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

REPORTABLE
CASE NO: C.A.& R: 8/2021
DATE HEARD: 03 SEPTEMBER 2021
DATE HANDED DOWN: 21 JANUARY 2022

In the matter between:

J A[....] on behalf of DMA **APPELLANT**

and

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

FULL COURT APPEAL JUDGMENT

D VAN ZYL DJP:

[1] This an appeal against an order granting absolution from the instance. The appellant's claim was for delictual damages on behalf of her minor child (as is customary, I shall refer to him as DMA to protect his identity). The claim was founded on the allegation that DMA suffered a brain injury, caused by the negligence of the respondent's employees when the appellant gave birth to DMA at the Andries Vosloo Hospital in Somerset East in the Eastern Cape. The respondent admitted that the hospital staff were negligent.

[2] The sole issue in the appeal is that of factual causation, that is, whether or not it can be said that the negligent conduct of the medical staff at the hospital was the cause of the injury suffered by DMA. That this was the only issue, was confirmed by the parties during argument. On the facts, and in the context of the evidence presented in this matter, a determination of the primary issue in turn raises two secondary issues. The first issue is how to deal with a conflict in the evidence of opposing expert witnesses, while the second raises the question when factual causation must be found to have been proved in a delictual claim of this nature.

[3] On the common cause evidence placed before the trial court, DMA had suffered a brain injury. His clinical condition is that he has a left sided spastic hemiplegic type of cerebral palsy with microcephaly.[1] Furthermore, he suffers from epilepsy, and what is described as severe global developmental delay. The disagreement lies in what caused the brain injury, and of the clinical condition which resulted therefrom. The position adopted by the parties in their respective expert witnesses' summaries is as follows: according to the appellant, DMA's condition is the result of a brain injury known as hypoxic-ischemic encephalopathy, which he sustained during labour, and was caused by prolonged partial hypoxic ischemia. The respondent's expert witness on the other hand contended that on the clinical evidence, DMA has an underlying neurometabolic disorder in keeping with Nonketotic Hyperglycinemia, which may account for his clinical condition.

[4] Hypoxic-ischemic encephalopathy (HIE) is a brain injury as a result of impaired cerebral blood flow and oxygen delivery to the brain of the foetus during birth. Hypoxemia occurs when there is a decrease in oxygen levels in the blood of the foetus, and ischemia is a lack of blood flow to the brain. The compensating response of the foetus to hypoxia is to redistribute oxygenated blood to more deserving organs, such as the heart and the brain. The redistribution of blood in the brain itself ensures that during

[1] Microcephaly is when the brain develops abnormally, causing the head of the child to be smaller than expected for the child's age.

episodes of prolonged foetal hypoxia, blood is directed to vital brain structures at the expense of less metabolically active structures of the brain, namely the cerebral cortex and the white matter. This protects the brain stem, cerebellum and deep grey matter from injury where the vital centres of life, like the respiratory and cardiac centres, are situated. The pattern of the insult that results from the directing of blood from the areas of the brain around the vital brain structures presents itself as an injury to the peripheral watershed areas of the brain where the white matter of the brain is situated. The mechanism responsible for the injury means that the injury takes place over a long period of time, and can last for several hours.

[5] According to the literature to which the respondent's paediatric neurologist made reference to in his evidence, Nonketotic Hyperglycinemia (NKH) is a rare genetic metabolic disorder caused by a defect in the enzyme system responsible for breaking down the amino acid, glycine, in the body. The defect in the system results in an accumulation of glycine in all the body tissues, including the brain. There is a classical and a variant form of NKH. Classic NKH, which is the relevant form in the present context, is caused by genetic variants in the genes of a person, and is an inherited condition. Based on its ultimate outcome, classic NKH can be categorised as either severe, or a mild form of the disorder. The disorder may be diagnosed by elevated glycine in blood plasma and a compatible pattern of brain imaging. Increased levels of glycine in the spinal fluid is highly indicative of NKH, and is consequently the preferred diagnostic test. Based on the extent of NKH, a sufferer may present with intractable epilepsy and no developmental progress, or in its attenuated form, with variable developmental progress and treatable epilepsy, or an absence of epilepsy.

[6] The appellant led evidence from three witnesses, namely Professor Lotz, Dr Murray and Prof van Toorn. Professor Lotz is a qualified radiologist. Dr Murray is an obstetrician and a gynaecologist, and Prof von Toorn is a paediatric neurologist. The defendant in turn adduced the evidence of a paediatric neurologist, Dr Keshave. The two neurologists testified to the possible causes of DMA's condition. In addition to the oral evidence of these witnesses, the parties placed before the trial court the joint

minutes of van Toorn and Keshave, as well as that of the respective radiologists; obstetricians; and geneticists from whom the parties had commissioned expert witness reports. The expertise of all the witnesses in their respective fields was not placed in issue. Except for one or two aspects, the clinical evidence on which the expert witnesses based their opinions, was similarly not in dispute.

[7] In its assessment of the evidence placed before it, the trial court proceeded from the premise that the respondent had admitted that DMA suffered a brain injury, and that due to the negligence of the respondent's employees, the foetus was deprived of oxygen during the birth process. The issue to be decided, said the court, was whether or not the oxygen deprivation during birth was the cause of the brain impairment which DMA was diagnosed to be suffering from when the action was instituted. In short, was the cause of the admitted injury oxygen deprivation as contended by van Toorn, the appellant's expert neurologist, or was it NKH as opined by Keshave, the respondent's expert witness?

[8] The trial court concluded that on the evidence placed before it, it could not decide to its satisfaction where the truth lay, and ordered absolution from the instance. It found that where the expert opinions presented by the respective parties with regard to the cause of DMA's injury were both capable of logical support, the matter should not be decided by a simple preference of one opinion over the other, and that in the present matter, Keshave's opinion could not be found not to have a logical basis. This finding was undoubtedly based on the decision in Michael and Another v Linksfield Park Clinic (Pty) Ltd and Another,[2] where the court dealt with a conflict in expert evidence with whether the diagnosis and treatment in question accorded with sound medical practice. It held that **"… it would be wrong to decide a case by simple preference where there are conflicting views on either side, both capable of logical support."**[3] Whilst the nature of the conflict in the expert opinion in Linksfield was different from the one in the present matter, the principle expressed equally finds application.

