IN THE HIGH COURT OF SOUTH AFRICA

NORTH WEST DIVISION, MAHIKENG

Not reportable

Case No: 2044/2013

In the matter between:

K[...] E[...] M[...]
obo O[...] M[...] Plaintiff

and

THE MEMBER OF THE EXECUTIVE COUNCIL FOR HEALTH, NORTH WEST PROVINCIAL GOVERNMENT Defendant

Coram: Petersen ADJP

Heard: 18–19 August 2025; 26–28 January 2026

Judgment reserved: 18 February 2026

Delivered: This judgment was handed down electronically, circulated to the parties' representatives via email, uploaded to CaseLines, and released to SAFLII. The date and time for the handing down of the judgment are deemed to be 14h00 on 13 May 2026.

Summary: Medical negligence — delict — liability of provincial health authority for intrapartum hypoxic-ischaemic brain injury — prolonged rupture of membranes at term — materially inadequate foetal surveillance during active labour — no continuous foetal monitoring — MRI demonstrating watershed hypoxic-ischaemic injury consistent with prolonged evolving intrapartum

hypoxia — alternative explanations not established as more probable — negligence and causation proved on a balance of probabilities.

JUDGMENT

PETERSEN ADJP:

Introduction

[1] This is an action for damages instituted by the plaintiff in her representative capacity as mother and natural guardian of her minor daughter, O[...] M[...] ("O[...]"), who was born on 28 December 2006 at Schweizer-Reneke Hospital. The plaintiff alleges that O[...] sustained a severe hypoxic-ischaemic brain injury during the intrapartum period as a consequence of negligent obstetric and neonatal management by the defendant's medical and nursing staff, resulting in cerebral palsy and associated sequelae.

[2] At the commencement of the trial, and by agreement between the parties, an order was granted in terms of Rule 33(4) separating the issue of liability from the determination of quantum. This Court is accordingly required to determine only whether the defendant is liable for the damages alleged.

[3] The claim arises from events occurring between 27 and 28 December 2006, when the plaintiff presented at Schweizer-Reneke Hospital with prolonged rupture of membranes of approximately 48 hours' duration. The plaintiff's case is, in essence, that the defendant's employees failed adequately to monitor the progress of labour and foetal wellbeing, administered labour augmentation in circumstances of inadequate surveillance, and thereby failed to detect and respond to evolving foetal compromise, resulting in hypoxic-ischaemic brain injury.

[4] The trial was conducted over four hearing days in August 2025 and January 2026. The plaintiff adduced the expert evidence of Dr L Murray, an obstetrician and gynaecologist; Dr H Lewis, a paediatrician; and Dr D Pearce, a paediatric neurologist. The defendant adduced the evidence of Dr D Bowen, an obstetrician and gynaecologist; and Dr P Mteshana, a paediatric neurologist. The parties further placed before the Court, by agreement, the joint minute of the radiologists, Dr B Alheit for the plaintiff and Dr J Swartzberg for the defendant.

[5] Following the conclusion of the evidence, written heads of argument were filed at the direction of the Court. Both parties indicated that oral argument was unnecessary, and judgment was accordingly reserved.

The factual background

[6] The material factual chronology is largely common cause. The plaintiff, then a 23-year-old primigravida, attended antenatal care at Ipelegeng Clinic. No material antenatal complications were identified. On 27 December 2006 at approximately 12h30, the plaintiff presented at Schweizer-Reneke Hospital complaining of clear liquor drainage since approximately 25 December 2006. She reported no vaginal bleeding and no significant labour pains. Examination revealed a term pregnancy, cephalic presentation, no palpable contractions, and a recorded foetal heart rate of 134 beats per minute. Vaginal examination revealed a closed cervix with continued liquor drainage. Maternal observations were within normal limits.

[7] At approximately 15h51, a doctor examined the plaintiff. The history of prolonged rupture of membranes was confirmed. Ultrasound assessment suggested a term gestation. The diagnosis was prelabour rupture of membranes at term, and a decision was taken to induce labour. No criticism was directed at that decision, which all obstetric experts accepted as clinically appropriate.

[8] At approximately 18h00, misoprostol (Cytotec) was administered for induction. Subsequent labour progress was recorded at intervals. At approximately 20h00, mild contractions were noted, with a foetal heart rate recorded within normal limits and cervical dilatation of 2 cm. At approximately 22h30, moderate contractions were present, the cervix was 4 cm dilated, and a partogram was commenced.

