

SOUTH AFRICA / JUDGMENT

V.N obo M.N v Member of the Executive Council for Health, Eastern Cape Province (10/2020) [2025] ZAECBHC 16 (29 July 2025) [2025] ZAECBHC 16

COURT
Eastern Cape High Court, Bhisho

BEFORE
Laing

CASE / FILE NUMBER
10/2020

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IN

THE HIGH COURT OF SOUTH AFRICA

(EASTERN

CAPE DIVISION, BHISHO)

Not

reportable

CASE

NO. 10/2020

In

the matter between: V[...]
N[...] obo
M[...]
N[...]
Plaintiff
and
MEMBER
OF THE EXECUTIVE COUNCIL FOR
HEALTH,
EASTERN CAPE
PROVINCE
Defendant

JUDGMENT

LAIN J

- [1] This is an action for damages arising from the birth of the plaintiff's son, M, on 3 July 2012 at the Isilimela Hospital, in the district of Port St Johns.

Background

- [2] At the time of the incident, the plaintiff was 25 years old and had previously delivered. She underwent a prolonged labour before the birth of M; she pleaded that the failure of the medical staff to perform a caesarean section led to her son's experiencing a hypoxic incident, which caused damage to his brain and resulted in spastic quadriplegic cerebral palsy.
- [3] The plaintiff alleged negligence on the part of the medical staff, which the defendant eventually conceded. It was the defendant's case, however, that there was no causal connection between the conduct of its staff and M's condition.
- [4] At the commencement of trial, the court ordered that the question of liability be decided separately from that of quantum. The focus of the trial was the issue of causation. It is useful, at this stage, to describe the areas of common ground agreed upon by the experts, as set out in the joint minutes, and to provide a rudimentary indication

of the areas of difference.

Joint minutes

- [5] The radiologists, Dr Andre MacDonald and Dr Bates Alheit, agreed that the magnetic resonance imaging (MRI) brain scan displayed the features of a peripartum[1] hypoxic ischaemic injury (HII)[2] to the brain. In the absence of a sentinel event, the injury resulted from intermittent or prolonged hypoxia, with a severe, final episode occurring during labour. The radiologists agreed that it was unlikely that a genetic disorder or an infective disease was the cause of M's brain damage.
- [6] The obstetricians and gynaecologists, Dr Krzysztof Janowski and Dr Michael Wright, agreed that the plaintiff had been a high-risk

patient upon her admission to the Isilimela Hospital, requiring continuous electronic foetal monitoring (EFM). At 19h00, 3 July 2012, her dilation was 6 cm. The nursing staff administered oxytocin for purposes of uterine stimulation but failed to perform

cardiotocographic (CTG) monitoring[3] to detect tachysystole.[4] At 20h00, the plaintiff was fully dilated. This, said the experts, suggested hyperstimulation and inadequate monitoring. At 20h30, she gave birth to M, who presented with low Apgar scores,[5] required resuscitation, and developed hypoxic ischaemic encephalopathy (HIE).[6] The experts agreed that it was 'very likely' that a sub-acute hypoxic process began after the administration of oxytocin.[7] It was probable, too, that there had been an obstructed labour because of M's cephalopelvic disproportion.[8] In the absence of proper medical records, the experts could not rule out the possibility of undetected over-stimulation, leading to foetal distress; this, in turn, led to sub-acute hypoxia after the administration of oxytocin, which rapidly became acute. The MRI scan confirmed the presence of an HII. The available records indicated the onset of moderate to severe neonatal encephalopathy (NE) within 24 hours of delivery. There were several examples of sub-standard care, including inadequate monitoring, lack of clinical knowledge and skills, and non-compliance with the maternity guidelines. The experts agreed that the above factors had a bearing on causation.

- [7] The joint minute of the paediatricians reflected numerous areas of disagreement. Dr Yatish Kara believed that M had cerebral palsy with gross motor developmental delay; there were no indications of a genetic or chromosomal cause for the brain injury, but the MRI scan, the nature of the plaintiff's labour, and M's presentation at birth suggested that the cause was an HII that occurred during labour.
- [8] In contrast, Dr Amith Keshave believed that M had autistic spectrum disorder (ASD); an underlying genetic condition could account for his clinical presentation.
- [9] The differences in opinion between the paediatricians are critical to the determination of the present dispute. This will be discussed further in due course.

At trial

- [10] The plaintiff testified that her waters had broken during the early hours of 3 July 2012. She experienced abdominal pain which forced her to visit the Tombo Clinic where she was monitored. When the clinic closed, the plaintiff made her way to the Isilimela Hospital

where she was examined. Nurses monitored the foetal heart rate; the baby was far from delivery. A drip was set up and oxytocin was administered. Shortly afterwards, the plaintiff's contractions intensified; she was instructed to lie on her side. Eventually, she succeeded in pushing the baby's head out and called to the nursing staff for assistance. M was delivered in a compromised condition. A doctor arrived, turned the baby upside down, and slapped his feet. M did not cry. The doctor took the baby away for resuscitation.

