IN THE HIGH COURT OF SOUTH AFRICA

KWAZULU-NATAL DIVISION, PIETERMARITZBURG

CASE NO: 8700/2013

13/3/2019

In the matter between:

A D Plaintiff

(and on behalf of K L O)

and

MEC FOR HEALTH FOR THE PROVINCE OF KWAZULU-NATAL Defendant

ORDER

(a) The defendant is directed to compensate the plaintiff, in her representative capacity as mother and as natural guardian of K, a boy born on the 14th of May 2009, and in her personal capacity, for the damages claimed in this action, either as proved or agreed;

(b) The defendant is directed to pay the plaintiff's taxed or agreed party and party costs on the High Court scale, such costs to include:

(i) the costs of the plaintiff's attorney attending upon any consultations with witnesses in preparation for trial, including the consultations with the under-mentioned expert witnesses;

(ii) the costs of senior and junior counsel where employed, including the reasonable and necessary costs of their preparation for trial, and for their attendance upon consultations with the under-mentioned expert witnesses and the plaintiff;

(iii) the qualifying fees of the under-mentioned expert witnesses, including the costs of the preparation of their reports, their attendance fees, and the fees to qualify themselves to testify at the trial and for any necessary consultations with the plaintiff's attorney and counsel (with the quantum of fees, and expenses, to be determined by the taxing master), namely:

(aa) Dr Y Kara, the specialist paediatrician;

(bb) Dr D McLynn, the obstetrician and gynaecologist;

(cc) Prof J Lotz, the professor of radiology; and

(c) The proceedings are otherwise adjourned sine die.

JUDGMENT

Koen J

Introduction

[1] This is an action in which the plaintiff, in her personal capacity and in her representative capacity as guardian of her minor child, K L O ('K'), a boy born on 14 of May 2009, claims damages from the defendant. The damages are alleged to arise as a consequence of the defendant's medical personnel's negligence in treating the plaintiff and her unborn foetus whilst she was an in-patient at the Wentworth Hospital ('the hospital') prior to, during and/or immediately after K's birth. The negligence is alleged to have resulted in K suffering birth asphyxia, resulting in hypoxic ischemic encephalopathy ('HIE') with consequent brain damage.

[2] At the commencement of the trial the issue of liability, including causation, was separated for determination before all other issues. This judgment deals only with those separated issues.

Background facts

[3] The following background facts are common cause:

(a) At all material times the defendant administered and operated the hospital. In 2009, the hospital contained a midwife run obstetric unit which functioned as a community health centre. It was not staffed with an obstetrician who could carry out caesarean section deliveries. It was only able to deal with low risk births. High risk labours and patients who required caesarean section deliveries would generally be referred to the King Edward VIII Hospital ("the King Edward Hospital").

(b) The medical and nursing personnel at the hospital at all material times acted within the course and scope of their employment with the defendant;

(c) The plaintiff was admitted to the hospital, pursuant to the agreement[1] pleaded by her, at approximately 02h00 on 14 May 2009 for the purposes of giving birth to her first child. She was examined by the defendant's medical personnel and admitted to the labour ward;

(d) The defendant's medical personnel were under a legal duty to ensure that the rendering of medical care, treatment and advice to the plaintiff and her unborn child, would be done with the skill, care and diligence that can reasonably be expected of medical practitioners and/or nursing staff in similar circumstances;

(e) K was born at the hospital at 19h10 on 14 May 2009.

The pleadings

[4] The plaintiff alleges that the defendant's medical personnel were negligent in treating her and K, resulting in his condition, in one or more of the following respects:

(a) they failed to diagnose bloodstained liquor observed after the plaintiff's membranes had ruptured, as being abnormal and failed to report that fact to a doctor on duty;

(b) they failed to observe and record the foetal heart rate with a cardio-tachograph ('CTG') machine half hourly, or at all, after 16h00;

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[1] In order to succeed with a claim for damages based on medical negligence, a patient has to allege and prove the contract, negligence, and that such negligence caused damages – *Blyth v Van den Heever* 1980 (1) SA 191 (A).

(c) they failed to continuously monitor the foetal heart rate with a cardio tachograph ('CTG') after decelerations in the foetal heart rate were recorded;
(d) they failed to diagnose foetal compromise from the CTG readings, or otherwise;
(e) they failed to recognise timeously that the foetus was in distress and to arrange for a caesarean section delivery;
(f) they applied fundal pressure during the second stage of the plaintiff's labour when they ought reasonably to have known that it was dangerous to do so; and
(g) they accordingly failed to provide the plaintiff and K with appropriate medical treatment as would reasonably be required in the circumstances for their well-being;
(h) as a result of such negligence K was born severely mentally and physically handicapped, and has irreparable brain damage.

[5] The defendant in his plea denies these allegations of negligence on the part of the medical personnel. Initially the defendant contended that K was born in a healthy state, as an MRI scan performed on him at the King Edward Hospital subsequent to his birth yielded a normal result.[2] But the defendant pleaded over that even if K was not born in a healthy state, the medical personnel at the hospital had provided all reasonable and necessary medical care and treatment to the plaintiff and K. Specifically it is pleaded that:
(a) the bloodstained liquor observed, on its own, is not an abnormal event during the birthing process indicative only of a placental abruption, but is generally caused by the patient pushing against a not fully dilated cervix;
(b) the plaintiff did not have a retro placental clot subsequent to K's delivery and therefore, did not suffer from a placental abruption;
(c) the defendant's personnel had reasonably and appropriately observed and recorded K's foetal heart rate during the birthing process;
(d) it is not uncommon for decelerations in the foetal heart rate to occur during the birthing process;
(e) there were no clinical signs demonstrating that the foetus was in distress which required immediate invasive medical intervention;
(f) at no stage was fundal pressure applied;
(g) K was provided with emergency resuscitation upon his birth;

[2] This contention has subsequently been reviewed, as will appear below.

(h) the second stage of labour was not prolonged.

[6] The defendant pleaded in the alternative that in the event of it being found that K presents with cerebral palsy, such cerebral palsy was not caused by any negligence on the part of the defendant's employees, but was a direct and natural consequence of one or more of the following factors:
(a) the exposure by the plaintiff of K to the risks associated with the plaintiff being afflicted with syphilis;
(b) the plaintiff smoking cigarettes during the course of her pregnancy;
(c) that K suffered from a perinatal ischaemic stroke/neonatal stroke and not from any perinatal asphyxia.

[7] In further amplification of his denials, the defendant pleaded that:
(a) pethidine was administered to the plaintiff during the course of her labour, which would account for the lower Apgar scores which were recorded in respect of K shortly after his birth;
(b) K demonstrated no meconium stained liquor at birth;
(c) If it is found that K suffered from seizures, that these seizures occurred during a period which indicates that he suffered an insult ante-natally and prior to the plaintiff's admission to the hospital;
(d) K could not have suffered from a partial prolonged hypoxic ischaemic insult during the plaintiffs labour as there were no signs of foetal distress, her labour was not prolonged, and accordingly there was insufficient time for the foetus to have suffered such an insult to the brain during labour;
(e) K's symptoms failed to meet the criteria stated by the American Academy of Obstetrics and Gynaecology (ACOG) and therefore cannot be held to be as a result of intrapartum hypoxaemia;
(f) If it is found that K suffered from an acute profound hypoxic insult, it would have made no difference to the ultimate outcome and sequela suffered by him, as the midwives would have had insufficient time to transfer him to King Edward Hospital for a caesarean section to be performed;[3]

[3] Briefly stated, the argument in this regard is that the plaintiff was fully dilated at 18h00 and K was born at 19h10. Consequently, even if he suffered from an acute intrapartum hypoxic event during this period, a caesarean section delivery would not have been performed on the plaintiff to extract the minor child as she

(g) If it is found that K suffers from cerebral palsy, then it was caused by a perinatal stroke.

The witnesses

[8] In order to discharge the burden of proof upon her, the plaintiff testified and the following expert witnesses were called on her behalf:

(a) Prof Lotz, a professor in radiology attached to the University of Stellenbosch;

(b) Dr Kara, a specialist paediatrician who practices for his own account;

(c) Dr McLynn, a specialist gynaecologist and obstetrician in private practice.

[9] The defendant adduced the evidence of inter alia:

(a) Dr Moran, a specialist gynaecologist and obstetrician employed by the defendant;

(b) Dr Naidoo, a family physician employed by the defendant;

(c) Dr V Govender, a paediatric neurologist employed by the defendant,

(d) Dr R Singh, a neonatologist employed by the defendant;

(e) Ms MF Adams, a midwife employed by the defendant at the hospital, at the material time, and who attended to the plaintiff;

(f) Mr Neville Padayachee, an ambulance attendant employed by EMRS;

(g) Dr Reitz, a radiologist employed by the defendant.