[9] At approximately 00h30 on 28 December 2006, the cervix was recorded as 8 cm dilated. At approximately 02h30, full dilatation was recorded. The contemporaneous records described foetal observations at those recorded intervals as reassuring.

[10] At approximately 02h30, an intravenous infusion containing oxytocin was commenced to augment labour because the plaintiff was noted to have poor pushing effort. At approximately 02h45, the plaintiff removed the intravenous line. At approximately 02h55, she delivered a live female infant vaginally following episiotomy. The infant weighed 3.74 kg. The umbilical cord was recorded as wrapped twice around the neck. Thick meconium-stained liquor was noted after delivery of the infant. Recorded Apgar scores were 5/10 at one

minute and 8/10 at five minutes. The infant required tracheal suctioning, oxygen administration, medication, and monitoring.

[11] On 29 December 2006, the infant was transferred to Klerksdorp Hospital with respiratory distress. Complete records from that institution were unavailable.

[12] In subsequent years, O[...] manifested significant developmental and neurological impairment. In 2012, a developmental assessment raised the possibility of cerebral palsy secondary to hypoxic-ischaemic encephalopathy. Chromosomal investigations did not identify deletions or duplications, rendering a chromosomal explanation less likely.

[13] An MRI brain scan performed on 6 November 2013 revealed findings subsequently agreed by the radiologists to demonstrate a watershed hypoxic-ischaemic brain injury.

The expert evidence

The plaintiff's obstetric evidence: Dr Murray

[14] Dr Murray, an experienced obstetrician and gynaecologist, testified that the plaintiff's labour required enhanced vigilance because prolonged rupture of membranes of approximately 48 hours materially elevated obstetric risk. She referred to the applicable Guidelines for Maternity Care in South Africa (2000), which required materially closer foetal surveillance during active labour and enhanced monitoring in higher-risk circumstances.

[15] Her evidence was that no CTG monitoring was performed at any stage and that foetal surveillance during active labour fell materially below acceptable obstetric standards. She emphasised that the significance of monitoring lies not in record-keeping as an end in itself, but in the clinical opportunity it creates to detect evolving foetal compromise and intervene appropriately.

[16] Dr Murray further testified that oxytocin is a recognised clinical intervention, but that augmentation of labour in circumstances of inadequate foetal surveillance requires particular caution because increased uterine activity may exacerbate foetal compromise where such compromise exists. Importantly, Dr Murray explained that the MRI pattern of watershed hypoxic-ischaemic injury is consistent with a prolonged evolving hypoxic insult rather than an instantaneous catastrophic event. In her opinion, such a pattern is consistent with progressive foetal compromise developing during labour.

The plaintiff's paediatric evidence: Dr Lewis

[17] Dr Lewis addressed the neonatal condition and early postnatal management. He expressed reservations regarding the internal consistency of

the recorded Apgar scores and criticised aspects of neonatal management. More materially for present purposes, Dr Lewis distinguished between the mere presence of meconium and clinically significant meconium aspiration syndrome causing profound hypoxic injury. His evidence was that the available objective evidence did not establish meconium aspiration syndrome as the probable explanation for O[...]'s later neurological condition.

The plaintiff's paediatric neurology evidence: Dr Pearce

[18] Dr Pearce examined O[...] several years later and recorded findings consistent with significant permanent neurological impairment. She accepted the MRI findings as demonstrating hypoxic-ischaemic injury and concluded that the most probable timing of the insult was intrapartum.

The defendant's obstetric evidence: Dr Bowen

[19] Dr Bowen accepted that induction of labour was clinically appropriate, that prolonged rupture of membranes materially elevated obstetric risk, and that foetal monitoring during active labour was less frequent than contemplated by accepted obstetric practice. He nevertheless relied on intermittently recorded reassuring foetal observations, emphasised the modest oxytocin dose and limited duration of administration, and explored alternative explanations for the child's condition.

[20] Dr Bowen testified that the dose of oxytocin administered was modest and that, because the plaintiff removed the intravenous line after a short interval, he considered it unlikely that the infusion, viewed in isolation, materially altered the foetal condition. His evidence in this regard was directed principally at the question of causation rather than the standard of care.