---

[2] 2001 (3) SA 1188 (SCA) (Linksfield) at para [37].
[3] Supra at para [39].

[9] The trial court correctly in my view approached the issue for determination on the basis of there being two conflicting expert opinions with regard to what the factual cause of DMA's injury was. The question in this appeal is accordingly whether the court was correct in concluding that the two opinions were equally placed on the evidence before it, and that the appellant must as a consequence be found not to have discharged the burden of proving that it was the respondent's negligence that was the cause of the injury sustained by DMA. Before I proceed to determine this issue, it may be convenient to say something about how expert opinion evidence is to be approached and evaluated when there is conflicting or inconsistent evidence from two or more expert witnesses.

[10] The opinion of a witness is generally inadmissible. "**In the law of evidence 'opinion' means any inference from observed facts, and the law on the subject derives from the general rule that witnesses must speak only to that which was directly observed by them."**[4] Opinion is admissible if it is relevant. Relevance is in turn determined by the issues in the matter. If the opinion can assist the court in determining an issue, is has probative value, otherwise it is superfluous.[5] Expert opinion evidence is received when the issues require special skill and knowledge to draw the right inferences from the facts stated by the witnesses.[6]

[11] Conceptually there are several types of conflicts in expert evidence that may present itself in any given case. The first is a conflict with regard to the assumed facts. By reason of its very nature, expert opinion must have a factual basis. The facts upon which an expert's opinion is based must be proved by admissible evidence. An expert opinion based entirely on inadmissible evidence is itself inadmissible. The facts may be established by asking the expert witness in examination-in-chief what those facts are.[7]

---

[4] Cross on Evidence 7th Ed at page 489. See also Schmidt and Rademeyer Law of Evidence at page 17 – 4 and McGregor and Another v MEC for Health Western Cape (1258/2018) [2020] ZASCA 89 (31 July 2020) (McGregor) at para [21].
[5] Ruto Flour Mills (Pty) Ltd v Adelson (1) 1958 (4) SA 235 (T) at 237 A – B. See generally Schwikkard v van der Merwe Principles of Evidence 3rd Ed at page 83 and 87.
[6] Menday v Protea Assurance Co Ltd 1976 (1) SA 565 (E) at 569 and Coopers (South Africa) (Pty) Ltd v Deutsche Gesellschaft Für Schädlingsbekämpfung Mbh 1976 (3) SA 352 (A) (Coopers) at 370 F – G.
[7] Cross op cit at page 494. See also Schmidt and Rademeyer op cit at page 17 – 14.

"**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."**[8] How those facts are proven is determined by the principles of evidence and the usual methods used for judicial fact-finding and rational decision-making. Where the expert him or herself observed relevant facts, that evidence will be evidence of fact and admissible as such.[9] Where the opinion seeks to take issue on the facts with the version of direct eyewitness evidence, credible eyewitness evidence that conforms to the probabilities, will generally take preference to the opinion of an expert of what the facts are.[10] In the final result, the decision of what the facts are must be founded on an assessment of the evidence as a whole and the probabilities as they appear therefrom.[11]

[12] Secondly, a conflict in the expert opinion may lie in the analysis of the established facts and the inferences drawn therefrom by opposing expert witnesses. A proper evaluation of the evidence in this context focuses primarily on "**the process of reasoning which led to the conclusion, including the premise from which the reasoning proceeds…"**[12] The reason for interrogating the underlying premise of expert opinion lies in its nature. In essence it amounts, as in the present context, to a statement that established medical opinion, as the expert witness interprets it, dictates a particular result under an assumed set of facts. This requires an assessment of the rationality and internal consistency of the evidence of each of the expert witnesses.[13] "**The cogency of an expert opinion depends on its consistency with proven facts**

---

[8] Coopers at 371 F-H.
[9] AM and Another v MEC for Health, Western Cape (1258/2018) [2020] ZASCA 89 (31 July 2020) at para [17].
[10] Mapota v Santam Versekeringsmaatskappy Bpk 1977 (4) SA 515 (A) from 527 to 528; Stacey v Kent 1995 (3) SA 344 (E) (Stacey) at 348 to 349; Motor Vehicle Assurance Fund v Kenny 1984 (4) SA 432 (E) and Representative of Lloyd's and Others v Classic Sailing Adventures (Pty) Ltd 2010 (4) All SA 366 (SCA) at para [60].
[11] Stacey supra.
[12] Coopers at 371 H.
[13] Linksfield supra; Oppelt v Department of Health 2016 (1) SA 325 (CC) (Oppelt) at para [36]; and Masstores (Pty) Ltd v Pick'n Pay Retailers (Pty) Ltd and Another 2016 (2) SA 586 (SCA) at para [15].

and on the reasoning by which the conclusion is reached."[14] The source for the evaluation of this evidence for its cogency and reliability are (i) the reasons that have been provided by the expert for the position adopted by him/her; (ii) whether that reasoning has a logical basis when measured against the established facts; and (iii) the probabilities raised on the facts of the matter.[15] It means that the opinion must be logical in its own context, that is, it must accord with, and be consistent with all the established facts, and must not postulate facts which have not been proved.[16]

[13] The inferences drawn from the facts must be sound. The internal logic of the opinion must be consistent, and the reasoning adopted in arriving at the conclusion in question must accord with what the accepted standards of methodology are in the relevant discipline.[17] The reasoning will be illogical or irrational and consequently unreliable, if (i) it is based on a misinterpretation of the facts; (ii) it is speculative, or internally contradictory or inconsistent to be unreliable; (iii) if the opinion is based on a standard of conduct that is higher or lower than what has been found to be the acceptable standard; (iv) if the methodology employed by the expert witness is flawed. What flows from this is that the mere fact that an expert opinion is unchallenged, does not necessarily mean that it must be accepted. However, if that evidence is based on sound grounds and is supported by the facts, there exists no reason not to accept it.