Assessing negligence

[10] The question of reasonableness and negligence is for the court to decide on the basis of the evidence and various and often conflicting expert opinions presented. As a general rule, this determination does not primarily involve considerations of credibility, but the examination by the court of the opinions and essential reasoning of the experts before reaching its own conclusion on the issues raised. What is required in evaluating evidence is a determination of whether, and to what extent the opinions advanced are reasonably justified as being founded on logical reasoning.[4]

[11] However, the fact that the defendant's experts are all employed by the defendant and could therefore have some loyalty to or sympathy for their employer, cannot be ignored altogether either. Specifically in the case of Ms Adams, there is the additional factor that she was attending to the plaintiff during the crucial final stages of her

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was ready to deliver at the hospital.

[4] *Michael & another v Linksfield Park Clinic (Pty) Ltd & another* 2001 (3) SA 1188 (SCA); *Medi -Clinic Ltd v Vermeulen* 2015 (1) SA 241 (SCA).

pregnancy and labour, and that any culpable omissions or conduct on her part will reflect negatively on her professional proficiency. In this regard it was quite surprising that although Ms Adams testified some nine years after the event, she claimed to still have an independent recollection of what period a specific recordal of the foetal heart rate related to, although this was not recorded on the available hospital records, and notwithstanding that evidence prima facie being in conflict with other evidence. I shall return to this aspect below.

K's cerebral palsy

[12] Although initially disputed on the pleadings, it became common cause that K has cerebral palsy.[5] What is in dispute is the nature and cause thereof. Regarding the nature thereof, Dr Kara testified that K suffers from 'dyskinetic cerebral palsy with some spastic quadriparesis (a mixed type of cerebral palsy)', whereas Dr Govender is of the view that K has 'spastic hemiparetic cerebral palsy' with a gross motor classification of four (4), impaired speech and communication skills and poor hand function, the spasticity being particularly on the right side. The nature of the cerebral palsy is not just simply a matter of terminology and academic interest, but significant in considering the more important issue for the purpose of this judgment, namely the cause of K's cerebral palsy and specifically whether it can be attributed to any negligence on the part of the defendant's medical personnel in their treatment of the plaintiff prior to K's birth.

The possible causes of K's cerebral palsy

[13] Although the defendant specifically pleaded that K's cerebral palsy 'was a direct and natural consequence of one or more' of the following exposures of K, by the plaintiff, to the risks associated with her being afflicted with syphilis, the plaintiff smoking

[5] Per the joint minute of Dr Singh, the defendant's neonatologist, Dr Govender, the defendant's paediatric neurologist and Dr Kara, the plaintiff's paediatrician. The joint minute further recorded inter alia that K has severe cerebral palsy, which Dr Kara contended was dyskinetic and spastic, while Dr Govender indicated that it was spastic hemi-paretic with a gross motor functional classification of four (4), impaired speech and communication skills and poor hand function. K's vision and hearing appears to be reasonable and he has microcephaly. There are no dysmorphic or syndromic features. K was born at term and was appropriate for gestation. His head size was normal at birth. K had Apgar scores of three (3) and five (5), and he was resuscitated at birth and had convulsions soon after birth. Dr Kara noted that the minor child had a mixed type of cerebral palsy, while Dr Govender indicated that the minor child was predominantly hemiplegic with spasticity especially on the right side and his power on the right side was of a grade three (3) and he had contractures of the right elbow and wrist and used his left hand predominantly. Dr Govender contended that the minor child had bilateral short Achilles tendons. Dr Kara submitted that these findings by Dr Govender did not describe hemiplegia but rather described asymmetric spastic quadriplegia. Dr Kara noted the joint minute by Dr Reitz and Prof Lotz which commented on acute profound and partial prolonged hypoxic ischemic injury which is asymmetrical and involved with the left hemisphere more than the right one. Dr Govender disagreed.

cigarettes during the course of her pregnancy, and K suffering a stroke, Dr Moran and Dr Singh, both called by the defendant, conceded that the plaintiff's smoking[6] and the syphilis could be excluded as the cause of K's condition. They however persisted with the view that these factors might have made K more vulnerable to injury.[7]

[14] The only causes, amongst the possible causes for K's cerebral palsy suggested by the expert witnesses that remained were whether, as a matter of probability, K's condition was consequent upon him suffering brain damage as a result of being distressed at some stage or stages during the birthing process (as contended for by the plaintiff and which was alleged to be due to negligence on the part of the defendant's employees), or whether his condition is as a consequence of him having suffered a perinatal ischaemic stroke/neonatal stroke (as contended for by the defendant). I turn then to consider the probability of these respective causes accounting for K's condition and whether these resulted from any negligence on the part of the defendant's personnel.

Negligence and the standard of care

[15] In *Van Wyk v Lewis*[8] it was stated, with reference to *Mitchell v Dixon*, [9] '. . . that "a medical practitioner is not expected to bring to bear upon the case entrusted to him the highest possible degree of professional skill, but he is bound to employ reasonable skill and care." And in deciding what is reasonable the court will have regard to the general level of skill and diligence possessed and exercised at the time by the members of the same profession in the circumstances. . . . The ordinary medical practitioner should . . . exercise the same degree of skill and care, whether he carries on his work in the town or the country. . . . The fact that several incompetent or careless practitioners happen to settle at the same place cannot affect the standard of diligence and skill which local patients have a right to expect.'

[16] The standard of care which the defendant's employees were required to provide inter alia had to meet the defendant's Guidelines for Maternity Care.

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[6] Prior to the plaintiff discovering that she was pregnant, she had smoked approximately one (1) cigarette a day. She stopped after she went to her second visit to the antenatal clinic. The plaintiff had her second antenatal visit to the clinic on the 7th of October 2009.

[7] It was inter alia maintained that pregnancy is a hyper co-aguable state and the fact that the plaintiff smoked during her pregnancy further increased the risk of coagulation which could cause a stroke.

[8] *Van Wyk v Lewis* 1924 AD 438 at 444.

[9] *Mitchell v Dixon* 1914 AD at 525.

The defendant's 'Guidelines for Maternity Care in South Africa 2007'

[17] According to the defendant's '*Guidelines for Maternity Care in South Africa 2007*'[10] ('the guidelines'):

(a) 'labour' is diagnosed if there are persistent painful uterine contractions accompanied by at least one of the following:

(i) cervical effacement and dilatation;

(ii) rupture of the membranes; or

(iii) a show.

(b) the first stage of labour comprises:

(i) the latent phase, commencing when 'the woman is in labour and the cervix is less than 4 cm dilated or more than 1 cm long'; and

(ii) the active phase, commencing when 'the woman is in labour and the cervix is 4cm or more dilated and less than 1 cm long';[11]

(c) As regards the first stage of labour, the latent phase of labour is said to be prolonged when it 'exceeds 8 hours' and the active phase of labour is said to be prolonged if 'the cervix dilates at a rate of less than 1 cm/hour';[12]

(d) the second stage of labour commences when the 'cervix reaches full dilation (10 cm)';[13]

(e) the second stage of labour is prolonged if:

(i) the fetal [foetal] head has not descended into the pelvic floor within two hours of full dilation; or

(ii) delivery has not occurred after 45 minutes of pushing in a *nullipara*, or 30 minutes of pushing in a *multipara*;[14]

(f) the fetal [foetal] heart rate ought to be monitored:

(i) two hourly during the latent phase of labour; and

(ii) half hourly (before, during and after contractions) during the active phase of labour;

(g) a cardio tachograph (CTG) is used 'for high risk labour only'; [15]

(h) fetal [foetal] distress is suspected when the following signs are observed:

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[10] Available at : http://www.kznhealth.gov.za/family/Maternity_care_guidelines_2007.pdf, accessed 20 February 2019

[11] The guidelines at 34.

[12] The guidelines at 44.

[13] The guidelines at 40.

[14] The guidelines at 52.

[15] The guidelines at 51.

(i) a baseline fetal [foetal] heart rate of more than or equal to 160 beats per minute (bpm);
(ii) a baseline fetal [foetal] heart rate of less than 110 bpm;
(iii) variability persistently less than 5 bpm on the CTG, in the absence of sedating drugs;
(iv) late decelerations of the fetal [foetal] heart rate;[16]
(i) problems requiring referral to a hospital during labour, before delivery, include:
(i) suspected fetal [foetal] distress; and
(ii) poor prognosis in the latent phase of labour (more than or equal to 8 hours).[17]

The hospital records[18]

[18] Regrettably, the clinical and obstetrical records relating to the plaintiff's labour are incomplete in certain crucial and material respects, notably the CTG's reflecting the foetal heart rate during the plaintiff's labour, and specifically from 16h00/16h20 onwards, assuming those entries to be accurate. This has impacted on the adjudication of this matter. It is a disturbing fact that in more than one of these medical negligence cases that have come before this court, involving the current defendant, incomplete records are produced in respect of a crucial stage of the labour of plaintiffs. The only possibly inference to be drawn is either that no records were kept, or if there is some evidential basis for concluding this, that whatever records there might have been, have been lost or otherwise became unavailable.

