY: Yes, and I will give you a good example, we deal with it all the time. Children with a perinatal stroke... if the child has had a stroke *in utero*, it might have occurred at any time; or a child that is... born without half [of] the brain... the CTG is completely normal because the injury did not occur at that time. So you can have severe brain malformations and severe problems with the brain and you can have a normal CTG... Because you must remember that the foetal heart rate is actually controlled from the brain stem. So not the top part of the brain, it is where the little stem that comes down, that controls [the] heart rate and unless there is specific damage there or there is something acute happening, you can actually a very compromised brain injury and a normal CTG...'

[110] Her explanation also addresses the argument made by the plaintiff's counsel that it was a fundamental improbability that U could have survived *in utero* for such a lengthy period without any signs of foetal distress. From Dr Reddy's opinion evidence, this was indeed a possibility, at the very least.

⁷⁵ Emphasis added.

[111] Dr Reddy commented, moreover, on the potentially asymptomatic nature of chorioamnionitis. The following exchange took place during cross-examination:

'MR MALUNGA: Okay, now as to the timing of that injury, you testified that the probable timing is anything from 37 weeks to birth... I put to you, Doc, that if the chorioamnionitis had occurred during that period, there would have been infective markers with the mother in that there would have been a temperature, a fever... if it happened within four weeks prior to... [intervenues]

DR REDDY: No, I disagree. As Dr Janowski mentioned, *this is sub-clinical chorioamnionitis or histological chorioamnionitis, which is far more common than clinical chorioamnionitis*. So clinical chorioamnionitis is where there is a fever in the mother, you can have abdominal pain, the septic markers go up. That is actually very unusual so it is actually more common to have sub-clinical or silent chorioamnionitis like in this case. *So you would not have actually seen any signs in the mother with this acute chorioamnionitis.*⁷⁶

[112] The mostly asymptomatic nature of chorioamnionitis, as described by Dr Janowski and Dr Reddy, countered Dr Keshave's observation that U's inflammatory markers had been normal, i.e. there had been no overt signs of infection. The evidence of chorioamnionitis only emerged in the histopathological report, after the plaintiff's placenta had been sent away for analysis.

[113] The plaintiff's experts conceded that there were limitations to CTG monitoring. Dr Ndjapa admitted that a CTG reading may indicate a normal foetus but the outcome could be an abnormal baby. Dr Keshave seemed to have gone further, admitting that, despite proper CTG monitoring, a baby could indeed be born with neonatal encephalopathy, especially where there was an infection.

⁷⁶ Emphasis added.

[114] In relation to the missing medical records, the plaintiff's counsel suggested that this had resulted from an attempt by the medical staff to conceal sub-standard care. Dr McConney's testimony, however, did not support this. The trial court was correct to have found that there was no reason to blame the Department of Health for the absence of the records. Nothing, in the end, turned on it.

[115] I am not persuaded that the plaintiff proved, on a balance of probabilities, that the staff had been negligent. The enquiry should end there. Nevertheless, considering Dr Janowski's concession in relation to the CTG monitoring that was (or was not) carried out, I deem it necessary to address, briefly, the final issue, viz. causation.

Causation

[116] Both parties referred to *Lee v Minister for Correctional Services*,⁷⁷ where the Constitutional Court dealt with the test for causation. Nkabinde J held:

'...The point of departure is to have clarity on what causation is. This element of liability gives rise to two distinct enquiries. The first is a factual enquiry into whether the negligent act or omission caused the harm giving rise to the claim. If it did not, then that is the end of the matter. If it did, the second enquiry, a juridical problem, arises. The question is then whether the negligent act or omission is linked to the harm sufficiently closely or directly for legal liability to ensue or whether the harm is too remote. This is termed legal causation.'

...This element of liability is complex and is surrounded by much controversy. There can be no liability if it is not proved, on a balance of probabilities, that the conduct of the defendant caused the harm. This is so because the net of liability will be cast too wide. A means of limiting liability, in cases where factual causation has been established, must therefore be applied. Whether an act can be identified as a cause depends on a conclusion drawn from available facts or evidence and relevant probabilities. Factual causation, unlike legal causation where the

⁷⁷ 2013 (2) SA 144 (CC).

question of the remoteness of the consequences is considered, is not in itself a policy matter but rather a question of fact which constitutes issues connected with decisions on constitutional matters as contemplated by s 167(3)(b) of the Constitution.