[21] Dr Bowen further explored the possibility that meconium aspiration syndrome may have contributed to the infant's later neurological condition. He accepted, however, that the diagnosis and clinical implications of meconium aspiration syndrome fall more appropriately within paediatric expertise than obstetric expertise.

[22] Under cross-examination, Dr Bowen accepted that foetal monitoring during the active phase of labour was materially inadequate and that intermittent surveillance necessarily creates the risk that clinically significant deterioration may occur between recorded observations without detection.

The defendant's paediatric neurology evidence: Dr Mteshana

[23] Dr Mteshana examined O[...] on 30 July 2025. Certain aspects of her clinical findings differed from those of Dr Pearce, particularly in relation to

motor manifestations. Dr Mteshana considered alternative neurodevelopmental explanations for aspects of O[...]'s presentation, including developmental and behavioural features not necessarily specific to hypoxic-ischaemic injury.

[24] However, Dr Mteshana accepted that the MRI findings demonstrated hypoxic-ischaemic injury of a watershed pattern and that such injury is consistent with a prolonged evolving hypoxic process rather than an acute catastrophic event. She further accepted that, on the available evidence, intrapartum hypoxia remained a probable explanation for the neurological injury reflected on imaging and manifested clinically.

[25] While certain alternative developmental possibilities were canvassed, no definitive alternative diagnosis was established sufficient to displace the objective radiological findings.

The radiologists' joint minute

[26] The parties placed before the Court the joint minute of Dr Alheit and Dr Swartzberg. The radiologists agreed that the MRI demonstrated a watershed hypoxic-ischaemic injury pattern. They further agreed that the imaging did not support infective or inflammatory pathology as the sole explanation for the injury, and that recognisable genetic disorders with characteristic imaging signatures were unlikely to account for the findings.

[27] The radiological evidence was therefore materially common cause and provides an important objective evidential anchor in the causation enquiry.

The issues

[28] The principal issues for determination are whether the defendant's employees were negligent in the management of the plaintiff's labour and delivery, and if so whether such negligence probably caused the hypoxic-ischaemic injury sustained by O[...].

The applicable legal principles

Negligence

[29] The test for negligence remains that articulated in *Kruger v Coetzee*[1], namely whether a reasonable person in the position of the defendant would foresee the reasonable possibility of harm and take reasonable steps to guard against it, and whether such steps were not taken.

[1] *Kruger v Coetzee* 1966 (2) SA 428 (A).

[30] In the context of medical negligence, the standard is one of reasonable professional competence rather than perfection. As recognised in *Mitchell v Dixon*[2], a medical practitioner is required to exercise reasonable skill and care.

[31] This Court must evaluate expert evidence critically and not defer to professional opinion merely because it is sincerely held. In *Michael and Another v Linksfield Park Clinic (Pty) Ltd and Another*[3], the Supreme Court of Appeal emphasised that expert opinion must be logically defensible, grounded in fact, and capable of withstanding analytical scrutiny.

Causation

[32] Factual causation is ordinarily determined by applying the but-for test articulated in *International Shipping Co (Pty) Ltd v Bentley*[4], namely whether the harm probably would have been avoided absent the negligent conduct. The plaintiff bears the legal onus throughout to establish causation on a balance of probabilities. Absolute scientific certainty is not required.

[33] In *Minister of Safety and Security v Van Duivenboden*[5], the Supreme Court of Appeal emphasised that causation in civil matters is determined on a practical and probabilistic basis, informed by common sense and the evidence.

[34] The Constitutional Court in *Oppelt v Department of Health, Western Cape*[6] reaffirmed that the causation enquiry in medical negligence matters requires a practical assessment of what probably would have occurred had reasonable medical intervention been provided.

Evaluation of expert evidence

[35] Before addressing the disputed issues, it is appropriate to identify the matters that emerged as common cause or substantially uncontested. The plaintiff presented with prolonged rupture of membranes of approximately 48 hours' duration. This elevated obstetric risk and justified induction of labour. It is uncontested that the MRI demonstrated a watershed hypoxic-ischaemic injury pattern consistent with a prolonged evolving hypoxic process rather than an acute sentinel catastrophic event. No expert credibly suggested placental abruption, uterine rupture, cord prolapse, or comparable acute intrapartum catastrophe as the explanation for the injury.