[19] At the outset of the trial it was said that these records had been lost. That is however assuming they ever existed. Ms Adams was probably the only one who could give direct evidence on whether there were additional records kept, specifically monitoring the foetal heart rate as there should have been. She testified, but nine years after the event, of alleged ongoing monitoring of the plaintiff and the heart rate of her unborn foetus until 16h50. There was however no mention made of CTG monitoring thereafter. CTG printout strips should form part of a continuous record retained in the same record storage location as the other printouts, and would therefore be unlikely to be lost separately if they existed. No CTG printouts of the latter stages of the plaintiff's

[16] The guidelines at 55.
[17] The guidelines at 65.
[18] These are clinical and obstetrical records.

labour were produced. Although it was indicated that these records have been lost, it is probably more correct to conclude that they never existed.

[20] The defendant and his staff are under a duty to properly monitor the plaintiff's condition and keep records of their attendance to the plaintiff and her unborn child. They failed to do so. Dr Moran, called by the defendant, conceded that there ought to have been further monitoring after the last CTG recording produced in evidence. He also agreed that the lack of monitoring by the midwives amounted to substandard care and that foetal distress might have occurred at some time during that period. That constitutes negligence. Further, K's cerebral palsy was consistent with having resulted, as a matter of probability, from a HIE, unless another clear credible cause was demonstrated.

[21] The maternity records that were produced in court reveal that:

(a) when the plaintiff was admitted to the hospital at 02h00, she was recorded to be already two (2) cm dilated and experiencing 'contractions moderate x 2 palpable';[19]

(b) at 06h00 her cervix was three (3) cm dilated and one (1) cm long;

(c) the plaintiff's condition was reassessed by Ms Adams at 08h00. This assessment revealed that she was coping with labour pains, the foetal heart rate was between 142 to 151 bpm via the CTG, which was reactive, and her cervix was still three (3) cm dilated;

(d) the plaintiff's membranes apparently ruptured at 10h30 while she was in the shower. This was reported by the plaintiff to the nursing staff;

(e) the plaintiff's labour was assessed at 12h00 on the 14th of May 2009 by Ms Adams. This assessment revealed that she was coping with labour pains, that the foetal heart rate was between 129 to 134 bpm, and that her cervix was four (4) cm dilated.[20] It was however queried,[21] whether the plaintiff's membranes had ruptured at 10h30 whilst she was in the shower;

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[19] The plaintiff was admitted to the hospital on the 14th of May 2009 at 02h00 complaining of lower abdominal pain and a 'show'. On assessment at 02h00 on the 14th of May 2009, it was revealed that her BP was 134/79, she was 38/40 weeks pregnant via palpation and 40/40 via sonar, presented with a SFH of 37 cm, the presentation of the foetus was cephalic with a longitudinal lie and posterior, she experienced moderate X 2 contractions which were palpable, on PV examination her cervix admitted one (1) finger and was two (2) cm long posteriorly and her membranes were intact with the presenting part of the foetus being 4/5 above the pelvic brim. The plan formulated for her treatment was to carry out CTG monitoring which revealed a reactive foetal baseline heart rate of 120 bpm. She was to be monitored and assessed within four (4) hours at 06h00. The plaintiff was in labour and according to the guidelines the foetal heart rate should thereafter be monitored two hourly.

[20] According to the guidelines the foetal heart rate should thereafter be monitored half hourly.

[21] It is not clear on what basis, but presumably because the nursing staff had not observed it for themselves.

(f) the plaintiff's labour was again assessed at 14h00 and it was recorded that she was not coping with labour pains, the foetal heart rate was 136 bpm, her cervix was five (5) cm dilated with the presenting part being 3/5 above the pelvic brim. The plaintiff's liquor was blood stained;

(g) Pethidine 50 mg and Maxolon IMI were administered to the plaintiff at 14h25;

(h) she was once more assessed by Ms Adams at 16h00 which assessment revealed that she was coping with labour pains, but her liquor was blood stained. She was experiencing strong contractions. Her management was to continue with CTG monitoring, and monitoring of her vital signs and she was encouraged to mobilise;

(i) the plaintiff's cervix was found to be fully dilated at 18h00 when it was recorded that she was 10 cm dilated;[22]

(j) K was born at 19h10. He weighed 3.5 kg, had a length of 53 cm, and had an Apgar score of 3/10 at one (1) minute and 5/10 at five (5) minutes. He was provided with oxygen via nasal prongs and naloxone to reverse the effects of the pethidine administered to the plaintiff during her labour;

(k) K suffered a seizure within the first six (6) hours of birth.

[22] From the above records it emerges that the only record of foetal heart rate monitoring during the latent phase of labour was at:

(a) 02h00, when it was 120 bpm;

(b) 06h00, when it was 130 bpm;

(c) 08h00, when it was 142 - 151 bpm;

The only record of foetal heart rate monitoring during the active phase of labour was at:

(a) 12h00, when it was 129 - 134 bpm;

(b) 13h00, when it was 129 bpm;

(c) 14h00, when it was 136 bpm;

(d) 14h25 (determined by having reference to the time pethidine was administered, as recorded on the CTG trace);[23]

(e) 16h00, when it was recorded at 118 bpm, and a second recording (allegedly at 16h20) of 130 - 135 bpm.

Discussion

[22] The plaintiff had now entered the second stage of labour.

[23] The first CTG is labelled with the plaintiff's name and the date of 14th May 2009. The CTG's appeared to be a progressive series of records in time but the exact time when they were recorded is not documented

[23] The mere fact that the medical treatment administered was either unsuccessful or not as successful as it might have been, or that the treatment administered did not have the desired effect, does not on its own necessarily justify the inference of a lack of diligence, skill or care on the part of a health care practitioner.[24] The question is whether the care received was substandard. Dr Moran, as already indicted above, conceded that the care given to the plaintiff during the latter stages of her labour had been substandard.

[24] Dr Moran and Dr McLynn agreed that the plaintiff was post-date by one (1) week when she presented in labour at the hospital. Nothing material appears to turn on that fact alone. What additionally was known, or should have been known, to the nursing staff shortly after the plaintiff's admission at the hospital over and above her being post date by one week is however material, namely that she was a *primigravida*, smoked and had been treated for syphilis. Although these factors did not causing K's cerebral palsy, they individually and collectively were risk factors which, even on the evidence of the defendant's experts, would have made K more vulnerable to injury.

Cephalo-pelvic disproportion

[25] Dr McLynn raised the possibility of the plaintiff suffering from cephalo-pelvic disproportion ('CPD'), stressing that it is the disproportion of the size of a big baby, compared to the pelvic area of a mother with a smaller frame in relative terms, that is important, rather than the actual size of either the baby or the mother in isolation. He expressed the view that there was cephalo-pelvic disproportion.

[26] That view was not shared by Dr Moran. Indeed he was not challenged thereon during his cross-examination. If there was CPD, the total duration of the plaintiff's labour would probably not have been within the normal time limits as agreed in the joint minute executed between Dr Moran and Dr McLynn. But in any event, Dr McLynn's opinion was that the plaintiff's labour was prolonged by ten minutes in the second stage which would seem to be of little consequence as no caesarean section delivery could have been offered to her within those ten minutes. The plaintiff did have an episiotomy but this was described by Dr Moran, although he did not attend to her, as just a cutting of soft tissue at the vaginal entrance which would be done even if there was no CPD. In his opinion had there been CPD, the foetal head would have never descended into the

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on any of them, and therefore there is uncertainty as to what stage of labour the last CTG was done.

pelvis and there would not have been a need to do an episiotomy. Accordingly, the episiotomy demonstrated that the foetus successfully negotiated its way into the pelvis of the plaintiff, thus confirming that there was no CPD.

[27] In my view CPD can probably be excluded on the probabilities.

The progression of the plaintiff's labour and non-compliance with the guidelines

[28] The plaintiff's pregnancy should have from the time of her admission been viewed as a pregnancy with at least a moderate to higher risk. It becomes necessary then to consider whether the guidelines were observed.

[29] According to the maternity records, the latent first stage extended from her admission at 02h00, when the plaintiff was already experiencing contractions and was two (2) cm dilated, to the latest 12h00, when it was reflected that she was already four (4) cm dilated. The foetal heart rate should have been monitored two hourly during that time. That notwithstanding, there was no recordal of the foetal heart rate at 04h00 or at 10h00. The guidelines inter alia require that 'poor prognosis in the latent phase of labour (more than or equal to 8 hours)'[25] require referral to a hospital.

[30] The active phase extended from 12h00 until the plaintiff was fully dilated at 18h00, which is six hours. Monitoring during this period had to have taken place half hourly. There were no regular half hourly recordals of the foetal heart rate during the active stage.