[14] Other considerations relevant in this context are (i) the qualifications and the experience of the expert witnesses with regard to the issue he or she is asked to express an opinion on; (ii) support by authoritative, peer-reviewed literature;[18] (iii) the measure of equivocality with which the opinion is expressed; (iv) the quality of the investigation done by the expert; (v) and the presence or absence of impartiality or a lack of objectivity. What is ultimately required is a critical evaluation of the reasoning on

---

[14] MEC for Health and Social Development, Gauteng v TM obo MM (380/2019) [2021] ZASCA 110 (10 August 2021) at para [125]. Also Buthelezi v Ndaba 2013 (5) SA 437 (SCA) (Buthelezi) at para [14].
[15] Oppelt supra at para [35].
[16] MEC for Health and Social Development, Gauteng v TM obo MM supra at para [126] and BEE v Road Accident Fund 2018 (4) SA 366 (SCA) at para [23].
[17] Schwikkard and van der Merwe op cit at page 99 and the authorities referred to in fn 102.
[18] AN v MEC for Health, Eastern Cape [2019] 4 All SA 1 (SCA) at para [22] and MEC for Health and Social Development, Gauteng v TM obo MM supra at para [126].

which the opinion is based, rather than considerations of credibility.[19] Should it not be possible to resolve a conflict in the expert opinion presented to the court in this manner, that is, when the two opposing opinions are both found to be sound and reasonable, the position of the overall burden of proof will inevitably determine which party must fail. It is worth emphasising that the onus as a determining factor **"can only arise if the tribunal finds the evidence pro and con so evenly balanced that it can come to no such conclusion. Then the onus will determine the matter. But if the tribunal, after hearing and weighing the evidence, comes to a determinate conclusion, the onus has nothing to do with it, and need not be further considered."**[20]

[15] A third type of conflict which may arise in expert evidence, is that of competing theories of a purely scientific nature. The choice between two conflicting theories is informed primarily by the extent to which the theory is regarded as being established and has gained general acceptance within the specific scientific community in the particular discipline to which it belongs. Whether or not a theory has been sufficiently established must be measured against considerations such as whether it can, and has been tested; whether it is the product of reliable principles and methods that have been reliably applied to the facts of the case; and whether it has been subjected to peer review and publication.

[16] Fourthly, a conflict may also arise in the context of what the accepted standard of conduct of a medical professional is in certain circumstances. Typically medical negligence cases deal with the situation where an injury is alleged to be in complete discord with the recognised therapeutic objective and techniques of the operation or treatment involved. Expert opinion, in this context, is aimed at determining whether the conduct of a professional person in a particular field accords with what is regarded as sound practice in that field. Again, the method adopted is to evaluate opinion evidence

[19] Oppelt supra at para [36].
[20] Robins v National Trust Co (4) [1927] AC at 520.

with the view of establishing the extent to which the opinions advanced are founded on logical reasoning. [21]

[17] What is evident from the aforegoing is that 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.[22] 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 and critical evaluation thereof.[23] Further, the primary function of an expert witness is to guide the court to a correct decision on questions, which fall 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.[24] Accordingly, the mere **"… pitting of one hypothesis against another does not constitute the discharge of the functions of an expert."[25]** 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.[26] *Ex hypothesi*, such evidence is outside the learning of the court. The function of the court is restricted to deciding a matter on the evidence placed before it by the parties, and to choose between conflicting expert evidence, or accepting or rejecting the proffered expert evidence.

---

[21] Linksfield supra at para [37] and [38]; Medi-Clinic v Vermeulen 2015 (1) SA 241 (SCA) (Medi-Clinic) at paras [4] to [8] and MEC for Health and Social Development, Gauteng v TM obo MM supra at para [125].
[22] Van Wyk v Lewis 1924 AD 438 at 447; S v Gouws 1967 (4) SA 527 (E) at 528D and Buthelezi supra at para [14]. See also Schmidt and Rademeyer op cit at page 17 – 16.
[23] Medi-Clinic supra at para [5].
[24] 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].
[25] Stacey supra at 350 G-H.
[26] MEC for Health, Eastern Cape v ZM obo LM (576/2019) [2020] ZASCA 160 (14 December 2020) at paras [12] and [13].

[18] Turning to the issue raised in this appeal, the conflict essentially lies in the opinion evidence of the two neurologists with regard to the cause of the injury. Van Toorn's evidence was that the nature of the injury, as it was identified by the radiologists, is consistent with the clinical records of what transpired during the appellant's labour, creating the risk of a hypoxic ischemic insult, and the clinical condition of DMA immediately after birth. Keshave's hypothesis of the cause, being NKH, was in essence premised on the following aspects: (i) the clinical features exhibited by DMA after birth; (ii) the fact that DMA was diagnosed with a type of cerebral palsy that is not usually associated with intrapartum asphyxia; (iii) in contrast to the magnetic resonance imaging (MRI) scan on which the radiologists based their opinion, the report of a radiologist on an earlier computerised tomography (CT) scan taken a year and 6 months after the birth of DMA, showed damage to only the one side of his brain, which in keeping with NKH, this is indicative of an injury that is progressive in nature; and (iv) elevated levels of glycine found in DMA's blood plasma.

[19] On a critical assessment of Van Toorn's evidence with regard to the cause of the injury, when measured against that of Keshave, his evidence proves to be more reliable with there being nothing, at least not of a material nature, that may detract from the probative value thereof. His evidence not only had a logical basis, but the conclusions reached by him were well reasoned, and consistent with the clinical evidence, the joint reports of the expert witnesses engaged by the respective parties, the oral evidence of the other expert witnesses, and the probabilities as the they arose therefrom.

[20] The salient features of the clinical evidence on which Van Toorn and the appellant's other expert witnesses based their opinions were in summary that the appellant gave birth to DMA by way of an emergency caesarean section. The reason for the absence of a natural delivery was noted as having been cephalopelvic disproportion, that is, the foetal head was too large, or in a position, that rendered it difficult to pass through the pelvis. DMA was born a week past the determined delivery date. During labour, the foetal heart rate was recorded as being irregular. The appellant's labour progressed poorly, and she was in labour for 35 hours and 45

minutes, of which 11 hours and 15 minutes were in the latent phase, and 24 hours in the active phase of the first stage of labour. The appellant's amniotic fluid liqour, when released by the rupture of the membranes was meconium stained. Meconium is foetal bowl content that is expelled into the amniotic fluid. The nursing staff administered oxytocin, a drug that augments delivery by causing an increase in contractions. DMA was born with a caput, being a soft tissue swelling on the head of a new born baby.