- [11] The baby failed to achieve the usual growth milestones. M does not talk, but points to what he wants. He is unable to wash or dress himself. He can feed himself but is a messy eater. He is unable to walk properly.
- [12] The first of the expert witnesses called by the plaintiff was an obstetrician and gynaecologist, Dr Michael Wright. He testified about the contents of his report and the joint minute concluded with Dr Krzysztof Janowski. In that regard, he merely reiterated the sub-standard care that the nursing staff had provided to the plaintiff; this was not in dispute and his evidence was not contested.
- [13] The second expert witness was Dr Kara, after which the plaintiff closed her case. The only witness for the defendant was Dr Keshave. Their opinions will be discussed in the paragraphs that follow.

Issues

for determination

- [14] With the defendant's having conceded negligence on the part of the medical staff, what remains for determination is the issue of causation. As will be considered further, the question to be answered is whether the action (or omission) of the staff was a *conditio sine qua non* for the harm that was suffered by the plaintiff and her son. In other words, can it be said that, but for the conduct of the staff, no harm would have been suffered?
- [15] The conflicting views of the paediatricians, Dr Kara and Dr Keshave, play a central role in making the necessary determination. A problem that emerges, notwithstanding the availability of joint minutes, is what to make of Dr Keshave's opinion that M's clinical presentation is different to that which would have been expected from the nature and timing of the injury that occurred. Whether this was sufficient to have prevented the plaintiff from discharging the

evidential onus remains to be seen.

- [16] At this stage, only the defendant's liability must be decided. If it is so proved, then the issue of quantum will remain for determination in due course. An overview follows of the principles relevant to the matter at hand.

Legal framework

[17] As already mentioned, the issue of causation lies at the heart of the matter. In *Lee v Minister for Correctional Services*,^[9] the Constitutional Court, per Nkabinde J, dealt with the test for causation as follows:

‘...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.’^[10]

The learned judge proceeded to state that:

‘...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” . . .

. . . In the case of “positive” conduct or commission on the part of the defendant, the conduct is mentally removed to determine whether the relevant consequence would still have resulted. However, in the case of an omission, the but-for test requires that a hypothetical positive act be inserted in the particular set of facts, the so-called mental removal of the defendant’s omission. This means that reasonable conduct of the defendant would be inserted into the set of facts. However, as will be shown in detail later, the rule regarding the application of the test in positive acts and omission cases is not inflexible. There are cases in which the strict application of the rule would result in an injustice, hence a requirement for flexibility. The other reason is because it is not always easy to draw the line between a positive act and an omission. Indeed there is no magic formula by which one can generally establish a causal nexus. The existence of the nexus will be dependent on the facts of a particular case.’^[11]

Subject to the caveat mentioned by Nkabinde J, the but-for test remains the standard approach when dealing with the issue of causation. In *ZA v Smith and another*,^[12] Brand JA observed:

‘What it 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 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.’^[13]

[18] The test was reaffirmed in *Mashongwa v Passenger Rail Agency of South Africa*. [14] To that effect, the Constitutional Court held, per Mogoeng CJ, that Lee never sought to replace the pre-existing approach to causation. The question was whether there was a causal link between the defendant's

negligent conduct or omission and the plaintiff's injuries; it was also necessary to determine whether there was a close enough connection between the two elements. [15]

[19] Based on the above authorities, the standard approach is applicable in the present matter. The question is whether, but for the wrongful and negligent conduct of the medical staff at the Isilimela Hospital, M's brain injury would not have occurred. In other words, was such conduct a *conditio sine qua non* in relation to the harm suffered?

Discussion

[20] For disputes involving the complex issue of causation within a medical context, the contribution that can be made by an expert witness is potentially invaluable. In the present matter, the court has been presented with the opinion evidence of no less than six different medical specialists, whose views may prove to be decisive.

[21] 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:

'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]

[22] The above principles are particularly relevant to the different views advanced by the paediatricians, Dr Kara and Dr Keshave. The remaining experts, in contrast, reached agreement on key facts and opinions, as reflected in their joint minutes. These have provided much assistance in delineating the issues and curtailing the potential evidence required for the overall determination of the dispute.

[23] Beginning with the joint minute of the radiologists, the relevant portions stated as follows:

‘ . . . AM agrees with BA that the MR study displays chronic features related to a peripartum hypoxic ischaemic injury of the brain, as seen in an infant from 36 weeks gestation onwards.

. . . AM and BA agree that the imaging features indicate a BGT / central hypoxic ischaemic injury, which in the absence of an intrapartum sentinel event, most probably resulted from intermittent or prolonged hypoxia, with a more severe final episode once the circulation collapsed, which added the necessary ischaemic component, during labour.’

[24] From the above, it can be seen that the radiologists agreed on several factors: (a) M suffered an HII to his brain; (b) the injury occurred shortly before, during, or immediately after childbirth (i.e. peripartum); (c) it was a BGT type of injury; and (d) it most probably resulted from hypoxia during the labour process. The court must accept the above set of factors as a critical part of the factual matrix that overlays the present dispute. They can— and must— be used as fixed points against which the remaining opinion evidence can be measured.