[31] Based on the summary of labour:

(a) the plaintiff's first stage of labour had lasted more than eight (8) hours, and was therefore prima facie prolonged. There are no records of monitoring K's foetal heart rate after 16h20;

(b) The plaintiff's second stage of labour lasted one (1) hour and 10 minutes.

[32] The neonatal records indicated that K was admitted inter alia for severe birth asphyxia and respiratory distress, had an abnormal neurology with convulsions, and had raised cardiac enzymes (CK 1238). That recorded observation is consistent with prolonged labour and foetal distress. These are clinical impressions recorded by experienced attending staff, and are significant.

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[24] *Louwrens v Oldwage* 2006 (2) SA 161 (SCA).

[25] The guidelines at 65.

[33] In evidence the defendant however maintained, contrary to the impressions noted and recorded by his staff, that the labour was not prolonged and that there was no indication of foetal distress.

[34] Dr Moran testified that in his view the labour was not prolonged. Dr McLynn however expressed a contrary view. Prima facie, having regard to the guidelines, the labour was prolonged. That should have prompted considerable more vigilance and more regular monitoring, particularly in the light of the plaintiff's history of being *primigravida*, a smoker and having required treatment for syphilis. It would suggest a referral to a hospital with full obstetric facilities. The failure to have done so, in my view, constitutes sub-standard care and hence negligence. Such substandard care was compounded by the lack of regular monitoring and no monitoring (no records of monitoring being available) after 16h20.

[35] Considerations of prolonged labour apart, the monitoring to determine any possible foetal distress was not conducted in accordance with the guidelines and prima facie constitutes negligence. Proper monitoring was not done at the prescribed intervals. Given that the plaintiff's pregnancy should have alerted staff as being a risky one, and that monitoring was not done regularly in accordance with the guidelines, proper care required that even more care should have been devoted to proper monitoring.

[36] The defendant submitted that the entry by the nursing staff of their observations of K was erroneous, and that the CTG strips which reflect the monitored foetal heart rate until approximately 16h20, even if not at half hourly intervals, did not indicate foetal distress and hence not the possibility of hypoxic brain injury during the process of the plaintiff's labour.

[37] One does however not know what manifested itself after approximately 16h20.

[38] The proper monitoring of the foetal heart rate in correlation with the uterine contractions is vital, particularly during the active phase of labour in order to diagnose potential foetal distress. This was not disputed. Accurate monitoring is particularly relevant to identify and determine whether there are 'type)' decelerations, as explained by Dr McLynn and confirmed by Dr Moran under cross examination. The active phase of labour, as defined, was from 12h00 until the plaintiff was fully dilated allegedly at

18h00. The foetal heart rate was not monitored and recorded half hourly during this active phase as required by the guidelines.[26]

[39] In reviewing the available CTG recordings, Dr McLynn and Dr Moran, in their joint minute, however agreed that they were normal, suggesting a healthy foetus during labour. That agreement did not however extend to the last section of the active phase, which included variable decelerations which Dr McLynn found alarming. The obstetricians were in agreement that there was no monitoring for at least two (2) hours and 40 minutes, from 16h00 (and not from 16h20).

[40] There is no written evidence in the medical notes or on the partograph of any foetal monitoring after 16h20, that is for the last two (2) hours and some 50 minutes of labour. Accordingly, one does not know what the foetal condition was during the last two (2) hours and 50 minutes of labour (from 16h20 until 19h10 when K was born), particularly whether or not the foetus was stressed.

[41] Ms Adams disputed that time interval, suggesting that there was at worst no foetal heart rate monitoring from 16h50 until K's birth at 19h10. Her viva voce evidence was to the effect that she made the entry in the records at 16h20 but that it was in respect of monitoring of the foetal heart rate which had commenced at 16h20 when she recorded the time of the monitoring, but that the reading recorded against 16h20 related to the monitoring for half an hour from 16h20, taking the monitoring to approximately 16h50.

[42] This evidence came at the end of the trial and after a number of adjournments, and well after the joint minute of the obstetricians had been concluded recording that there was monitoring (albeit inadequate) only until 16h20. The defendant, having investigated the relevant factual background to this matter, would presumably not have agreed to the terms of the join minute covering the period until 16h20 only, if Ms Adams

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[26] The defendant's medical personnel did not follow the defendant's own guidelines, as the foetal heart rate was not monitored every half hour during the active phase of the first stage of labour. The experts agreed that this is necessary to assess the condition of the foetus and to ensure that there are no 'late decelerations'. It is common cause that the 'mechanism' of late decelerations can cause a hypoxic ischaemic injury to the foetus' brain. Dr Singh agreed that late decelerations could have caused hypoxic ischaemic injury to the brain. With proper monitoring of late decelerations, or any similar problems which could cause or contribute to a hypoxic ischaemic injury, hypoxic ischaemic injury could be determined timeously in advance before such injury occurs. The lack of proper monitoring during this later phase of K's birth indicates that the defendant's medical personnel did not monitor whether the foetus was in distress with the level of care the defendant himself requires of his staff, which accordingly resulted in them failing to diagnose that the foetus was distressed. This failure by the defendant's professional nursing staff to adhere to the general level of skill and diligence possessed and exercised in the same circumstances by members of the same profession constitutes negligence.

had always disclosed that she had conducted continuous monitoring for half an hour after 16h20, that is until 16h50.

[43] It also seems improbable that the foetal heart rate would have been recorded at 16h00 at the end of the half hour monitoring period, from presumably 15h30 to 16h00, but that the next entry relating to a monitoring period would be identified with reference to the start of the monitoring period, but the result be inserted at a later stage at the end of that monitoring period against the starting time of the monitoring period. That would of course mean that if the previous monitoring period had been completed at 16h00, that the next half an hour monitoring period could not have been completed at 16h20. But that potential complication would not arise, if the result recorded at 16h20, was of a shorter monitoring period.

[44] The timing intervals on the incomplete CTG tracings that are available, are unhelpful in identifying real time, except by a fairly unreliable reconstruction with reference to the time recorded when pethidine was administered to the plaintiff. The recording does not seem to accord with the timeline evidenced by the CTG tracings, if one accepts the administration of pethidine recorded thereon and accepts that it is a continuous recording divided into ten (10) minute segments.[27]

[45] It is highly improbable that Ms Adams, after the lapse of time from the birth of K until she testified, would still have an independent recollection (seemingly in conflict with the practice relating to the recordal of monitoring results up to then, and prima facie in conflict with what was agreed between the obstetricians), that what was recorded as the foetal heart rate at 16h20 was in fact the heart rate covering the 30 minutes post 16h20. Her evidence in this regard must be viewed with suspicion, and as possibly representing what she believed might have happened based on a retrospective reconstruction, or alternatively, having regard to the fact that it is her conduct which is under scrutiny, representing a biased, probably untruthful and improbable account to try and distance herself from any culpability or criticism. This must inevitably casts doubt also on the reliability of the heart rate recorded by her.

[46] I agree with the submission by the plaintiff that this ‘second recording’ of the foetal heart rate must be viewed with suspicion.

Should the plaintiff have been transferred earlier?

[27] Exhibit "C" at 19-21, if one calculates the ten (10) minute time segments from 14h30, the tracings end at

[47] Instead the defendant has sought, with reference to the CTG's that are available, to suggest that the foetal heart rate recorded indicated a healthy foetus. However the recordings were not done in accordance with the guidelines and are open to conflicting interpretation.

[48] With regard to the last recording, Dr McLynn cautioned that it revealed three decelerations that were greater than 15 bpm, which ought to have been viewed with 'suspicion'.

[49] In that respect, Dr Moran testified that the foetal heart rate was normal throughout the labour until 16h00 when it was 118 bpm, which was probably because the plaintiff had been administered pethidine which would have depressed the foetal heart rate. He further pointed out that when the foetal heart rate was rechecked thereafter, it went to normal limits and was reactive and had improved to 130 - 135 bpm at 16h30. Accordingly, in his view the foetus was healthy during the period of the last variable decelerations as there were also accelerations.

[50] Dr Naidoo stated that for a CTG to be suspicious and non-reassuring relating to variable decelerations, the decelerations must drop to less than 60 bpm and must occur for at least 90 minutes and must occur in at least 50 per cent of contractions. Dr Naidoo concluded, looking at the available trace containing the variable decelerations, that one can see they did not occur for 90 minutes and in 50 per cent of the contractions. Dr Naidoo accordingly opined that the CTG with variable decelerations is not a non-reassuring trace and not suspicious.

[51] That might be so having regard to the available CTG, but these were still decelerations which should have been monitored continuously to determine whether there was cause for concern, particularly in the light of the plaintiff's history. There were clearly indicators which required further careful and continuous monitoring, rather than one simply concluding that because there are no records after that time, that the start of decelerations which were noticed were innocuous.