...Although different theories have developed on causation, the one frequently employed by courts in determining factual causation is the *conditio sine qua non* theory or but-for test. This test is not without problems, especially when determining whether a specific omission caused a certain consequence. According to this test the enquiry to determine a causal link, put in its simplest formulation, is whether “one fact follows from another.”⁷⁸

[117] Nkabinde J held, further, that the rule regarding the application of the test was not inflexible. There were situations where its strict application would result in injustice.

[118] The plaintiff’s counsel contended that a flexible approach should be adopted in the present matter, as endorsed by the Constitutional Court in *Mashongwa v Passenger Rail Agency of South Africa*.⁷⁹ As Mogoeng CJ pointed out, however, the decision in *Lee* never sought to replace the pre-existing approach to factual causation, premised on the flexibility always recognized in the common law.⁸⁰

[119] In *ZA v Smith and another*,⁸¹ Brand JA observed:

‘What it [the but-for test] essentially lays down is the enquiry- in the case of an omission- as to whether, but for the defendant’s wrongful and negligent failure to take reasonable steps, the plaintiff’s loss would not have ensued. In this regard this court has said on more than one occasion that the application of the “but-for test” is not based on mathematics, pure science or philosophy. It is a matter of common sense, based on the practical way in which the minds of ordinary people work, against the background of everyday-life experiences. In applying this common-sense, practical test, a plaintiff therefore has to establish that it is more likely than not

⁷⁸ At paragraphs [38] to [40].

⁷⁹ 2016 (3) SA 528 (CC).

⁸⁰ At paragraph [65].

⁸¹ 2015 (4) SA 574 (SCA).

that, but for the defendant's wrongful and negligent conduct, his or her harm would not have ensued. The plaintiff is not required to establish this causal link with certainty.¹⁸²

[120] If the test is applied to the present matter, then it cannot be said that the plaintiff proved that, but for the negligence of the medical staff, U would not have suffered harm. Based on the plaintiff's evidence and the opinion evidence of the various experts involved, I am satisfied that the baby's cerebral palsy was, on a balance of probabilities, caused by a hypoxic ischaemic injury that occurred before the commencement of labour and which was the result of placental FVM. This had arisen from severe acute chorioamnionitis, accompanied by chorionic vasculitis and funisitis; it had been asymptomatic in nature.

[121] Consequently, I am not persuaded that any negligence on the part of the staff caused or materially contributed to the injury to U's brain when this could have been prevented by exercising reasonable care and skill. There was simply no causal link.

RELIEF AND ORDER

[122] The trial court found that the plaintiff failed to establish that the defendant was delictually liable to her. For the reasons set out above, I am of the view that there is no basis upon which to interfere with such a finding. The appeal cannot succeed on any of the grounds listed by the plaintiff.

[123] Regarding costs, there is no reason why the general rule should not apply. The defendant is entitled to recover the expenses involved.

⁸² At paragraph [30].

[124] The following order is made:

- (a) the appeal is dismissed; and
- (b) the plaintiff is directed to pay the defendant's costs, including those of two counsel.

J LAING
JUDGE OF THE HIGH COURT

I agree.

L RUSI
JUDGE OF THE HIGH COURT

I agree.

I BANDS
JUDGE OF THE HIGH COURT

Appearances

For the appellant: Adv AG Dugmore SC and Adv Y Malunga

Instructed by: S Booi & Sons Attorneys

6A Sansom Road, Vincent, EAST LONDON

Ref: K58/10/19

Tel: 043 721 1701

Email: sbooi@telkomsa.net

c/o Luvuyo Solvern Attorneys

Office no. 2, 1st floor, Old King Theatre

QONCE (King William's Town)

Tel: 043 642 4922

Email: solvernattorneys@gmail.com

For the respondent: Adv PJ de Bruyn SC and Adv N Nabela

Instructed by: State Attorney

Old Spoornet Building

17 Fleet Street, EAST LONDON

Ref: 1085/19-P6 (Ms Dlanjwa)

c/o Office of the Premier

32 Alexandra Road

QONCE (King William's Town)

Date for final submissions: 18 December 2023

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