[25] Regarding the obstetricians and gynaecologists, there was agreement that there had been sub-standard care. As with the radiologists, it is possible to identify certain key factors that they agreed upon:

- (a) there were no known antenatal risks; (b) the plaintiff became a high-risk patient after her labour failed to progress, requiring continuous EFM; (c) she was 6 cm dilated at 19h00 on 3 July 2012; (d) no assessment was carried out by a doctor; (e) the medical staff commenced with uterine stimulation by administering oxytocin, despite the plaintiff’s having had a normal previous delivery, and notwithstanding other contraindications; (f) the medical staff failed to carry out CTG monitoring to detect tachysystole, essential for ensuring that sufficient oxygen reached M’s brain where oxytocin had been used to augment labour; (g) there was no indication that the medical staff conducted foetal heart rate (FHR) monitoring, necessary for high-risk patients undergoing labour augmentation; (h) the plaintiff was 10 cm (fully) dilated at 20h00, suggesting hyperstimulation; (i) it was probable that the plaintiff’s labour was obstructed because of cephalopelvic disproportion; (j) at 20h30, she delivered M, who displayed low Apgar scores, required resuscitation, and developed HIE; (k) the early onset of moderate to severe neonatal encephalopathy was recorded within 24 hours of delivery;

- (l) M's condition at birth was compatible with intrauterine hypoxia; (m) it was 'very likely' that the sub-acute hypoxic process started after the administration of oxytocin at approximately 19h00; (n) the hypoxic process quickly became acute, prior to delivery at 20h30; (o) the scenario described was consistent with the acute profound HII revealed on the MRI scan; and
- (p) there were several elements of sub-standard care in the medical staff's management of the plaintiff's labour; and
- (q) such elements 'are probably related to causation'.

[26] The obstetricians and gynaecologist concluded their joint minute by stating that it was 'most likely' that peripartum hypoxic ischaemia played a role in causing M's neonatal encephalopathy. They went on to say that:

'... in view of the MRI report and maternity case records, we conclude, based on available scientific evidence, that neurodevelopment delay most probably was preventable had expedited delivery by C/S [i.e. caesarean section], after detecting CPD [i.e. cephalopelvic

disproportion] been done, instead of the incorrect management of Ms N[...]s labour by injudicious use of oxytocin, despite clear contraindications and without proper FHR [i.e. foetal heart rate] monitoring. Proper and timeous management of the prolonged active phase of the first stage could almost surely have prevented the acute and profound brain injury.'

- [27] The experts acknowledged, admittedly, that there were many other causes for neurodevelopment delay; an HII was one of many possibilities. The findings of other experts, such as radiologists and paediatricians, were required. Neither party, in this regard, called either of the obstetricians and gynaecologists to clarify their statement.
- [28] It is helpful, at this stage, to reiterate the principles enunciated in *Bee v Road Accident Fund*, [20] where the Supreme Court of Appeal dealt with the effect of an agreement recorded in a joint minute. In that regard, *Rogers AJA* held as follows:

'... The appellant's counsel referred us to the judgment of Sutherland J in *Thomas v BD Sarens (Pty) Ltd*

[2012] ZAGPJHC 161. The learned judge said that where certain facts are agreed between the parties in civil litigation, the court is bound by such agreement, even if it is sceptical about those facts (para 9). 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).'[21]

- [29] The court in the present matter is bound by the facts agreed upon by the radiologists, and the obstetricians and gynaecologists, respectively. Similarly, the court must accept the experts' shared opinions. Their qualifications, skills, and experience in the field of medicine placed them in a substantially better position than the court to draw inferences and conclusions from the available facts. A court can only reject such opinion evidence in exceptional circumstances, where (for example) there is a clear and material error in either the factual matrix or the reasoning based thereon.
- [30] Considering the extent of the facts and opinions agreed upon by the experts, the onus borne by the plaintiff was considerably lessened by the time of trial. The defendant, conversely, had a more difficult case to defend. At the core of the dispute were the divergent opinions of the paediatricians.

Dr Kara's evidence

- [31] Dr Kara testified that he had interviewed the plaintiff for the purposes of his report, and she had informed him that this had been her second pregnancy; there had been no problems. There was no family history of epilepsy or mental illness, and she had not taken herbal medicines or drugs. Dr Kara examined M, who presented with mild cerebral palsy. This was a non-progressive lesion of the brain that occurred during early development, within the first two years of life. It manifested itself in a child's delayed achievement of motor milestones, an impairment of his or her gait, some impairment of fine motor coordination, and epilepsy. The symptoms matched M's condition. Turning to the maternity case records, Dr Kara stated that

the absence of reflexes and crying at birth were indicative of neurological compromise. He went on to say that the records mentioned

that M had suffered convulsions during the first day of life.