[52] The incomplete CTG records must also not be viewed in isolation. Regard must also be had to whether any risk indicating factors had developed during the labour process.

[53] Dr McLynn referred to the following factors which would have persuaded him to have opted for a caesarean section delivery [28] earlier:

(a) Blood stained liquor in the first stage of labour;

(b) An abnormal CTG;

(c) Post maturity;[29]

(d) A positive VDRL and signs of borderline CPD; and

(e) A high head.

None of these in isolation might have been sufficient to require a caesarean section delivery, but they had to be viewed in totality. It appears to me that his opinion in this regard is reasonable and that the evidence does not detract from his opinion.

[54] Ms Adams, who had monitored the plaintiff's labour and recorded that the plaintiff's liquor was blood stained, stated, again apparently on pure mental recollection, that the plaintiff was not bleeding, the implication being that this did not represent an obstetric emergency.

[55] I view this evidence, as I did with other aspects of her evidence relating to the monitoring of the foetal heart rate at the critical stage, with suspicion. It seems highly improbable that she, being employed as a midwife in busy hospitals, could have a detailed independent recollection of the peculiar facts of this matter, having dealt with numerous births since that of the plaintiff over the last eight plus years.

[56] Dr Moran, in seeming support of Ms Adams' evidence, testified that the tissues in the vagina are fragile and bleed easily, and therefore that during the process of examination, it is not uncommon to cause a little bit of contact bleeding. He was however not present, does not have personal knowledge of the plaintiff's condition, and expressed a general view. He drew attention to the fact that Ms Adams had recorded that the liquor was blood stained and not that the plaintiff was 'bleeding'.

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[28] The defendant has raised the fact that caesarean section deliveries themselves carry risks, including deaths due to bleeding during and after the delivery, surgical complications in terms of damage to the bladder and other tissues, anaesthetic complications, and longer term complications as many women with previous caesarean section deliveries are at a higher risk for future pregnancies. Dr Moran expressed the view that there were no indications which warranted the plaintiff's exposure and that of the foetus with which she was pregnant, to the inherent risks associated with a caesarean section delivery. Dr Naidoo also testified that the National Committee For Confidential Enquiries Into Maternal Deaths released in 2011 to 2013 had found that caesarean section deliveries were associated with a 2.8 per cent increase in the chance of maternal death compared to normal vaginal deliveries. The risk of a caesarean section does not arise in this case, although it is not an insignificant matter. If the need for a caesarean was indicated, then the plaintiff should have at least been given that option after a full explanation of the risks. The fact of the matter is that she was not given that option. The issue is whether the need therefore was indicated.

[29] The plaintiff being post-mature does not feature as a material aspect of her case and correctly so, and stands to be discounted. She was post-date only by approximately one (1) week as confirmed in the joint minute executed between Dr McLynn and Dr Moran. Dr McLynn conceded that K did not have any vernix cream left on his skin, nor did he have red, dry, peeling or wrinkled skin at birth and long nails, which are

[57] If what Ms Adam observed as blood in the liquor was simply a common consequence of conducting an examination, then she would hardly have considered it sufficiently significant to record it specifically. It should have been a factor of serious concern.

[58] The same sentiment applies in respect of Dr Naidoo's evidence that the blood stained liquor could be contact bleeding. The presence of blood in the liquor should have been treated as a cause for concern. More significantly, is the further opinion of Dr Naidoo that the blood stained liquor could be as a result of the plaintiff not coping well with labour pains, and she would have pushed against her undilated cervix which would have caused trauma and a bit of bleeding, even if not active bleeding (even if there was no evidence to suggest that the plaintiff was suffering an intrapartum haemorrhage which would constitute an obstetric emergency). It is correct that in retrospect that there might not be evidence of an intrapartum haemorrhage, but this would not necessarily have been known at the time, should not have been assumed to be absent, and should have given rise to heightened caution.

[59] At the very least, even if the blood stained liquor might not have suggested an immediate obstetric emergency requiring an immediate caesarean section delivery, it was an important indicator of risk at an early stage, as Dr McLynn rightly maintained.

[60] Dr McLynn expressed the view that it was inappropriate for the plaintiff's labour to be managed at the hospital and that there were signs which were misinterpreted by the defendant's employees which indicated that the plaintiff should have been transferred to a level two (2) facility which had an obstetrician in the theatre. These signs included the plaintiff's *primigravida* status, her smoking and the diagnosis of syphilis.

[61] That view was however contradicted by Dr Moran and Dr Naidoo who maintained that the plaintiff's *primigravida* status did not render her labour a high risk one which warranted referral to a secondary or tertiary hospital such as the King Edward Hospital. Dr Moran stated that if every *primigravida* mother is a high risk patient, every first pregnancy would be one and should be referred to a high level of care, which was inappropriate. That argument however elevates the plaintiff's *primigravida* status to being the only factor. It was only one factor, in the opinion of Dr McLynn of at least three factors, and they should be viewed holistically and cumulatively.

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all characteristics of post-maturity.

[62] Dr Moran further testified that although the fact that the plaintiff smoked was a known risk factor to the foetus, it was not a risk factor which warranted the plaintiff's referral to a level two (2) hospital for her labour. Nor in his view was the fact that the plaintiff was treated for syphilis via an effective form of treatment a risk factor which warranted her labour being referred to a high level of care. He pointed out that the positive test could indeed have been a false positive for syphilis.

[63] Again I am of the view that it is incorrect to consider the smoke and syphilis issues in isolation. They are factors which would make K more vulnerable to injury. It is reasonable to expect that they collectively contributed to an increased risk as Dr McLynn stated, which required increased monitoring and intervention.

[64] Dr Naidoo, the defendant's expert physician, also opined that the plaintiff's labour was not a high risk as she only smoked one (1) cigarette a day, which she had stopped. Further, she was treated for syphilis and her *primigravida* status did not render her pregnancy a high risk one, but a pregnancy routinely managed by a midwife run obstetric unit. In my view however, these factors did point to and required more vigilance in monitoring.

[65] The question that however remains is whether on a totality of all the evidence, it is established on a preponderance of probability that a HIE, as opposed to some other cause, for example a peri-natal stroke, is the cause of K's cerebral palsy, the defendant's aforesaid negligence notwithstanding. If a stroke presents as the probable cause of his condition then, the defendant argued, the plaintiff's action must fail.

Did K's cerebral palsy on a balance of probability arise from a hypoxic ischemic injury or a peri-natal stroke?

The radiological evidence

[66] Radiological evidence often provides an objective conclusive answer to this question. Prof Lotz confirmed that there was no doubt that K's brain suffered an infarct.

[67] Prof Lotz outlined the following during his evidence with reference to a slide presentation:

(a) although the brain consists of several parts, the basal ganglia, which he referred to as the 'reptilian brain', and the neocortex, which he referred to as the 'human brain', require to be considered in the context of K's condition;

(b) the reptilian brain is essential to 'sustain life' at the time of birth, whilst the human brain is 'essentially asleep' during this time, being only needed once the child develops;
(c) accordingly, if there are any problems at birth, resulting in a lack of oxygen supply to the reptilian brain, the body's 'auto regulatory system' will 'steal' blood and oxygen from those areas of the brain not essential at birth, in order to maintain life;
(d) this, he describes, often happens during the first stage of labour when the mother and foetus start developing some kind of distress and the oxygen supply 'is not what it should be'. He emphasised that this will take place over a period of hours when the 'salvage team' will set up a system to redirect blood away from those areas not needed, namely the human brain, to the reptilian brain;
(e) he describes two processes that can occur, one is described as a 'prolonged partial' process and the other is described as an 'acute profound' process. The prolonged partial process is when tacuteneocortex to the basal nuclei. This leaves a 'specific footprint' that can be identified. In this process one does not see an injury to the reptilian brain, because it has not been compromised as blood flow has been redirected to it. However, the human brain would be compromised by this process and one would then see an injury in the periphery. This is the 'hallmark of the prolonged partial' process. The auto regulatory system will initially endeavour to 'steal' blood from the 'watershed' areas of the brain. Consequently, if injury can be identified within the watershed areas of the brain it suggests a partial prolonged process;
(f) The acute profound injury occurs when there is 'an acute obstetrical emergency'. Here there is insufficient or no time for a redirection of blood as in the partial prolonged process. One can recognise the mechanism by looking at the pattern of injury. If the injury is outside the reptilian brain, it is a prolonged partial injury, and if inside, it is more likely an acute profound injury. In an acute profound injury, one would also expect to see injury to the 'motor strip', which is the portion of the brain which controls the essential functions immediately after birth, as this area is also already activated during the birthing process. The basal ganglia and the motor strip 'go together'. The motor strip is extremely sensitive to injury in an acute profound scenario for the simple reason that it is the only area in the neocortex that is active during birth.
(g) With reference to the MRI scan taken of K's brain on the 3rd of February 2016, Prof Lotz concluded that K had sustained an acute profound hypoxic ischaemic injury by virtue of an injury to the central part of the brain, as well as his motor strip being

'completely destroyed'. K also sustained a 'second infarct', being an injury which was 'slap bang within the watershed territory', which extended further along into the 'true arterial territory'. The injury was of a partial prolonged process by virtue of the 'mushroom shaped' gyrus in this area, but it was not a complete infarction of the gyrus but only a partial infarction. This he testified is indicative of the gyrus being 'undermined', as there was still blood 'coming through' and the 'injury pattern is a sort of excavation of the gyrus'. In his view, the MRI features 'are diagnostic of an acute profound and partial prolonged hypoxic ischaemic injury in the term brain at a chronic stage of evolution', which he referred to as a 'mixed pattern'. Under cross-examination he described this mixed pattern as 'classically' occurring when there is a long stage of labour, with the partial prolonged occurring initially and the acute profound occurring during the second stage of labour when 'there is a crisis on hand'.