[36] It further emerged, both from the documentary records and the expert evidence, that foetal monitoring during active labour was materially less

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[2] *Mitchell v Dixon* 1914 AD 519.
[3] *Michael and Another v Linksfield Park Clinic (Pty) Ltd and Another* 2001 (3) SA 1188 (SCA).
[4] *International Shipping Co (Pty) Ltd v Bentley* 1990 (1) SA 680 (A).
[5] *Minister of Safety and Security v Van Duivenboden* 2002 (6) SA 431 (SCA).
[6] *Oppelt v Department of Health, Western Cape* 2016 (1) SA 325 (CC).

frequent than contemplated by accepted obstetric standards. It is common cause that no CTG monitoring was performed at any stage.

[37] Dr Bowen suggested that CTG equipment may not have been available at Schweizer-Reneke Hospital at the relevant time. That proposition remained speculative. No factual evidence was adduced from hospital personnel concerning equipment availability, staffing constraints, operational limitations, or other institutional impediments that may have affected monitoring capability. In any event, even if CTG monitoring were unavailable, that would not absolve the defendant if reasonable foetal surveillance by other means was not provided.

[38] The defendant relied in part on recorded notations suggesting reassuring foetal status, including references to apparently normal foetal heart observations. Those entries are contemporaneous and therefore important. However, they represent isolated time-point assessments only. Their evidential significance must be evaluated in that context. Sparse intermittent reassuring observations do not establish continuous foetal wellbeing throughout the entire active phase of labour, particularly where the central issue is whether evolving deterioration occurred between those observations.

[39] Particular reliance was placed on the notation suggesting "good variability". Dr Murray explained that beat-to-beat variability is most reliably assessed through objective continuous monitoring. Dr Bowen accepted limitations in drawing firm conclusions from such entries absent formal CTG evidence. The notation cannot be equated with reliable continuous foetal assessment sufficient to exclude evolving compromise. At best, it records a contemporaneous clinical impression at a particular moment.

[40] The administration of oxytocin at approximately 02h30 is common cause. Oxytocin is a recognised obstetric intervention and its use is not inherently negligent. Its significance depends on context. Dr Murray's evidence, which I accept, was that augmentation of labour in circumstances of inadequate foetal surveillance requires particular caution because increased uterine activity may exacerbate foetal compromise if compromise is already developing. Dr Bowen's evidence that the dose was modest and administration brief is relevant to causation, but does not negate the broader significance of administering augmentation in circumstances of inadequate surveillance.

[41] Dr Lewis criticised the internal consistency of the recorded Apgar scores. While his reasoning in that regard was logical, caution is required in attributing determinative forensic significance to retrospective reconstruction of neonatal scoring. Apgar scores are clinical tools subject to observer variability. It is unnecessary to resolve the precise reliability of the recorded scores, as the causation analysis does not depend materially on that issue.

[42] The meconium aspiration theory requires careful scrutiny. The presence of meconium is not disputed. However, the mere presence of meconium does not establish meconium aspiration syndrome as the probable cause of profound neurological injury. Dr Lewis drew a clear distinction between aspiration of meconium and clinically significant meconium aspiration syndrome causing severe hypoxic injury. No objective paediatric respiratory evidence was produced establishing the latter as the probable explanation for the injury demonstrated on MRI.

[43] Dr Bowen's exploration of meconium aspiration as a causative mechanism must be viewed in the context of his expertise. He appropriately accepted that the diagnosis and clinical implications of meconium aspiration syndrome fall more naturally within paediatric rather than obstetric expertise. More importantly, the radiological evidence reflects a prolonged evolving watershed injury pattern, which is materially inconsistent with a purely post-delivery respiratory event as the most probable explanation.

[44] The alternative developmental or neurobehavioural explanations canvassed through Dr Mteshana similarly do not displace the plaintiff's case. While certain developmental features were identified, no definitive alternative diagnosis was established sufficient to explain away the objective hypoxic-ischaemic radiological findings. Dr Mteshana ultimately accepted that intrapartum hypoxia remained a probable explanation.

[45] Evaluating the expert evidence in accordance with *Linksfield*, Dr Murray's evidence was careful, clinically coherent, and logically grounded in the objective evidence. Her reasoning concerning the significance of inadequate monitoring and the implications of the MRI findings was persuasive.