- [32] Regarding causation, Dr Kara pointed out that the MRI scan revealed an HII, which was the cause of the child's cerebral palsy. The question to be answered was when this occurred. The radiologists agreed that it could not have been prior to 36 weeks of gestation. There had been no antenatal risks, and there was no evidence of an intrauterine infection. Dr Kara stated that the administration of oxytocin caused uterine hyperstimulation which, in turn, resulted in excessively long contractions that reduced the blood supply to the foetus and introduced a high risk of hypoxic ischaemia. There was significant encephalopathy at birth, with two convulsions during the first two days of life. The plaintiff and M were transferred to the Nelson Mandela Academic Hospital, which recorded birth asphyxia and HIE. There was no sign of a congenital brain or chromosomal abnormality; there was no sign of any metabolic disease. Dr Kara went on to testify that there were clinical signs that were consistent with HIE: a delay in fine motor development; a speech impairment; poor oral motor functioning; and epilepsy. He said that there was sufficient evidence to indicate the

probability that intrapartum asphyxia was the cause of M's cerebral palsy. The records indicated sub-standard foetal monitoring, the augmentation of labour, neurological depression at birth with the need for resuscitation, neonatal encephalopathy, as well as the finding of an HII on the MRI scan; these all helped to link the brain injury to the period immediately before birth. The critical or sentinel event was the augmentation of labour. The brain injury was not unforeseeable.

[33] Addressing Dr Keshave's findings, Dr Kara did not agree that M had ASD. He said that M had suffered a lesion to the brain, resulting in HIE; it was obvious that he had cerebral palsy. He conceded that the child had signs that were in keeping with ASD, but they were the consequence of an HII to his brain. This had been the primary event. The radiologists had confirmed this, too. A genetic study requested by Dr Keshave indicated that no genetic defect had been present. Dr Kara reiterated that a sudden and catastrophic injury occurred between 19h00 and 20h30 on 3 July 2012, when the nursing staff

administered an inappropriate dose of oxytocin while failing to implement proper foetal monitoring. This, he said, was consistent,

too, with the findings of the obstetricians and gynaecologists.

Dr Keshave's evidence

[34] Dr Keshave testified that spasticity and dystonia were clinical manifestations of the type of cerebral palsy associated with an HII. More specifically, an acute profound or BGT type of injury presented with dystonic cerebral palsy; in other words, a child would display random and involuntary movements, affecting the limbs and head. The child would have alternating tone, i.e. he or she would be stiff, then floppy. He distinguished this from a watershed type of injury, resulting in spastic cerebral palsy. Dr Keshave further stated that the radiologists' report to the effect that there had been an acute profound or BGT injury did not correspond with the child's clinical presentation. From the history narrated by the plaintiff, M sat when he was one year old and walked at the age of two years. He could run, albeit with an irregular gait; he had fine motor skills and could use his hands to feed himself, reach for and manipulate objects, and hold a pen. None of the features mentioned was typical for a BGT injury. Dr Keshave went on to say that M's language delay, with selective mutism, as well as echolalia,[22] was common for a child with ASD. So, too, was M's preference for playing by himself, poor eye contact, and aggressive tendencies

towards himself and others. M also presented with a sensory processing disorder; he was a picky eater, was hypersensitive to anyone touching his ears or head, was fearful of haircuts, and had a high pain threshold. Dr Keshave mentioned other features, too, confirming that the child's predominant neurological problem was an underlying ASD.

- [35] Regarding M's MRI scan, Dr Keshave testified, with reference to academic literature, that the abnormal results could also be associated with a child having ASD. These included cerebral profusion abnormalities, which could have placed M at an increased risk of suffering an HII. Dr Keshave stated that, from his physical examination of M, he noticed several dysmorphic features such as small, wide-spaced eyes and asymmetrical, low-set ears, but everything else appeared to have been normal. His weight and height were age-appropriate; his head size was normal; his muscular-skeletal system was normal. M displayed abnormal movements, as well as features of a hyperactive behavioural disorder. These were not, however, the primary neurological conditions observed in children with cerebral palsy caused by intrapartum birth asphyxia; they were more in keeping with a child having ASD. Dr Keshave admitted that M presented with some spasticity on his left side, but this was extremely subtle. For a BGT injury, both sides of the brain would have been affected. The child would have displayed marked dystonia, alternating muscle tone, and markedly decreased power in the limbs; this was not the case with M.
- [36] Dealing with the differences between his and Dr Kara's views, Dr Keshave reiterated that M had ASD, not cerebral palsy. This was because of how he presented clinically. Dr Keshave had recommended genetic testing as studies had shown that an underlying genetic condition could relate to ASD. An absence of anything in this regard did not mean, however, that the child did not have ASD; it could simply mean that the gene had not yet been found. Regarding M's seizures, Dr Keshave said that the occurrence of these within the first 24 hours would have been consistent with HIE; after such period, they would have been more in keeping with arterial or stroke-type injuries; with M, the records indicated that he had suffered seizures only on 5 July 2012. Furthermore, the MRI scan could not be used to time when the HII happened. A BGT injury could occur from 34 weeks until three months, even five years, afterwards.
- [37] The cause of an underlying ASD was unknown, said Dr Keshave. In the present matter, M did not present with typical features of an HII. He was neither spastic nor dystonic; he presented, however, with features of ASD. There was a mismatch between the MRI findings and how M presented clinically.