(h) Under cross-examination, Prof Lotz stated that the MRI scan mitigated against a conclusion that K had suffered from an isolated arterial occlusion, or stroke, because:

(i) if there is such an arterial occlusion, the brain tissue dies *in toto* by virtue of there being no blood supply, whereas there are features of the injury being due to a partial prolonged hypoxic ischaemic injury by virtue of the presence of ulegyria;

(ii) the arteries supplying the motor strip are the anterior cerebral artery and the middle cerebral artery. Accordingly, for the motor strip to have been damaged, there would have had to have been two arterial infarctions. The injury in the motor strip is partly in the middle cerebral artery territory and partly in the anterior cerebral artery territory. There would therefore had to have been one in the middle cerebral artery territory and one in the anterior cerebral artery territory, which he believed was 'hugely unlikely'.

[68] Much of the radiological evidence is not in dispute between Prof Lotz and the radiologist called by the defendant, Dr Reitz. In a joint minute they agreed that:

(a) a generic disorder was unlikely to have caused K's brain damage;

(b) there were bilateral sonic abnormalities, bilateral subcortical and periventricular white matter abnormalities, and that these findings are compatible with a mixed pattern of hypoxic ischaemic injury to a term brain;

(c) the MRI findings of hypoxic ischaemic injury to the term brain indicate that a hypoxic-ischaemic injury occurred at some point after 36 weeks of gestation;

(d) additionally, there is asymmetrical focal atrophy of the left posterior parietal region , including the left sensorimotor cortex;

(e) asymmetrical damage has been described as part of the spectrum of prolonged partial hypoxic ischaemic injuries, although Dr Reitz maintains that it is relatively rare; and
(f) a final determination of the probable cause and probable timing of the injury would also involve a consideration of the clinical and obstetrical records.

[69] Dr Kara, the paediatrician called by the plaintiff, in a joint minute with Dr Singh, the defendant's neonatologist and Dr Govender, the defendant's paediatric neurologist, also expressed the view that the likely cause of the minor child's cerebral palsy was an intrapartum hypoxic ischemic injury based on one or more of the following:
(a) a prolonged second stage of labour;
(b) no record of foetal monitoring for three hours prior to delivery;
(c) caput and moulding;
(d) depression at birth with resuscitation, respiratory distress, and encephalopathy;
(e) an admission diagnosis of HIE;
(f) normal head size at birth, with no foetal growth restriction; and
(g) an MRI scan showing features of HIE.

[70] The defendant's reply to the individual indicators relied upon by Dr Kara (referred to in the preceding paragraph) in brief, is as follows:
(a) Dr Moran was of the view that the plaintiff's labour was not prolonged (he said the total duration was 11 hours and 30 minutes) but normal including the latent phase as well;
(b) The failure to monitor and/or to record the foetal heart rate during the two (2) hours 40 minutes prior to birth was conceded. However it was contended that this was not conclusive as the foetal heart rate was not alarming prior to that, and because the defendant believes that K's condition was caused by a peri-natal stroke and not HIE;
(c) Dr Moran testified that there was no caput and no moulding, which would certainly have been signs of a prolonged labour and foetal distress;
(d) As regards depression at birth with resuscitation, and respiratory distress, Dr Singh stated that neurological depression at birth may have been due to the maternal sedation with pethidine;

(e) A normal head size at birth with no foetal growth restriction would also not be inconsistent with a peri-natal stroke, as opposed to one earlier in the pregnancy. Dr Kara confirmed that if K suffered a stroke after 36 or 37 weeks, his brain is 80 to 90 per cent developed and therefore, one would not get microcephaly (a small head) at birth;
(f) The MRI scan showing features of HIE is not conclusive, as the MRI scan also has features consistent with a perinatal stroke.

[71] Dr Singh, a neonatologist, concluded that K's cerebral injury was due to a perinatal stroke, which being a vascular event left predominant lesion on the left. This it was said, is evidenced on the MRI which also did reveal ~~also~~ a left middle cerebral artery territory infarct.

[72] Dr Govender concluded K suffered a neonatal stroke based on the MRI findings which revealed this left middle cerebral artery territory infarct. Dr Govender suggested that K may have been predisposed thereafter to HIE as a result of the stroke, and/or that the HIE could have also been caused by seizures after the peri-natal stroke. Dr Govender assessed K on two separate occasions and found, consistent with the notion of a stroke, that:

(a) K predominantly uses the left side of his body and that the right side of his body showed spasticity resulting in right sided hemiparesis;
(b) K had a contracture at his right elbow joint which was fibrosed from disuse, indicating that he suffered right sided hemiparesis as his left side and other elbow joint did not have any contractures;
(c) K's right lower limb had decreased power and a contracture of the right ankle, which indicated that he suffered from hemiparesis as he suffered no such contracture to his left ankle;
(d) The right side of K's body had a power grade of 4/5 and his injury was predominantly on the right side, and therefore he is classified as a hemiplegic;
(e) The MRI scan of K's brain showed a left MCA infarct which predominantly affected the left side of his brain with encephalomalacia. The left side of the brain controls the right side of the body, and therefore K suffered from hemiparesis, as the predominant injury is on the left side of his brain which confirms that the minor child suffered a stroke and not HIE, as with HIE one would get dual affectation;
(f) There is significant asymmetry with regards to the minor child's brain which is very remarkable.

Dr Govender accordingly was of the view that the clinical examination is consistent with the left middle cerebral artery territory artery infarction on K's MRI scan as he displays right sided contractures and hemiparesis which in her view confirms a stroke. Dr Kara agreed that if there is affectation of the right side of K's brain, that this could also happen with a stroke and that his circumstances could be such a case. But he also confirmed that strokes sometimes mimic HIE.

[73] The findings during the clinical examination are supported by the following evidence of the plaintiff herself, namely:

(a) K has weakness only on the right side of his body and has good use of his left arm and leg;

(b) K is able to use his right arm and is able to grasp, but not hold onto something;

(c) K is able to throw with his left hand and kick with his left leg but is unable to do so with the right side.

[74] Dr Govender testified that there are many factors which could have caused a stroke during the plaintiff's pregnancy. A pregnancy state is a pro-coagulant state during which a lot of clotting factors are present, including from infections such as syphilis. During the last three days of pregnancy, there is statistically a 34 fold increase in the risk of clots occurring which could cause a stroke. Dr Kara confirmed that there is a high risk of stroke in pregnancy.

[75] Dr McLynn conceded that smoking may be a risk factor which can cause a stroke in a minor child.

[76] Insofar as the causality and timing of the insult is concerned, Dr Kara relied on the following factors as being relevant, which they indeed are at the level of determining the probable cause of K's condition:

(a) that there were no pre-conceptual risk factors;[30]

(b) the comments in the medical records recorded by the defendant's staff that the labour was prolonged;[31]

(c) the lack of proper foetal heart rate monitoring during the last vital hours of labour;[32]

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[30] Smoking and syphilis were in his view unlikely causal factors (Exhibit "B": report of Dr Kara para 9.1 at 235; Dr Kara's evidence at 419-421).

[31] Exhibit "B": report of Dr Kara para 9.2 at 235. This comment is recorded in exhibit D": the neonatal records at 14. The plaintiff is recorded as being two (2) cm dilated and having moderate contractions at 02h00 and only being four (4) cm dilated at 12h00 - see exhibit "C" at 4 and 22. This would equate to at least 10 hours in the latent phase of labour. The summary of the plaintiff's labour records her first stage as being 10 hours and 10 minutes in duration - exhibit "C" at 23.

[32] Exhibit "B" report of Dr Kara para 9.2 at 235.