[21] At birth DMA had a lack of muscle tone, and he had to be resuscitated with oxygen for ten minutes. He was nursed in an incubator. He vomited when cup fed, but retained later feeds. The following day DMA was reported to have had a seizure, described as **"twitching on the right of the body."** Medication was prescribed and administered intravenously on two more occasions. He was discharged from the hospital after five days. He received physiotherapy from which he benefitted. At the age of one, a head measurement noted a head circumference indicative of substandard head growth. Six years later DMA was diagnosed with cerebral palsy. He presented with seizures on a number of occasions, and was diagnosed as having epilepsy. His epilepsy was treated with medication.

[22] The parties placed two joint minutes from three radiologists into evidence. Two of the radiologists, namely Alheit and Lotz, were engaged by the appellant, and the third, Dr Ahmed, by the respondent. In the earlier of the two joint minutes, Lotz and Ahmed agreed on the following: (i) that the MRI scan demonstrates features which are, in the appropriate clinical context, compatible with a prolonged partial hypoxic ischemic insult to the brain; (ii) that the injury asymmetrically involves the posterior watershed regions of the brain; (iii) that in view of extensive occipital white matter damage, the clinical records should be evaluated for possible episodes of low blood sugar (hypoglycaemia) postnatally; and lastly, (iv) that genetic disorders as a cause of the brain damage was unlikely, according to Lotz, and less likely, according to Ahmed.

[23] In the joint minute of Alheit and Ahmed there are four features that stand out. The first is that they were in agreement that the imaging of the MRI scan of DMA's brain

presented a brain injury, which was the likely result of a prolonged partial hypoxic ischaemic cerebral insult. Alheit, set the likelihood of that being the position as **"exceedingly high."** The second feature is that the presence of ulegyria makes it likely that the injury is the result of a prolonged partial hypoxic ischaemic insult. Ulegyria refers to mushroom shaped scarring of the brain, indicative of dead brain cells. The third feature is that the two radiologists agreed that the brain imaging is consistent with additional hypoglycaemic encephalopathy. That is, additional brain damage caused by episodes of low blood sugar. Alheit was of the opinion that the dominant injury was exceedingly likely to be prolonged partial hypoxic ischemic in nature, while Ahmed did not want to commit himself on which one of the two was the dominant insult simply on the imaging, and in the absence of clinical data. The fourth, and an important feature of the joint minute, is that both radiologists concluded that the findings of the MRI study rendered genetic disorders as a cause of DMA's brain damage unlikely.

[24] The two obstetricians, Murray and Janowski, agreed that (i) the appellant was more than 41 weeks pregnant when she was admitted to the maternity ward; (ii) that her labour must be regarded as having been severely prolonged by reason of a prolonged active phase of the first stage of labour; (iii) that the foetus was at an increased risk of intrapartum hypoxia due to the prolonged labour; and (iv) that the administering of oxytocin to augment the appellant's labour; the signs of cephalopelvic disproportion; and the prolonged active phase of the first stage of labour, were all indicators for the need to perform a caesarean section to prevent injury to the foetus.

[25] The question whether there was a genetic cause for DMA's condition, was addressed by the two geneticists in a joint minute. Dr Gericke and Prof Christianson were in agreement that there was no evidence of dysmorphic features present with DMA associated with any recognisable genetic condition or syndromic disorder. That would include dysmorphic features of alcohol syndrome, or partial foetal alcohol syndrome. Gericke pertinently noted that DMA's ears were not low set, and that the presence of minor epicanthic folds could be a normal variant physical feature. Neither of the geneticists expressed the need for further testing to be conducted. They instead

deferred to the other experts in their respective fields with regard to the cause of DMA's condition.

[26] The two-paediatric neurologists, Van Toorn and Keshave authored two joint minutes. On a reading of the preamble to the second joint minute, the reason for the compilation of a second joint minute lies in the fact that Keshave in the first written summary of his evidence, expressed the opinion that certain facts, such as the presence of occipital white matter injuries, and the deceleration of DMA's head growth, may possibly be due to other causes, which may include genetic metabolic disorders. Subsequently, metabolic tests were performed which *inter alia* noted that DMA had elevated glycine levels, followed by a recommendation that further testing for glycine be performed to rule out the presence of NKH. As a consequence, the focus of the second minute between Van Toorn and Keshave was whether NKH could be considered as being a cause of DMA's condition.

[27] Some of the aspects on which van Toorn and Keshave were in agreement with in their joint minutes, and which are relevant in the context of the issue raised, are as follows: The pattern of the injury to DMA's brain, as shown by the MRI images, was compatible with a prolonged partial hypoxic ischemic injury. The developmental outcome associated with intrapartum asphyxia was either spastic quadriplegia or dyskinetic cerebral palsy. Other subtypes of cerebral palsy were less likely to be associated with an intrapartum hypoxic ischemic event. DMA has a left sided hemiplegia. In the case of children with hemiplegic cerebral palsy, the left cerebral hemisphere is usually normal. In the case of DMA, his brain injury presented with damage to both the right and the left, although the left side was damaged to a lesser extent. Van Toorn and Keshave agreed that the reason why he was not manifesting quadriplegia, is **"simply that his left motor area was spared from hypoxic ischemic hypoglycaemic injury."** They further agreed that a diagnosis of NKA should not be based on a single elevated serum glycine level, and that the deceleration of DMA's head growth since birth supports an early hypoxic ischemic injury. Finally, they agreed

that features such as apnea, poor feeding and the early onset of seizures, were compatible with both HIE and NKH.

[28] The oral testimony of the radiologist, Professor Lotz was that the MRI scan of the brain of DMA presented a typical example of a watershed injury, which injury is the hallmark of a prolonged partial hypoxic ischemic insult. It is a pattern of injury that occurs at 36 weeks of gestation onwards. The injury is situated at the posterior of DMA's brain, on both the left and the right side. The injury on the right is more severe and more prominent. According to Lotz, the brain injury is not reflective of having been caused by a genetic disorder, inflammation or infection, and there can be no doubt that it is a watershed injury, which could only have been caused by a prolonged partial hypoxic ischemic insult. The reason is to be found in the fact that neurological injuries have certain defined patterns. By evaluating the pattern presented by imaging, neuroradiology as a field of expertise, is able to draw a conclusion with regard to the cause of a brain injury. Accordingly, if the injury was caused by a genetic disorder or such, the injury would have presented itself with a different pattern, and would have been identifiable as such by a radiologist. Lotz testified that MRI is an advanced technology that gives radiologists the ability to more accurately diagnose the nature of a brain injury, and that it simply cannot be compared to older and more limited technology, such as a CT scan.