[46] Dr Lewis's evidence was helpful, particularly in relation to the limits of the meconium aspiration theory, though I do not regard retrospective precision concerning Apgar scoring as materially determinative.

[47] Dr Pearce's neurological conclusions aligned with the objective radiological evidence and were not materially undermined.

[48] Dr Bowen was appropriately candid in important respects, particularly his acceptance that foetal monitoring during active labour was materially inadequate. His evidence on the meconium theory, however, remained tentative and insufficiently supported by objective evidence.

[49] Dr Mteshana's evidence narrowed rather than materially expanded the dispute, given her acceptance of the objective hypoxic-ischaemic radiological findings and the probability of intrapartum hypoxia.

Negligence

[50] The question is whether the conduct of the defendant's employees fell below the standard reasonably to be expected of competent practitioners in the prevailing circumstances. Those circumstances included a term primigravida undergoing induced labour following prolonged rupture of membranes of approximately 48 hours. This was not an uncomplicated low-risk labour. A reasonable practitioner would foresee the possibility that inadequate foetal surveillance in such circumstances could permit evolving foetal compromise to go undetected, with potentially grave neurological consequences.

[51] The evidence establishes that foetal monitoring during active labour was materially below accepted standards. This conclusion does not depend solely upon proof that CTG monitoring ought specifically to have been deployed. Even absent CTG availability, materially closer surveillance by reasonable means was required. That did not occur.

[52] The defendant's own obstetric expert accepted that the monitoring was materially inadequate. The records reflect only intermittent observations over a prolonged active labour period. The foreseeable risk created by such inadequate surveillance is precisely that clinically significant deterioration may occur between observations without detection.

[53] The administration of oxytocin in circumstances of inadequate foetal surveillance reinforced the substandard nature of the intrapartum management. While it is unnecessary at this stage to attribute independent causal significance to the brief infusion, its administration formed part of the broader pattern of inadequate clinical management.

[54] Applying the principles in *Kruger v Coetzee*, I am satisfied that the defendant's employees failed to take reasonable steps that competent practitioners would have taken in the circumstances to guard against foreseeable foetal harm.

[55] Negligence is accordingly established.

Causation

[56] Negligence alone does not determine the matter. The plaintiff bears the onus of establishing, on a balance of probabilities, that the negligent conduct found was a factual cause of the injury sustained by O[...]. The enquiry is whether, had reasonable intrapartum care been provided, the injury probably would have been avoided.

[57] The starting point in that enquiry is the objective radiological evidence. The agreed MRI findings demonstrate a watershed hypoxic-ischaemic injury. That finding is of central significance because it materially informs both mechanism and timing. A watershed injury is consistent with a prolonged

evolving hypoxic process rather than an instantaneous catastrophic insult. That proposition was materially common cause.

[58] The plaintiff was not required to establish the exact minute at which irreversible injury commenced. Civil causation does not demand scientific precision of that order. What must be shown is probability. The evidence establishes that the injury pattern is materially consistent with progressive hypoxic compromise developing during the labour process.

[59] The plaintiff's case is not that inadequate monitoring itself caused neurological injury. Monitoring is diagnostically significant because it creates the opportunity to detect evolving foetal compromise and intervene before injury becomes irreversible. The relevant enquiry is therefore sequential, whether proper monitoring would probably have detected evolving compromise, whether timely clinical response would probably have followed, and whether such intervention would probably have prevented the injury.

[60] Dr Murray testified that progressive hypoxic compromise of the kind reflected in watershed injury would ordinarily manifest through evolving foetal heart abnormalities capable of detection through reasonable foetal surveillance. Her evidence in this regard was logically coherent, clinically grounded, and not materially displaced. Dr Bowen accepted that inadequate monitoring creates the risk that clinically significant deterioration may occur undetected between observations.

[61] The defendant relied on intermittently reassuring recorded foetal observations. Those entries do not defeat the plaintiff's case. The purpose of regular monitoring is precisely because foetal condition may evolve between observations. Sparse isolated reassuring entries do not establish sustained foetal wellbeing throughout a prolonged active labour period, particularly where the objective radiological evidence demonstrates that a prolonged evolving hypoxic process did in fact occur.