(d) the alleged application of fundal pressure and the delivery being facilitated by an episiotomy;[33]
(e) the neurological depression at birth, with low Apgar scores,[34] resuscitation being performed with oxygen, suction and naloxone;
(f) that there was adequate evidence to confirm a moderate severe encephalopathy where K had tone disturbances, depressed reflexes and convulsions soon after birth, poor suck and he did not cry for several days;
(g) that there were no major antenatal factors that affected the placental circulation, or infections or injuries that could cause encephalopathy;
(h) that the neonatal records indicated that K was admitted for severe birth asphyxia and respiratory distress, was obviously compromised at birth and had an abnormal neurology with convulsions, had raised cardiac enzymes (CK 1238), had no biochemical suspicion of infection, had a relatively normal spinal puncture (no meningitis) and that there was a normal CT scan and cranial ultrasound, which were done seven days after the birth;
(i) there did not appear to be any other cause of the neonatal encephalopathy other than hypoxic ischaemic injury; and
(j) there is no documented evidence or history of any postnatal event that could have caused the cerebral palsy.

[77] With reference to the authoritative work of *Dr Volpe: Neurology of the Newborn, 2008,* Dr Kara was of the opinion that the criteria set for a diagnosis of intrapartum insult had been met and that, on probability, K had suffered such an insult.
[78] With reference to the American Congress of Obstetrics and Gynaecology (ACOG) criteria, Dr Kara was of the opinion that the most probable conclusion was that 'there was acute intrapartum hypoxic injury of sufficient severity to cause cerebral palsy', that probability being indicated by:
(a) the lowered Apgar scores;
(b) the MRI scan showing features consistent with acute profound hypoxic injury and prolonged partial injury at term;
(c) the respiratory compromise and probable cardiac compromise/muscle injury experienced by K at birth;

[33] Exhibit "B" report of Dr Kara para 9.2 at 236.

(d) the absence of a sentinel hypoxic event occurring before delivery or during labour being recorded;
(e) the lack of foetal monitoring immediately prior to birth;
(f) there being no evidence of other proximal or distal factors that could be considered as contributing; and
(g) the development of spastic quadriplegia or dyskinetic cerebral palsy.
He testified that the new criteria (2014) consider all potential contributing factors and, even where all criteria might not be met, if the probability favours intrapartum hypoxia, then that is likely. He concludes that the probable cause of K's dyskinetic cerebral palsy was an intrapartum hypoxic injury.

[79] Dr Singh agreed that the neonatal stroke contended for by the defendant could not have happened after K's birth, and that if that was the cause for his condition, that it had to have occurred shortly prior to his birth. Yet there was no sentinel event.

[80] The defendant's contention that K suffered a perinatal ischaemic stroke/neonatal stroke rather than birth asphyxia, is premised upon the assumption that the injury to K's brain was asymmetrical, and confined to the left middle cerebral artery distribution, rather than being bilateral. A stroke would involve an occlusion. Dr Singh was adamant that the occlusion would have been in the left middle cerebral artery, that the artery would have been totally occluded for some time (even weeks) and that the tissue immediately adjacent to the occlusion would have died. She accepted that, for her version to be correct, the damage would have been to the left-hand side of K's brain given the paralysis predominantly to his right side.

[81] That notion is however largely dispelled by the joint minute of the radiologists Prof Lotz and Dr Reitz who found the injury to be bilateral, albeit with an additional asymmetrical focal atrophy in the left posterior parietal region. The question arising then is whether there was anything that could have contributed to the bilateral injury observed but would also be compatible with K's condition being caused by a perinatal ischaemic stroke/neonatal stroke which would account for the asymmetry.

[82] Dr Singh proffered that the bilateral damage might be accounted for by K's brain having been more susceptible to the rigours of birth, due to him having suffered a stroke shortly prior to his birth. That hypothesis was however only suggested by Dr Singh under cross-examination, when faced with the fact that there was bilateral damage to

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[34] Even accepting the effects of pethidine having been administered.

K's brain. It was not mentioned in the summary of her expert opinions, not put to any of the Plaintiff's witnesses (the inference being that she had also not mentioned it to the defendant's counsel), and was not supported by other evidence adduced by the defendant. She conceded that this hypothesis had not been offered in her report.[35]

[83] Although Dr Singh initially contended that the stroke could have occurred between the 20th week of foetal life through to the 28th post-natal day, she confined the occurrence of such a stroke in the case of K to after the 36th week of gestation until birth. There was however no evidence of any event recorded in the plaintiff's medical history that supported this notion. Indeed, even the first stage of labour indicated a seemingly healthy foetus. The CT and ultrasound scans conducted seven days after K's birth, although contended not to be definitive, also did not demonstrate that K had in fact suffered a stroke. Dr Singh conceded under cross-examination that there is no evidence of a stroke on the scans conducted of K's brain. She explained this on the basis that the occlusion must have dissolved. There is however no evidence of this. In the final analysis of her evidence, Dr Singh conceded that there was bilateral damage to K's brain and she accepted that this would have occurred during labour. The explanation, at best for the defendant, she could advance was that his brain was more susceptible during birth by virtue of the stroke.[36]

[84] As regards the timing of the infarct, Prof Lotz could only state that it was after 36 weeks. Dr Govender stated that with the 500 odd babies that she has treated, seizures which occur within the first six (6) hours of birth, indicate that the insult had occurred in the antenatal period, and not the intrapartum period. K suffered seizures within the first six hours of birth. She therefore contended that he had suffered a stroke in the antenatal period but after 36 weeks of gestation. This conclusion it was said, was further confirmed in an article by *Philan et al* which found that seizures within the first 12 hours of birth are likely to be indicative of an antenatal insult. Dr Govender testified that with a HIE, seizures occur later, after 12 hours from her clinical experience with children in the nursery who have HIE, because the damage does not occur immediately.

[85] Prof Lotz accepted that the injury seen on the left of K's brain could be as a result of a stroke, and that the injury is predominantly on the left hemisphere. There are two arteries which supply blood to the brain, namely the left carotid artery and the right carotid artery. With a HIE during labour in the majority of cases, one usually finds that

[35] Cross-examination of Dr Singh at 936.

there is a decreased blood flow in both arteries, and therefore one would get global cerebral insult and not usually the degree of asymmetry observed in K's brain. Global hypoxic insult in most cases affects both arteries. Prof Lotz conceded that in less than 10 per cent of cases would one find one sided encephalomalacia with global hypoxic ischemic insult. That would on probability make a HIE rare or unlikely in the present case. He maintained however that although K's set of circumstances would be rare, he found support for his conclusion that K had not suffered a stroke but HIE in an article that was published in 2008 titled *Parasagittal Lesions and Ulegyria in Hypoxic Ischemic Encephalopathy, Neuroimaging Findings and Review of Pathogenesis.*

[86] Doubt was sought to be cast on the reliability of that study and whether it is reasonable, sound and logical. The study apparently considered only 14 patients, which is a relatively small statistical group. The 14 patients further all had suffered HIE and none a stroke and only four (4) patients presented with the ulegyria. Prof Lotz could not give evidence regarding the remainder of the patients who did not present with ulegyria.

[87] Dr Reitz's conclusion was that K had suffered a stroke and an infarct in a specific area of the brain which is supplied by a specific blood vessel, namely the left carotid artery, which explains the damage predominantly to the left hemisphere of K's brain. It was argued that a perinatal stroke after 36 weeks' of gestation would have predisposed K to suffer injury and not withstand the rigours of labour.[37] Such a conclusion, it was said was, corroborated by the MRI findings which confirm limited damage to the right side of K's brain, which would have occurred during the birthing process as his brain would be susceptible to the rigours of labour, as confirmed by both Dr Singh and Dr Govender.[38] Dr Singh, the defendant's neonatologist, opined that the limited damage to the right side of the minor child's brain on the MRI could have been caused by the collateral supply to the right side of K's brain, which supply originates from the left side

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[36] Cross-examination of Dr Singh at 922-923.

[37] A normal baby going through the normal stresses of labour will react normally, but a baby who has an abnormal brain, in that the blood supply is affected, might not respond to hypoxia or hypercarbia similarly like a normal baby, and will come out with low Apgar scores as was the case with K.

[38] The opinions of Dr Singh and Dr Govender are further confirmed by medical articles which are reflected in exhibit F. F33 refers to a study by Karen B. Nelson which confirms that K's clinical features at birth did not exhibit signs of asymmetrical hemiplegia, and that he presented with neonatal seizures and hypotonia at birth. F34 confirms that a CT scan is not sensitive enough to detect a stroke, contrary to the opinion of Dr Kara. A stroke may cause children to present with neonatal depression, suggestive of birth asphyxia. Normal pregnancy is a pro-coagulant and pro-inflammatory condition and a risk of a stroke is higher in pregnancy than in a non-pregnant state. F91 confirms that a baby with a pre-existing cerebral insult reacts differently to the stresses of labour, referred to as brain priming, which would result in a baby not reacting appropriately to the stresses of labour, recognised also in Volpe's standard textbook.

of the brain, and is supplied by the left carotid artery, which could also explain the limited affectation on the right side of the minor child's brain.