[29] The evidence of Lotz with regard to the nature of the brain injury sustained by DMA was not disputed in cross-examination. Rather, the focus of the respondent's questioning of this witness was limited to two aspects. The first was whether or not the injury could have been caused subsequent to the birth process, without proposing what other likely cause may account for the type of injury diagnosed by the three radiologists. In particular, Lotz was not asked whether the injury could have been caused by an accumulation of glycine in the brain of DMA as a result of him suffering from NKH. The importance of the failure of this having been put to Lotz, must be assessed in the context of all three radiologists having agreed on the nature of the injury; the uncontested evidence of Lotz of when and how such an injury is sustained; and

importantly, the evidence of Keshave, with reference to the literature on which he relied, that the injury produced to the brain of someone suffering from NKH has an identifiable profile when it is viewed on an MRI scan. The second aspect raised with Lotz in cross-examination was that he was not qualified to express an opinion on the statement that **"not all hypoxic ischemic injuries result in cerebral palsy."** Lotz responded to the question by firstly making the concession that there may be other causes for cerebral palsy, and that with regard to what those causes may be, should more appropriately be dealt with by someone with the necessary expertise, namely a neurologist.

[30] There is nothing that detracts from the reliability of the evidence of the plaintiff's obstetrician, Dr Murray, and none was suggested. Her evidence was consistent with the respondent's admission on the pleadings that the nursing staff allowed the appellant's labour to be unduly prolonged, and that it caused DMA to sustain a hypoxic ischemic injury. Murray expressed the opinion that during labour there were several reasons for the foetus to have become hypoxic. These were as follows. Delivery took place at about 41weeks and 3 days, which meant that the appellant was at least a week over her due date. A foetus that is delivered late may not be able to manage the stresses posed by labour as well as a foetus that is born earlier. The fact that the appellant was over her due date necessitated the need for careful monitoring of her labour, which did not happen. According to Murray, a striking feature of the appellant's labour was the length thereof. She testified that she had never seen a labour as long as that of the appellant. It meant that she gave birth nearly 24 hours later than what was expected according to recommended norms. She was 8 centimetres dilated for 18 hours, a length of time, which Murray described as being incredibly long. According to Murray, it must have been fairly obvious at an early stage of labour that delivery of the baby was obstructed, in that the foetal head was too big to progress through the maternal pelvis.

[31] Further, the appellant's liquor was meconium stained. As stated, meconium refers to the faeces of the foetus, which is passed in the uterus, and said Murray, it is considered to be a response to stresses during labour. Instead of performing a caesarean section, the appellant was given oxytocin. That, Murray said, would have had

the effect of increasing contractions, which in turn would have had the effect of placing further stress on the foetus. She explained that during labour the foetus would naturally undergo brief bouts of hypoxia when blood flow is interrupted by the mother's contractions. The foetus is naturally well equipped to deal with this. However, if the contractions are too strong, occur too often and carry on for too long, it may be harmful to the foetus.

[32] Oxytocin, said Murray, is a dangerous drug that must be used cautiously. If not properly monitored, it may cause contractions which may be too much for the foetus to deal with. On a whole, Murray's evidence was that the foetus was likely in distress during labour, and that all the risk factors for hypoxia were present. This, according to Murray, is a reflection of a labour that was grossly mismanaged, poorly monitored, and allowed to continue for too long before a caesarean section was performed despite the obvious presence of an obstructed labour and a non-reassuring foetal condition.

[33] In his oral testimony, Van Toorn agreed with the obstetrician, Dr Murray, that the risk of this type of injury was created by (i) the severely prolonged obstructed labour that was characterised by poor progress; (ii) the administering of oxytocin under those circumstances; (iii) the presence of meconium stained liquor; and (iv) the advanced gestational age and the poor monitoring of the appellant's labour. Clinical evidence of the type of injury identified by the radiologists was the fact that DMA was not breathing at birth, and had to be resuscitated with oxygen. Van Toorn testified that the most common cause of the seizures suffered by DMA shortly after birth were hypoxia ischemia and hypoglycaemia, both of which cause the death of cells in the brain, as is evidenced by the MRI imaging of DMA's brain. His further evidence was that the presence of ulegyria in the MRI imaging was further consistent with hypoxia ischemia and hypoglycaemia.

[34] Van Toorn was asked to deal with Keshave's opinion that DMA has an underlying neurometabolic disorder that may account for his clinical condition. According to van Toorn there exists identifiable differences between an injury caused by

a hypoxic insult, and that which is caused by a neurometabolic disorder, such as NKH. Both causes would present itself with identifiable abnormalities that will be evident from an MRI scan. In the instant matter, the damage to the brain of DMA as seen on the MRI scan, and confirmed by all the radiologists, was consistent with a hypoxic ischemic brain injury. The changes to DMA's brain was of a destructive nature, which was supported by the presence of ulegyria, something that is seen with a hypoxic ischemic brain injury. Importantly, this type of injury is static in nature, whereas a metabolic disorder is progressive, in that it raises glycine levels in the body tissues causing damage over time. Consequently, it presents with conditions which get worse over time, and a regression of developmental milestones.

[35] With regard to the expert report on an earlier CT scan, on which Keshave relied for arriving at his opinion that the brain injury was of a progressive nature in keeping with NKH, van Toorn agreed with Lotz that it does not necessarily exclude the existence of an injury to both sides of the brain having been present as indicated on the later MRI scans. He agreed with the undisputed evidence of Lotz that MRI is a much more advanced technology when compared to a CT scan, which is a much older and more limited technology. The limitations posed by a CT scan, and the likelihood postulated by these two witnesses that the injury to the one side of the brain did not show on the CT scan, is supported by the fact that the injury to DMA's brain was found by the radiologists to have been more pronounced on the right side of the brain than on the left.