[62] Detection alone would not suffice unless timely intervention would probably have followed. The evidence supports that inference. If foetal compromise had been detected in a monitored obstetric setting, ordinary clinical responses would probably have included cessation of augmentation, intrauterine resuscitative measures, escalation of clinical concern, and expedited delivery if deterioration persisted. Dr Murray's evidence supports that conclusion. There is no evidential basis to conclude that appropriate intervention, had foetal compromise been detected, would probably have been unavailable.

[63] The nature of the injury remains significant. A prolonged evolving hypoxic insult inherently presents a window during which detection and intervention may prevent irreversible injury. This case is materially distinguishable from one

involving an acute catastrophic event producing injury instantaneously. The MRI findings materially support the conclusion that the injury evolved over time.

[64] The alternative explanations advanced by the defendant do not materially displace the plaintiff's causation case. Meconium aspiration syndrome was not established as the probable cause of the injury. The developmental or neurobehavioural alternatives canvassed similarly lacked sufficient evidential foundation to displace the objective hypoxic-ischaemic explanation.

[65] The oxytocin administration forms part of the broader clinical context, but I do not consider it necessary to make a discrete finding that the brief infusion independently caused material deterioration. The causation finding does not depend on such a conclusion.

[66] Considering the totality of the evidence, including the agreed MRI findings, the prolonged evolving nature of the injury, the materially inadequate foetal surveillance, the probability that evolving compromise would have manifested detectably, and the probability that timely clinical response would have followed if deterioration were detected, I am satisfied that the plaintiff has established factual causation on a balance of probabilities.

[67] Legal causation presents no independent difficulty. Permanent hypoxic neurological injury is plainly the kind of harm reasonably foreseeable from negligent failure adequately to monitor foetal wellbeing during a high-risk labour.

[68] I accordingly find that both factual and legal causation have been established.

Conclusion

[69] The plaintiff has proved, on a balance of probabilities, that the defendant's employees were negligent in their management of her labour and delivery, and that such negligence probably caused O[...] to sustain the hypoxic-ischaemic brain injury reflected on MRI and manifested clinically thereafter.

Costs

[70] Costs ordinarily follow the result, and no persuasive reason exists to depart from that principle. The plaintiff has succeeded on the separated issue of liability in a matter involving complex expert medical evidence across multiple disciplines. The employment of counsel and expert witnesses was plainly reasonable and necessary for the proper adjudication of the issues.

Order

[71] In the premises, the following order is made:

1. The defendant is declared liable for payment of 100% (one hundred percent) of the plaintiff's proven or agreed damages, in her representative capacity on behalf of O[...] M[...], arising from the negligent management of the plaintiff's labour and delivery at Schweizer-Reneke Hospital on 27 and 28 December 2006, resulting in hypoxic-ischaemic brain injury and its sequelae.

2. The defendant shall pay the plaintiff's costs relating to the separated issue of liability, such costs to include:

   2.1 the costs of counsel;

   2.2 the reasonable qualifying, preparation, reservation (where applicable), attendance and testimony fees of the plaintiff's expert witnesses, insofar as such costs relate to the issue of liability;

   2.3 the reasonable costs associated with consultations, joint meetings and preparation of joint minutes by the plaintiff's expert witnesses relevant to the issue of liability;

   2.4 the reasonable costs of obtaining medico-legal reports relevant to the determination of liability;

   2.5 the reasonable costs associated with the MRI investigation and radiological reporting relied upon for purposes of the liability determination;

   2.6 the reasonable travelling and accommodation costs necessarily incurred by the plaintiff and the minor child in attending medico-legal consultations and examinations relevant to the issue of liability, subject to taxation where not agreed.

3. In the event that the costs are not agreed:

   3.1 the plaintiff shall be entitled to have such costs taxed;

   3.2 the defendant shall pay the taxed costs within 30 (thirty) calendar days of taxation or agreement;

   3.3 failing such payment, interest shall accrue on the taxed or agreed costs at the prescribed legal rate from the 31st day after taxation or agreement to date of payment.

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A H PETERSEN
ACTING DEPUTY JUDGE PRESIDENT OF THE HIGH COURT
NORTH WEST DIVISION, MAHIKENG

Appearances:

For the Plaintiff:	Adv M Coetzer
Instructed by:	Wim Krynauw Attorneys
	c/o Nienaber & Wissing Attorneys, Mafikeng
For the Defendant:	Adv T Kwape
Instructed by:	The State Attorney, Mafikeng