[88] The defendant was in my view not able to dispel the opinion of Prof Lotz that a strong probability factor is the presence of uligyria in the affected area, which mitigates against K having suffered a stroke and suggests as a more probable conclusion that there was a partial prolonged hypoxic ischaemic event. The defendant could also not dispel Prof Lotz's opinion that there would have to have been an occlusion of two arteries to cause the bilateral damage seen on the MRI scan. Dr Govender was not able to provide any positive evidence to dispel this evidence of Prof Lotz as a matter of probability.

[89] In my view K's condition was caused by the negligence of the defendant's employees.

Was the injury in any event unavoidable?

[90] The defendant argued in the alternative, that even if K suffered a HIE resulting in his cerebral palsy, which was caused by the negligence of the defendant's employees at the level of factual causation, that it should nevertheless be excused from legal liability as such injury, and the damages arising there from, would have occurred in any event. Such a defence is in the nature of a confession and avoidance.[39]

[91] There is no onus on a plaintiff to adduce evidence to prove, on a balance of probabilities, what the lawful non-negligent conduct of the defendant should have been. All that is required, is the substitution of a hypothetical course of lawful action, and posing the question as to whether upon such hypothesis, the plaintiff's loss would have ensued or not.[40]

[92] The argument is based on the hypothesis that if it is found that the foetus only displayed signs of foetal distress after 16h50 / 16h20, which should have been detected but were not, that the procedure by which the plaintiff would have had to have been transferred to the King Edward Hospital for an emergency caesarean section delivery, would have resulted in same only being offered to her after a period in excess of two (2) hours, during which time the foetus in any event and already have suffered foetal distress. Accordingly, it is argued that the failure to monitor the foetus after 16h20 but prior to delivery, made no difference in the outcome of the foetus, as a caesarean

[39] *Johannesburg City Council v Television and Electrical Distributors* 1997 (1) SA 157 (A).

section delivery could never timeously have been performed upon her at a referral hospital.

[93] The factual basis for that argument was developed as follows in the evidence of Dr Moran, who contended that even **if** there was documented foetal monitoring from the period 16h30 to 19h10 it would have made no difference in the outcome of the minor child for the following reasons:

(a) The last documented foetal monitoring at 16h20 / 16h50 (according to Ms Adams) indicated that the foetal condition was good. If the CTG had ran for the 90 minutes as mandated and required in terms of the guidelines and if one were to have discovered variable decelerations at this point, only then would the nurses have been justified in assuming that it was a suspicious CTG;

(b) Thereafter, the nurses at 18h00 (90 minutes after 16h30) would have to reassess the plaintiff to determine how her labour was progressing. If foetal distress was diagnosed, the nurses at the hospital would have to contact the doctors at the King Edward Hospital. Once contact with the doctor was established, the doctor would have to be advised of the plaintiff's condition;

(c) If the doctor, after being informed of the CTG, determined that the plaintiff was a candidate for transferal, the doctor would have to provide instructions to transfer the plaintiff to the King Edward Hospital, unless the plaintiff was 'fully dilated at the time'. If she was fully dilated, the advice would have been to extract the minor child at the hospital and resuscitate him depending on how far the plaintiff progressed in labour. As the plaintiff was fully dilated at 18h00 it was unlikely that a doctor would have performed a caesarean section delivery to extract the foetus thereafter, as the delivery was imminent.

(d) If the plaintiff was not yet fully dilated, the doctor may have called for an ambulance for her transfer to the King Edward Hospital. The hospital would then have to contact the ambulance services for transfer of the plaintiff which is a laborious process. The EMRS services would have dispatched an ambulance in accordance with their transfer system, and the plaintiff would have been categorized as a 'red code'. The ambulance would have to travel from town to the hospital to transfer the plaintiff, which would take time. On arrival at the hospital, the EMRS services would have to evaluate the plaintiff and thereafter obtained a handover from the hospital staff. The plaintiff would have to

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[40] *Lee v Minister of Correctional Services* 2013 (2) SA 144 (CC).

be trolleyed out to the ambulance and transported to the King Edward Hospital. The process of the ambulance arriving and the plaintiff being transferred to the King Edward Hospital would have taken at the very least, according to Dr Moran, one and a half hours.[41] If it was decided at the King Edward Hospital that the plaintiff required a caesarean section delivery, this could not be done immediately. Consent would have to be obtained, tests would have to be done, and an anaesthetist would have to assess the plaintiff to make sure she is fit for anaesthetic. Dr Moran stated that the pre-surgery safety checklist, draping the plaintiff, cleaning the plaintiff and putting the plaintiff on anaesthetic, would have taken more time before the plaintiff actually underwent a caesarean section delivery. Accordingly, Dr Moran was of the view that it was highly unlikely that the plaintiff would have been able to get a caesarean section delivery prior to the birth of the minor child.

(e) Based on the evidence of Ms Adams, the midwife monitoring the plaintiff's condition who had testified that she had commenced the CTG at 16h30 (in fact she had said 16h20 being the time of the entry) and ended same at approximately 16h50, and that the foetal heart rate was reactive, indicating a normal foetus, and that Ms Adams testified that the plaintiff was fully dilated at 18h00, and if the King Edward Hospital had to have been contacted for the transfer of the plaintiff, Dr Moran argued that if foetal distress should have been detected after 16h50, it would have made no difference in the outcome of the minor child as he would have already suffered hypoxic ischemic insult to the brain even on the plaintiff's version of events. The harm would have in any event occurred.

[94] The aforesaid hypothesis however proceeds on the premise that the last CTG monitoring ended at 16h50, that no greater or more vigilant monitoring was required prior to that (notwithstanding Dr Moran conceding that the level of care received by the plaintiff prior to that was substandard), and that the labour was not one with risk factors to which more attention had not been given, all of which are incorrect as I have endeavoured to show above.

[95] Having regard to my findings earlier in this judgment, the transfer of the plaintiff to King Edward hospital or a similar institution was indicated earlier that day, at a stage which would have left sufficient time for the plaintiff to have been afforded proper care.

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[41] Mr Padayachee who was in charge of the EMRS services testified that the ambulance could have taken approximately two (2) to three (3) hours before the plaintiff was transported to the King Edward Hospital.

In my view the alternative argument advanced by the defendant has therefore also not been established.

Conclusion

[96] As sought to be demonstrated above, the probabilities favour the conclusion that K suffered a mixed pattern of a partial prolonged albeit of shorter duration, and acute profound hypoxic ischaemic injury prior to his birth, which became acute during the period when there is no record of his condition.

[97] The hypoxic-ischaemic insult, as described by Prof Lotz, and agreed to by Dr Reitz, would accord with the fact that the foetus was in some distress during the active phase of labour (approximately 12h00 to 16h20), probably as a consequence of it being partially starved of both blood flow and oxygen, by virtue of the placenta being 'squeezed off' during the plaintiff's contractions immediately prior to birth, and the foetus being slow to recover from such contractions.

[98] The plaintiff has accordingly discharged the burden of proving that the defendant's medical personnel were negligent in their treatment of or their failure to treat the plaintiff prior to K's birth and that the injury to his brain, and subsequent cerebral palsy, was caused as a consequence of that negligence.

Costs

[99] The plaintiff has been successful. In the exercise of my discretion on costs, it is appropriate that the defendant be directed to pay the plaintiff's legal costs to date on the High Court scale, including the costs of the experts, in accordance with the terms of the order set out below.

Order

[100] The following order is made:

(a) The defendant is directed to compensate the plaintiff, in her representative capacity as mother and as natural guardian of K, a boy born on the 14th of May 2009, and in her personal capacity, for the damages claimed in this action, either as proved or agreed;

(b) The defendant is directed to pay the plaintiff's taxed or agreed party and party costs on the High Court scale, such costs to include:

(i) the costs of the plaintiff's attorney attending upon any consultations with witnesses in preparation for trial, including the consultations with the under-mentioned expert witnesses;

(ii) the costs of senior and junior counsel where employed, including the reasonable and necessary costs of their preparation for trial, and for their attendance upon consultations with the under-mentioned expert witnesses and the plaintiff;

(iii) the qualifying fees of the under-mentioned expert witnesses, including the costs of the preparation of their reports, their attendance fees, and the fees to qualify themselves to testify at the trial and for any necessary consultations with the plaintiff's attorney and counsel (with the quantum of fees, and expenses, to be determined by the taxing master), namely:

(aa) Dr Y Kara, the specialist paediatrician;

(bb) Dr D McLynn, the obstetrician and gynaecologist;

(cc) Prof J Lotz, the professor of radiology; and

(c) The proceedings are otherwise adjourned sine die.

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Koen J

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