[36] Van Toorn further testified that although the type of epilepsy which DMA has presented is less likely associated with the type of injury identified by the radiologists, the fact is that his brain was damaged on both sides, and the presence of only a left sided hemiplegia, as in the present matter, is not inconsistent with the other side of the brain having been able to overcome the injury. This is something, according to van Toorn, that is typically seen in children. With regard to the elevated glycine levels, the most likely cause of this, said van Toorn, was the fact that DMA was using medication

for his epilepsy. The medication in question is known to raise the glycine levels in the blood of its users.

[37] Van Toorn stood firm in his opinion that although some features, like apnea and seizures after birth are similar to HIE and NKH, there are marked differences between the clinical profile of the two, and that NKH could be excluded as a cause of DMA's injury. The imaging of the MRI scan was also not in keeping with NKH, and there was no need, according to him, to perform any further tests to exclude a neurometabolic disorder as a cause of DMA's condition. The accepted medical practice, according to him, is that further testing is only required if there is a clinical presentation of NKH, which was absent in this case. If DMA was suffering from a milder form of NKH, symptoms of the condition, according to the publications on which Keshave had based his opinion, would only have manifested itself after 3 months. That was not the case with DMA. Also, visual impairment and the making of unusual movements, which were similarly absent, would have been indicative of the presence of NKH. Further, NKH causes symmetrical spasticity. That was similarly absent in the case of DMA. According to van Toorn, NKH is a very rare metabolic condition that he has only seen twice in his 20 years of experience.

[38] That brings me to Dr Keshave's evidence. A closer examination of his expressed opinion on what the cause of DMA's injury and clinical condition is, shows that it does not support a defensible conclusion when measured against the facts and the other largely uncontested evidence. He wavered between different opinions; he sought to draw inferences which were tenuous; he expressed views on aspects he was not qualified to do, and he gave evidence that was largely of a speculative nature. I will proceed to illustrate this. For a start, in the written summary of his expert opinion he expressed the opinion that there exists **"the possibility of an underlying genetic/neurometabolic disorder"** that needed to be considered. He later supplemented his written report by the positive statement that DMA **"has an underlying neurometabolic disorder"** that could account for his current clinical condition. In his oral testimony he ended up saying, in cross-examination, that the

possible cause of DMA's neurological impairment may either be an underlying neurometabolic disorder or a hypoxic injury.

[39] The profound change in opinion in his written opinions was premised on the results of the blood test showing that DMA had elevated serum glycine levels. This fact alone could not in my view support the inference, which Keshave sought to draw in the summary of evidence. As stated, it was an accepted fact that DMA was prescribed medication for his epilepsy that is known to raise glycine levels in the body. As Van Toorn, as a matter of logic pointed out in his evidence, he would not have expected this type of medication to have been prescribed by a medical practitioner if DMA's glycine levels were already raised by reason of him suffering from NKH. It would only have exacerbated his seizures, of which there was no evidence. Further, Keshave in his subsequent joint minute with van Toorn, agreed that a diagnosis of NKH should not be based on a single elevated level of blood serum glycine. He however sought to speculate in the minute, and again in his oral testimony, that DMA's caregivers may not as regularly have given him his medication as they were supposed to, the suggestion being that the reason for the raised glycine levels must lie elsewhere. This is nothing more than speculation. Inferential reasoning is an accepted technique that is utilised in judicial fact-finding. However, the inference sought to be drawn must be capable of being drawn from the admitted or proven facts, and not matters of speculation.[27] If tenuous, or far-fetched, it cannot form the foundation for the court to make any finding of fact.[28] Further, the inference must be based on, and be consistent with all the admitted or proved facts, and not be matters of speculation.[29]

---

[27] The inference must be the readily apparent and acceptable inference from a number of possible inferences. See AA Onderlinge Assuransie Bpk v De Beer 1982 (2) SA 603 (A) at 620 E – G; Cooper and Another NNO v Merchant Trade Finance Ltd 2000 (3) SA 1009 (SCA); Goliath v MEC for Health, Eastern Cape 2015 (2) SA 97 (SCA) and M and Another v The MEC for Health, Western Cape (1258/2018) [2020] ZASCA (31 July 2020) at para [21]. **"Evidence does not include contention, submission or conjecture."** Great River Shipping Inc v Sunnyface Marine Limited 1994 (1) SA 65 (C) at 75 I – 76 C.

[28] Imperial Marine Co v Deiulemar Compagnia Di Navigazione Spa 2012 (1) SA 58 (SCA) at para [24] and Motor Vehicle Assurance Fund v Dubuzane 1984 (1) SA 700 (A) at 706 B – D.

[29] McGregor supra at para [21].

[40] Keshave's opinion that the nature of the injury to DMA's brain is supportive of a conclusion that he is suffering from NKH, is not supported by the evidence. The fact that DMA after birth presented with features such as apnea and seizures, standing alone, is insufficient to draw an inference for a diagnosis of NKH. It was agreed by Keshave to be equally consistent with HIE. The other factors he sought to place reliance on, also do not serve to justify such an inference. As stated earlier, he *inter alia* sought to base his opinion on the report of an earlier CT scan that only made mention of an injury to the one side of the brain. He further testified that there were features on the later MRI scan, such as the posterior areas of the brain that were damaged, which was consistent with the features of an injury that was caused by NKH. The difficulty with this is that Keshave's reliance on the report of the CT scan clearly did not account for the inferior quality of the technology involved, a fact that was not disputed. It is further inconsistent with the fact that he agreed with Van Toorn in their first joint minute that the changes to DMA's brain were compatible with a prolonged partial hypoxic ischemic injury, which injury Van Toorn testified is, otherwise than that caused by NKH, not a progressive, but a static injury. Further, Ahmed, the defendant's own expert radiologist, agreed with both Lotz and Alheit that the injury is consistent with a prolonged partial hypoxic ischemic cerebral insult, and that genetic disorders as a cause of the brain damage was unlikely.

[41] An important aspect in assessing the reliability of Keshave's opinion, is that Lotz was not referred to either the report, or the CT scan itself on which Keshave based his opinion, neither of which seemed to have been admitted into evidence. It could accordingly not be tested in cross-examination, and very little, if any, weight can be accorded to it. It was not suggested to Lotz that the injury was, or may have been progressive in nature, or that it is consistent with an injury caused by NKH. This failure must be assessed in the face of Lotz's undisputed evidence that neurological injuries have certain predictive patterns that can be identified from the imaging presented by a MRI scan. NKH, according to the literature on which Keshave relied, similarly presents with a compatible pattern of brain injury. As a radiologist, Lotz was clearly qualified to have dealt with this in his evidence and it should, of necessity, have been raised with him.