Key differences and concessions

- [38] Dr Kara was adamant that M had cerebral palsy. The features revealed by the MRI scan, the circumstances at the time of the plaintiff's labour, and how the child had presented at birth led him to believe that the cause of M's condition was a peripartum HII. Dr Keshave's views, however, were very different. He was adamant that M had an underlying ASD and not cerebral palsy. The child's clinical presentation was not what would have been expected from the nature and timing of the injury described by the remaining experts.

[39] In *Michael and Another v Linksfield Park Clinic (Pty) Ltd and Another*,^[23] the Supreme Court of Appeal addressed the determination of issues that required the opinions of expert witnesses. It emphasized that the exercise did not involve considerations of credibility but rather the examination of the opinions and an analysis of the experts' essential reasoning, before the court reached its own conclusion on the issues raised.^[24] The court went on to hold that:

'A defendant can properly be held liable, despite the support of a body of professional opinion sanctioning the conduct in issue, if that body of opinion is not capable of withstanding logical analysis and is therefore not reasonable. However, it will very seldom be right to conclude that views genuinely held by a competent expert are unreasonable. The assessment of medical risks and benefits is a matter of clinical judgment which the court would not normally be able to make without expert evidence and it would be wrong to decide a case by simple preference where there are conflicting views on either side, both capable of logical support. Only where expert opinion cannot be logically supported at all will it fail to provide "the benchmark by reference to which the defendant's conduct falls to be assessed".^[25]

[40] In *Louwrens v Oldwage*,^[26] the Supreme Court of Appeal confirmed the correctness of the approach taken in *Linksfield*. In considering how the trial court dealt with competing sets of evidence regarding the diagnosis given to a patient and the treatment

that followed, the appeal court was critical of the former's uncritical acceptance of an expert's opinion, pointing out that:

'... What was required of the trial judge was to determine to what extent the opinions advanced by the experts were founded on logical reasoning and how the competing sets of evidence stood in relation to one another, viewed in the light of the probabilities.'^[27]

[41] Under cross-examination, Dr Kara confirmed that the child had sustained a basal ganglia and thalamus (BGT) injury. This had affected the so-called primitive part of the brain, responsible for basic life functions such as breathing and temperature control. The injury, in this case, was mild to moderate, with M's having noticeable difficulty in using his mouth (oral motor dyskinesia), which affected his speech and swallowing functions. This was not, in the presence of an HII, a symptom associated with ASD. Dr Kara was adamant that the child had cerebral palsy; M had suffered a lesion to the brain during the development period, manifesting in motor and other disabilities. The MRI scan had clearly revealed the lesion. Dr Kara admitted that some of M's features matched those of a child with ASD, but that was a secondary issue; an HII during labour

was the primary issue. The key event had been the augmentation of labour by the administration of oxytocin. This had been done without an assessment of the mother and without CTG monitoring, either before or after induction to detect signs of any possible foetal compromise. Dr Kara conceded that there was no record of the foetal condition prior to 19h00. If it had been poor, then the brain injury could have occurred earlier. It was, however, most likely that the injury had occurred between 19h00 and the time of delivery because the records, as inadequate as they

were, suggested a normal foetal heart rate. An hour and a half later, the child presented with clear signs of injury, after the augmentation of labour.

[42] The child had presented with moderate neonatal encephalopathy at birth, meaning that there had been a 25% to 50% chance of a long-term adverse outcome. The fact that the records suggested that M had suffered convulsions only during the first 36, rather than 24, hours of birth did not matter; there had been signs of early neonatal encephalopathy. Dr Kara went on to say that there were any number of possible causes of ASD, including a brain injury. The main cause, however, was unknown.

[43] For his part, Dr Keshave accepted during cross-examination that it was possible for a child with cerebral palsy to have ASD. In the case of a BGT injury, severe (not mild) dystonia would be expected. He went on to acknowledge, with reference to the Volpe criteria,[28] that there were differences in the patterns of outcome for mild versus moderate to severe BGT injuries, with an increasing likelihood

of observing the clinical features of cerebral palsy when the injury was more severe. He said, however, that the main feature of a BGT injury was dystonia, which could manifest in different degrees. Dr Keshave stated that, when he examined M, he noticed his irregular gait but attributed this to a sensory processing disorder as part of his underlying ASD; an unsteady balance was typical for autistic children. He conceded that this could also be associated with a BGT injury. Regarding how M held a utensil with a clenched fist, Dr Keshave said that this could just be a preference; what was important was that he could use the utensil to feed himself. The same factors were pertinent to M's pen grip.

[44] The MRI scan, said Dr Keshave, revealed a moderate to severe BGT injury. It would, therefore, have been expected to result in a child with severe dystonic cerebral palsy, but that was not the case in the present matter. Instead, M presented with the features of ASD. Dr Keshave was adamant that an HII during the perinatal period did not make sense; he said that he had seen children who developed an HII at two or three years of age from a near-drowning or a severe and sustained epileptic seizure. There were studies that suggested that an HII could occur up to five years after birth. Dr Keshave stated that M's clinical features were unexpected in relation to the MRI findings and he could not say that an HII had resulted in the child's ASD. The causes of autism were unknown. There could, in the present case, be other factors that played a role.