[42] It is further evident from Keshave's testimony that he expressed his opinion with regard to the cause of the injury, without having had regard to what either the radiologists, or the two geneticists, agreed to in their respective joint minutes. He surprisingly acknowledged in cross-examination to not having had sight of the joint minutes. Furthermore, the drawing of conclusions from the imaging of either the CT and MRI scans, or from the imaging contained in the publications on which Keshave sought to place reliance, clearly fell outside his field of expertise. This is an aspect which the defendant's radiologist should have dealt with in evidence. The defendant however chose not to call Ahmed as a witness and was consequently otherwise not allowed to simply depart from what the radiologists had agreed to in their joint minutes.

[43] In BEE v RAF[30] and the cases referred to therein, it was held that in the absence of a timeous repudiation, a party is bound to the facts agreed to in a joint minute, and that agreement reached on a matter of opinion, may likewise not simply be departed from. **"Where the parties engage experts who investigate the facts, and where those experts meet and agree upon those facts, a litigant may not repudiate the 'agreement unless it does so clearly and, at the very latest, at the outset of the trial' (para 11). In the absence of a timeous repudiation, the facts agreed by the experts enjoy the same status as facts which are common cause on the pleadings or facts agreed in a pre-trial conference (para 12). Where the experts reach agreement on a matter of opinion the litigants are likewise not at liberty to repudiate the agreement. The trial court is not bound to adopt the opinion but the circumstances in which it would not do so are likely to be rare (para 13). Sutherland J's exposition has been approved in several subsequent cases, including in a decision of the full court of the Gauteng Division, Pretoria, in Malema v Road Accident Fund [2017] ZAGPJHC 275 para 92."**

[30]BEE v Road Accident Fund supra at para [64]. See also Thomas v B D Saracens (Pty) Ltd [2012] ZAGPJHC 161.

[44] Keshave's evidence that the form of cerebral palsy with which DMA was diagnosed was not typical of an injury caused by a hypoxic ischemic type of insult must be assessed against the fact that he agreed with Van Toorn in their joint minute that the form of cerebral palsy was less likely, not that it is incompatible with the nature of the injury. He also agreed with Van Toorn that the reason why DMA was not manifesting a quadriplegia, was simply that his left sided motor area was spared by the injury. This is consistent with Van Toorn's oral evidence that DMA in all likelihood managed to overcome the injury on the other side of his brain.

[45] In its assessment of Keshave's evidence, the trial court credited him with the fact that he had performed a physical examination of DMA. The question is whether this placed him in a better position than Van Toorn to make a diagnosis. Apparently not. The court found that having physically examined DMA, Keshave was able to determine that he had dysmorphic features indicative of an underlying genetic disorder. DMA was however subsequently examined by the two geneticists, namely Dr Gericke and Professor Christianson who, as stated earlier, concluded that the child did not have any dysmorphic features of any genetic or syndromic nature. In fact, Gericke pointed out in the joint minute that DMA's ears were not low set as stated by Keshave in the written summary of his evidence, and that the epicanthic folds to which Keshave also made reference to, can be attributed to a normal variant physical feature. Christianson chose not to disagree with this observation of Gericke.

[46] In addition to having relied on incorrect facts, a feature of Keshave's testimony, as previously stated, is the speculative nature thereof. In the written summary of his evidence, he was of the opinion that the degree of weakness to the left side of DMA's body would indicate that the insult to his brain occurred after delivery, rather than as a result of a hypoxic injury during birth. His oral testimony on the contrary was that the cause of the injury could possibly have been either an underlying metabolic disorder, or a hypoxic injury, the timing of which he would leave to the radiologists. With regard to the clinical conditions presented by DMA, he was unable to say, **"How much of it could have been from his NKH, from the hypoxic injury, from the alcohol. I am not**

sure. I cannot put a definitive answer to that. However, I do not think you can rule it out." His mention of alcohol appears to have arisen from a note by a therapist that the appellant had consumed alcohol during her pregnancy. When it was pointed out to Keshave that the two geneticists have excluded any likelihood of the presence of foetal alcohol syndrome, he gave the unmotivated response that there are a range of disorders that may arise from the use of alcohol during pregnancy. What the range of disorders are, was not stated. He disagreed in a similar fashion, in cross-examination, with the two geneticists when it was put to him that they were in agreement that they could not find any evidence of a genetic or metabolic disorder. His response was simply that **"those are their opinions."** He gave a similar unreasoned response when confronted in cross-examination with the opinion the joint minute of the two radiologists that a genetic disorder as a cause of the injury was unlikely.

[47] From a reading of Keshave's evidence and what he agreed to in the two joint minutes with Van Toorn, it is evident that he was not able to raise NKH as a cause of the injury any higher than it being a mere possibility. The reason for this seems to lie in the fact that his approach to the issue was that he was required to say definitively, or at least with a measure of certainty, what the cause of DMA's condition was. His view that where the clinical features overlap between two possible causes, the net must be thrown wider before a diagnosis was made, appear to have been motivated by his **"fear … that we always put the disability that the child presents with as hypoxic ischemic injury and then forget to investigate everything else the child could have."**

[48] The fact is that Keshave was not required to say with scientific or medical certainty what the cause of the injury was. The most commonly employed technique for determining factual causation is the **"but for"** test. This means that the appellant had to prove on a balance of probabilities that, **"but for"** the negligent actions or omissions of the respondent, the injury would not have occurred.[31] The test for factual causation was explained simply and precisely by Lord Denning in Cork v Kirby MacLean Ltd:

[31] Lee v Minister for Correctional Services 2013 (2) SA 144 (CC) (Lee) at para [40].

"[I]f you can say that the damage would not have happened BUT FOR a particular fault, then that is in fact the cause of the damage; but if you can say that the damage would have happened just the same, fault or no fault, then the fault is not the cause of the damage."[32]

[49] The test need not be applied rigidly.[33] It also does not require factual causation to be determined with scientific precision.[34] The reasons for this is that factual causation is a requirement of the substantive law for delictual liability, and its existence is determined by the rules of evidence, more particularly, the legal standard set by the burden of proof. It accordingly requires a finding based on the legal standard of proof, and not a higher standard that requires proof with any scientific precision.[35] **"It has often been said that the legal concept of causation differs from philosophical and scientific notions of causation. That is because 'questions of cause and consequence are not the same for law as for philosophy and science', as Windeyer J pointed out in The National Insurance Co. of New Zealand Ltd v Espagne (1916) 105 CLR 569, at p 591. In philosophy and science, the concept of causation has been developed in the context of explaining phenomena by reference to the relationship between conditions and occurrences. In law on the other hand, problems of causation arise in the context of ascertaining or apportioning legal responsibility for a given occurrence. The law does not accept John Stuart Mill's definition of cause as the sum of the conditions, which are jointly sufficient to produce it. Thus, at law, a person may be responsible for damage when his or her wrongful conduct is one of a number of conditions sufficient to produce that damage."[36]**

---

[32] [1952] All ER 402 CA at 407.
[33] Lee v Minister of Correctional Services supra at para [44].
[34] Oppelt supra at paras (36) to (38). See also Minister of Safety and Security v Van Duivenboden 2002 (6) SA 431 (SCA); Minister of Finance and Others v Gore NO 2007 (1) SA 111 (SCA).
[35] Ocean Accident and Guarantee Corporation Ltd v Koch 1963 (4) SA 147 (A) at 157 C – D and Blyth v Van den Heever 1980 (1) SA 191(A) at 2088.
[36] Mason CJ in March v E and MH Stramare (Pty) Ltd [1991] 171 CLR 506 ([1991] HCA 12).

[50] The burden of proof in a civil case requires a plaintiff to prove his case no higher than on a balance of probabilities. The probabilities are determined upon the facts and an element of experience and common sense.[37] It calls for a sensible retrospective analysis of what would probably have occurred, based upon the evidence and what can be expected to occur in the ordinary course.[38] Applying the standard of proof to the test for factual causation, the enquiry is directed at identifying the more plausible of any one cause against the backdrop of the negligent act found proved, including the available evidence as a whole, which in a matter such as the present, will include, but is not limited to, expert opinion. In Linksfield[39] the court pointed to the fact that expert scientific witnesses tend to assess the likelihood of something in terms of scientific certainty, and stated that the **"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 200 SC (HL) 77 and the warning given at 89 D-E that '(o)ne cannot entirely discount the risk that by immersing himself in every detail and by looking deeply into the minds of experts, a judge may be seduced into a 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."**

[51] The aforementioned shortcomings in the evidence of Keshave were not considered by the trial court in its assessment of his evidence. This resulted in an uncritical acceptance of his evidence as having a logical foundation, a finding that cannot stand. Keshave's evidence was speculative and incapable of throwing any doubt on the otherwise acceptable expert opinion of the appellant that was based on sound grounds and supported by the basic facts. Material factual errors that no doubt led to the aforementioned conclusion of the trial court, were (i) that the result of the blood tests

[37] Za v Smith and Another [2015] 3 All SA 288 (SCA) at para [30]. Lee supra at para [39].
[38] Minister of Safety and Security v Van Duivenboden 2002 (6) SA 431 (SCA) at para [25]. See also Lee supra at para [39].
[39] Supra at para [40].

was that DMA was suffering from an underlying neuro-metabolic disorder, which accounted for his clinical position, as opposed to the result simply indicating that he had raised glycine levels in his blood; and (ii) that it was common cause that the imaging patterns of DMA's brain did not typically follow the prescribed patterns seen in perinatal HIE. This finding is inconsistent with what was agreed to by the radiologists. [40]

[52] The question is then whether the appellant had proved her case. On the whole, the clinical evidence supports the appellant's expert opinion that it is more likely than not that the brain injury sustained by DMA, and the disabilities that later followed, were the result of prolonged partial hypoxic ischemia during labour, as opposed to NKH. The injury is consistent with the conduct of the respondent in allowing a severely prolonged obstructed labour of the appellant to continue, which exposed the foetus to a real risk of sustaining a hypoxic type of brain injury. It further accords with the clinical condition of DMA immediately following his birth. The conduct of the respondent's employees created a risk of harm, and the more plausible explanation is that the injury occurred within that area of risk. The opinion of Van Toorn was further supported by the uncontradicted evidence of the radiologist Lotz, namely that the type of brain injury with which DMA had been diagnosed, could only have been caused by a prolonged partial hypoxia ischemic cerebral insult during the birth process. As stated, Ahmed, the respondent's own radiologist, agreed in his joint minutes with both Lotz and Alheit, not only with the diagnosis of the type of brain injury sustained by DMA, but also with the opinion that the likely cause of the injury was a hypoxic ischaemic insult.

[53] For these reasons, the appeal must succeed with costs, which costs are to include the costs occasioned by the postponement of the appeal on 2 August 2021 by reason of the respondent's failure to attend the hearing. The order of the trial court is set aside, and it is substituted with the following order:

"It is ordered that:

---

[40] See paras [22] and [23] of this judgment.

1. The defendant is liable to compensate the plaintiff in her representative capacity as the mother and natural guardian of DMA for the damages claimed in this action in such sum as may be agreed to or determined in due course.

2. The defendant pay the plaintiff's costs of the action, such costs to include the costs occasioned by the declaration hereby made that the plaintiff's expert witnesses who testified, including Dr G S Gericke, were necessary witnesses.

D VAN ZYL
DEPUTY JUDGE PRESIDENT OF THE HIGH COURT

I agree:

B MAJIKI
JUDGE OF THE HIGH COURT

I agree:

T MALUSI
JUDGE OF THE HIGH COURT

Counsel for the Appellant: Mr G Austin

Instructed by: GARY AUSTIN INC
c/o GORDON McCUNE ATTORNEYS

	140 Alexander Road KING WILLIAMS TOWN
Counsel for the Respondent:	Adv H J van der Linde SC Ms T Mqobi
Instructed by:	The State Attorney c/o SHARED LEGAL SERVICES OFFICE OF THE PREMIER 32 Alexander Road KING WILLIAMS TOWN