[45] Importantly, there were several key concessions or admissions that Dr Keshave made while under cross-examination. The following exchange occurred:

'MS DA SILVA: . . . the child has suffered a hypoxic incident as a result of which he sustained severe brain damage. That is the starting point. The fact that he presents— I am not saying he presents with ASD— but the fact that he presents with ASD or CP is the secondary part of things. The first part of things, let us agree, was that there was a hypoxic ischaemic incident during labour?

DR KESHAVE: So, I cannot. . . [inaudible. . . intervenes]

MS DA SILVA: Intrapartum, in other words.

DR KESHAVE: So, in my opinion, M'Lord, I cannot say that, because as I have mentioned it does not tie up for me. It does not make sense. That hypoxic injury in the perinatal period does not clinically match what I have, regardless of the brain injury. So, for me, I cannot say this is only from the perinatal period, because I have seen children who are two, three years of age, who have developed a hypoxic ischaemic injury either from a near-drowning or a severe. . . epileptic. . . [inaudible]; they were fitting for so long, they developed similar patterns of injury. So, to say that it is only from one period, I cannot say. And also, when you present with autistic spectrum disorder, the cause is unknown. Nobody knows what causes autism. So, from that perspective, I cannot say that the brain injury resulted in this child's autism or CP or whatever it is.

MS DA SILVA: No, Doctor, we are not there yet. We are at the stage where the court has to determine: [was] the injury, as highlighted by the radiologists, caused, firstly, intrapartum; and, secondly, as a result of negligence on the part of the hospital. That is what we must first determine before we get to ASD or CP. . . Was there a brain injury that occurred intrapartum? That is where I am at.

DR KESHAVE: So, in my opinion, I am not disputing that there is a hypoxic injury on the MRI scan of the radiologists, and I cannot dispute the obstetrician saying that the intrapartum period could be a cause for the hypoxic injury. I am not disputing any of that. I am just saying that we need to, from a paediatric neurologist's perspective, look at it. . . there could be other factors that may be playing a role in this child.'

[46] Dr Keshave appears to have expressed scepticism about the accuracy of the findings and opinions of the radiologists as well as the obstetricians and gynaecologists. They did not correlate with his own findings. However, the cross-examination proceeded further as follows:

'MS DA SILVA: Now, Doctor, regard being had to. . . the only evidence that we have, and the joint minutes. . . this happened intrapartum, based on the evidence that exists, arising from the maternity case records. I put it to you that, on a balance of probabilities, the probable cause for the brain injury occurred intrapartum. . .

DR KESHAVE: Could, yes.'

[47] This was not an insignificant concession. Dr Keshave seems to have admitted, finally, that it was indeed possible that M's brain injury could have taken place during labour, notwithstanding his findings on the child's clinical presentation. The cross-examination continued:

‘MS DA SILVA: . . . I put it to you that the clinical outcome at present is secondary to the hypoxic ischaemic injury which happened intrapartum.

DR KESHAVE: . . . So, as a paediatric neurologist, I would say that the current clinical presentation does not meet everything that happened before that. I agree to everything that the obstetricians and radiologists

agreed to, but the current clinical presentation does not fit that.’

[48] If anything, then Dr Keshave expressed a scientist’s puzzlement, even frustration, at having been unable to explain with absolute certainty the inconsistencies between his findings and the opinions of the remaining experts. The court, however, is not required to decide the matter with 100% scientific certainty. The test remains whether the plaintiff has on a balance of probabilities discharged the onus of proof.

[49] Dr Keshave made further concessions. The exchange, below, is pertinent:

‘MS DA SILVA: . . . You will agree with me that the MRI report says nothing about the brain injury being consistent with ASD. All it says is that it is a hypoxic ischaemic injury?’

DR KESHAVE: . . . The radiologists’ report just describes the pattern of injury that is seen on the brain. They did not comment on the clinical presentation because they did not see the child.

MS DA SILVA: But you will agree that, whether you call it CP or ASD, hypoxia was the causative feature of the. . . child’s injury?

DR KESHAVE: Yes, there are features of a hypoxic ischaemic injury. . . I cannot dispute that.

MS DA SILVA: . . . You also cannot dispute that the sub-standard care during labour is what caused this hypoxic ischaemic brain injury?

DR KESHAVE: Yes, based on the obstetrician’s comments; that is what they said.’

[50] Later, Dr Keshave conceded that a child with cerebral palsy could present with a normal-sized head, as was the situation with M, and would not necessarily have cardiovascular or respiratory problems that could have placed him or her at risk of an HII. He admitted that there were grades of dyskinesia with different manifestations; in the case of mild to moderate BGT injury, the dyskinesia could affect just the limbs. Furthermore, Dr Keshave agreed that there were grades of cerebral palsy. A child with a mild condition could walk, albeit with an irregular gait; manage basic care; and present with only a mild

cognitive impairment.

[51] Dr Keshave remained adamant, however, that M presented with features of an underlying ASD. The child displayed language delay; he presented with selective mutism and echolalia. These features were not associated with cerebral palsy. Volpe, asserted Dr Keshave, had never suggested the presence of ASD consequent to an HII. During the neonatal period, M’s seizures did not occur during the first 24 hours, which would otherwise have indicated HIE;

seizures after such period were less likely to be associated with an HII. Regarding the plaintiff's testimony that she had overheard nurses commenting, on 4 July 2012, that the child had suffered seizures, Dr Keshave pointed out that if that had been so then there would have been moderate to severe HIE. M's clinical presentation contradicted this. Overall, for a BGT injury that occurred between 19h00 and 20h30, as agreed by the remaining experts, it would have been expected that the child should have presented with spastic quadriplegic cerebral palsy, accompanied by dystonia. That was not the case in the present matter. M presented predominantly with the features of an underlying ASD.

- [52] Dr Keshave agreed with Dr Kara that a child with mild cerebral palsy could walk, feed themselves, talk, and be aware of his or her environment. He pointed out, however, that Dr Kara had not dealt with tone, which was important for determining the type of cerebral palsy involved. His failure to have mentioned spasticity or dystonia in his report was a source of concern for Dr Keshave. The nature of the child's gross and fine motor skills, his feeding, and his speech could all be explained in terms of an underlying ASD, not cerebral palsy.

Assessment

- [53] It cannot be said that either Dr Kara or Dr Keshave was a poor witness. Each of their opinions was based on a sound premise and reached through a process of logical reasoning. There was little that separated the two.
- [54] The competing opinions must, however, be viewed in the light of the probabilities. The difficulty that faces the defendant in this regard is that the joint reports of the radiologists, as well as the obstetricians and gynaecologists, were never repudiated. Despite Dr Keshave's discomfort about the mismatch between the findings expressed in the joint reports and M's clinical presentation, the discrepancies were never tested at trial. Absent a clear and timeous repudiation of either or both the joint minutes, the parties were bound by the facts and opinions agreed upon. There was, moreover, no obvious error that would otherwise have obliged the court to have treated the joint minutes differently.
- [55] The radiologists agreed that M suffered a peripartum HII to his brain. It was a BGT-type injury and most probably resulted from hypoxia during the labour process. The obstetricians and gynaecologists agreed, inter alia, that there was sub-standard care on the part of the medical staff. At birth, M's condition was compatible with intrauterine hypoxia; he developed moderate to severe neonatal encephalopathy within 24 hours of delivery. They also agreed, notably, that it was very likely that the sub-acute hypoxic process started after the administration of oxytocin at approximately 19h00 and became acute before delivery at 20h30. The staff's sub-standard care was probably related to causation.
- [56] Dr Kara's views were consistent, in the main, with those of the remaining experts. The MRI scan results, the history of the plaintiff's labour, and how M presented at birth constituted the combination of factors that underpinned Dr Kara's opinion that the cause of M's condition was a peripartum HII. Dr Keshave's insistence that M presented with ASD, not

cerebral palsy, with the implication that the cause of the child's condition was not necessarily a peripartum HII, was the chief difference between the experts. However, the latter's ultimate acceptance of the views held by the remaining experts, his concession that the probable cause of M's brain injury was intrapartum and

that features of an HII were present, as well as his admission that he could not dispute that sub-standard care on the part of the medical staff had been the cause of the brain injury were sufficient to persuade the court that the former expert's opinion was to be preferred. Dr Keshave's acceptance, moreover, that there could be overlapping features of ASD and cerebral palsy appears to permit the possibility that M presents with varying degrees of both.

[57] The facts are similar to those in *NN obo ZN v MEC for Health, Eastern Cape Province*, [29] where the court assessed the competing opinions of the paediatricians involved. In that regard, after viewing the opinions in the light of the probabilities, the court preferred the testimony of the expert that found support from the remaining experts, and which was founded on logical reasoning. [30] The same approach must be adopted in the present matter.

Relief and order

[58] The plaintiff alleged that the negligence of the medical staff at Isilimela Hospital caused M to suffer a hypoxic incident that resulted in severe brain damage and spastic quadriplegic cerebral palsy. Overall, the court is satisfied that the cumulative effect of the experts' opinions is to demonstrate, on a balance of probabilities, that the negligence of the staff was the *conditio sine qua non* for M's brain injury. The child would have been delivered in a normal condition, without harm, but for the staff's inadequate monitoring, lack of clinical knowledge and skills, and non-compliance with the maternity guidelines. The failure to have facilitated delivery by caesarean section, consequent to the detection of cephalopelvic disproportion, together with the injudicious use of oxytocin without proper monitoring, were the critical elements in the chain of causation that led to M's brain injury.

[59] The vexing question of whether M suffered from cerebral palsy or presented with ASD was a matter that dominated trial proceedings. This was, of course, accompanied by the risk that sight was lost of the primary issue, viz. whether the plaintiff could prove that the negligence of the medical staff was the cause of the harm suffered. If the court accepted Dr Keshave's opinion that M presented with ASD, not cerebral palsy, then it could well be said that the plaintiff had failed to discharge the onus. Something else could have been the cause of M's condition, undermining the probability that it had been a peripartum HII. Considering, however, the compelling findings made by the remaining experts and expressed in

the unrepudiated joint minutes, the consistency of Dr Kara's opinion with such findings, and the concessions made by Dr Keshave,

it cannot be concluded that M presented with ASD. It is likely that, at best for the defendant, the cerebral palsy from which he suffers also displays features of ASD. This does not, however, detract in any way from the finding on the issue of causation.

[60] Consequently, the plaintiff's claim in her representative capacity must succeed. No evidence was led in relation to her claim in her personal capacity. Although counsel for the plaintiff referred to the court's decision in *Nkamela obo Nkuhle Nkamela v MEC for Health, Eastern Cape Province*,^[31] regarding the treatment of the plaintiff's personal claim in similar circumstances, the court is subsequently more inclined to follow the reasoning adopted in *NK v MEC for Health, Eastern Cape Province*,^[32] mentioned by counsel for the defendant. In the present matter, no factual matrix has been developed to support the plaintiff's claim in her personal capacity.

[61] What remains to be determined is the quantum of damages to be awarded. But that will be for another day. Regarding costs, there is no reason why the general rule should not apply; the plaintiff is entitled to recover her expenses.

[62] In the circumstances, the following order is made:

- (a) The defendant is liable for such of the plaintiff's damages, in her representative capacity, as the plaintiff shall prove or as might be agreed upon between the parties, arising from the negligent management of the plaintiff's labour and delivery of her son, M[...], on 3 July 2012 at Isilimela Hospital.
- (b) The defendant shall pay the plaintiff's taxed or agreed party and party costs incurred to date on Scale C, such costs to include the qualifying fees, charges or expenses, if any, of the following experts:
 - (i) Dr. Kara;
 - (i) Dr Wright; and
 - (i) Dr MacDonald.
- (c) The plaintiff shall further allow the defendant 14 calendar days to make payment of the taxed costs after the date of the Taxing Master's allocatur, whereafter the defendant shall pay interest on the costs at the legal rate then prevailing to date of payment.

JGA LAING

JUDGE OF THE HIGH COURT

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Ref: 68/20 – P9 (Mr Maqambayi)

Dates heard:

23 - 25 July 2024.

Date delivered:

29 July 2025.

- [1] Peripartum is understood to mean occurring in or being the period preceding or following childbirth. See <https://www.merriam-webster.com/medical/peripartum#:~:text=1%20of%202-,adjective,infected%20with%20the%20virus%20peripartum>, accessed on 24 July 2025.
- [2] The term is related to hypoxic ischaemic encephalopathy (HIE), which is understood to mean brain damage in a newborn infant as a result of the brain receiving inadequate oxygen. E Martin and J Law Oxford Concise Medical Dictionary 10 ed (2020) 380.
- [3] CTG monitoring is the electronic monitoring of the foetal heart rate and the frequency of uterine contractions. Martin and Law op cit 122.

- [4] Tachysystole is understood to mean excessively frequent uterine contractions during pregnancy. See https://en.wikipedia.org/wiki/Uterine_tachysystole#:~:text=Uterine%20Tachysystole%20is%20a%20condition,over%20a%2030%2Dminute%20period, accessed on 24 July 2025.
- [5] An Apgar score is defined as a method of rapidly assessing the general state of a baby immediately after birth, entailing the allocation of a score of 0, 1, or 2 points for each sign, usually measured at one minute and five minutes after delivery. Martin and Law op cit 48.
- [6] See n 2 above.
- [7] Oxytocin is a hormone that causes contraction of the uterus during labour and stimulates milk flow from the breasts. Intravenous infusions or injections thereof are used to induce labour. Martin and Law op cit 560.
- [8] Cephalopelvic disproportion is the state in which the diameter of the foetal head is greater than the pelvic outlet, preventing successful vaginal delivery. Martin and Law op cit 133.
- [9] 2013 (2) SA 144 (CC).
- [10] At paragraph [38].
- [11] At paragraphs [40] and [41].
- [12] 2015 (4) SA 574 (SCA).
- [13] At paragraph [30].
- [14] 2016 (3) SA 528 (CC).
- [15] At paragraphs [63] and [65].
- [16]
- [2015] 2 All SA 403 (SCA).
- [17] *Gentiruco 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] Pricewaterhousecoopers, at paragraph [97].
- [20] 2018 (4) SA 366 (SCA).
- [21] At paragraph [64].

- [22] Echolalia is the pathological repetition of the words spoken by another person. Martin and Law op cit 243.
- [23] 2001 (3) SA 1188 (SCA).
- [24] At paragraph [34].
- [25] At paragraph [39].
- [26] 2006 (2) SA 161 (SCA).
- [27] At paragraph [27].
- [28] The criteria were developed by the physician, Dr Joseph Volpe, of the Harvard Medical School, Boston, MA, USA. His research is well-known and frequently mentioned in matters involving medical negligence. The parties in the present matter referred to his work, Paediatric Neurology 18 ed (2018).
- [29] (CA 470/2017) [2020] ZAECBHC 14 (17 June 2020).
- [30] At paragraphs [25] to [28].
- [31] (308/2018) [2022] ZAECBHC 15 (31 May 2022).
- [32] (502/2017) [2023] ZAECBHC 24 (15 August 2023).

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