IN THE HIGH COURT OF SOUTH AFRICA

(GAUTENG DIVISION, PRETORIA)

Case Number: 83019/2016

M M obo O
Z M Plaintiff

and

THE MEMBER OF EXECUTIVE COUNCIL FOR
HEALTH, GAUTENG PROVINCIAL GOVERNMENT Defendant

JUDGMENT

MOLEFE J

[1] The plaintiff Ms M M, instituted this action on behalf of her minor daughter, O M ('O') against the defendant, the Member of the Executive Council for Health, following the plaintiff' s admission to the defendant's Pholosong Hospital on 21

March 2010, for the assessment, management and monitoring of the plaintiff's pregnancy and labour, the delivery of her baby O as well as the post-natal care and treatment of O.

[2] It is the plaintiffs case that as a result of the negligence of the medical and nursing staff at the Pholosong Hospital:

2.1 O suffered an intrapartum hypoxic-ischaemic insult to her brain, compounded by a post-partum hypoxic-ischaemic insult, as a result of which she has been born with severe permanent mixed cerebral palsy, microcephaly and profound intellectual disability;

2.2 O consequently suffered damages which the plaintiff as her mother and natural guardian is entitled to claim from the defendant.

[3] The parties have agreed that there shall be separation of the issues of liability and quantum in terms of rule 33(4)[1] subject to the approval of the Court. The separation of issues was granted. The trial proceeded only on the issue of liability and the issue of quantum was postponed *sine die.*

[4] The plaintiff's claim is based on delict, ie breach of legal duty (wrongfulness), and a breach of a duty of care (negligence), which the defendant's medical and nursing staff at the Pholosong Hospital had to the plaintiff and her baby O, which caused harm to O (causation and harm)[2].

[5] The defendant has admitted the legal duty of care relied upon by the plaintiff subject to the availability of resources at the Pholosong Hospital[3].

[6] The plaintiff relies on the vicarious liability of the defendant for the conduct of the medical and nursing staff at the Pholosong Hospital who attended to the plaintiff

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[1] Uniform Rules of Court.
[2] Pleadings pages 7-9 and 12, paras 5 and 8.
[3] Pleadings page 20, para 5.

and O[4].

[7] The defendant has admitted vicarious liability by admitting that the medical practitioners and nursing staff at the Pholosong Hospital who attended to the plaintiff before, during and after the birth of her baby, up until their discharge from the hospital acted within the course and scope of their employment[5].

[8] The defendant denies all allegation of breach of legal duty and duty of care which he and his medical and nursing staff had to the plaintiff, and denies any causal link between any such negligence and O's brain damage which led to spastic quadriplegic cerebral palsy, epilepsy and profound intellectual disability[6].

Relevant Facts

[9] The ante-natal records of the plaintiff and the hospital records relating to the plaintiffs admission to the hospital on 21 March 2010, for the monitoring, assessment and management of the plaintiff's labour, the delivery of her baby, and the baby's resuscitation after birth are not in dispute. These records are before the Court by agreement in that they correctly reflect the inscriptions made at the time by the medical and nursing staff, as well as the results of the monitoring and tests performed on the plaintiff and her baby.

[10] The plaintiff fell pregnant with baby O during 2009 and visited Dunottar Ante-Natal clinic during her pregnancy on 8 (eight) occasions from 12 October 2009 to 15 March 2010. She had a previous normal vaginal delivery in 1997 but the child died at the age of 8 (eight) years. O was her second pregnancy, and she had a third pregnancy and gave birth by normal vaginal delivery in May 2017. This third child is healthy and normal.

[11] The plaintiff was known to be HIV positive and was on anti-retroviral (ARV)

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[4] Pleadings page 9, para 6.
[5] Pleadings page 21, para 8.
[6] Pleading s p ages 21 -22, paras 10 -1 2.

treatment. O's HIV PCR subsequently tested negative. According to the ante. natal clinic card, there was concern about Symphysis Fundal Height (SFH) being reduced and the plaintiff was referred for an ultrasound.

[12] The plaintiff was admitted at the Pholosong Hospital on 21 March 2010 at 20:15 with complaints of lower abdominal pains since 4 (four) days ago. She was assessed by midwives and the assessment revealed no abnormalities or concerns. It was found that her membranes had not ruptured, there was no antepartum haemorrhage and the baby's heart rate was normal.

[13] A short while later at 20:35 on 21 March 2010, the plaintiff was examined by a Doctor who recorded that:

13.1 Her blood pressure was slightly raised and a note was made about PIH (Pregnancy Induced Hypertension). Foetal movements were present and a CTG (Cardiotocography) examination was re-active, and this examination of the heartrate of the baby and its well-being was normal.

13.2 On the vaginal examination, the cervix was closed and there was no dilation at that stage. She was therefore not yet in labour and the doctor admitted her to the ward.

[14] On 22 March 2010 at 06:00, the hospital records reveal that the plaintiff was feeling better. On vaginal examination by the nurse, she was found to be not in labour and her cervix was closed. The foetal heart rate was normal at 140-150 BMP (beats per minute).

[15] At 08:00 on 22 March 2010, the plaintiff was seen and examined by Dr Maseko and the following was recorded by the doctor in his clinical notes:

15.1 The plaintiff was admitted on 21 March 2010 with elevated blood pressure that subsided on treatment. and was stable with a normal blood pressure.

15.2 Her cervix was 50% effaced and admitted 1(one) finger. The height of

fundus (HOF) was 34/40 , and he questioned that the foetus *"was small for dates".* Her pelvis was considered to be adequate by the doctor and the estimated weight of the baby was 2.5 kg to 2.8 kg.

15.3 A CTG was done and the heart rate of the foetus had a normal base line of 130 8PM. The mother was informed about the results of the examination and the CTG, and of the need to take the baby out.

15.4 The doctor made a specific note at 08:00 that induction of Ms M's labour was to be commenced by 4 (four) Prostin tablets ("Prostin 4 stat"). The 4 (four) Prostin tablets were inserted into her vagina to induce labour. The doctor's instruction was that the plaintiff and her foetus were to be monitored by CTG after 4 (four) hours and thereafter every 2 (two) hours. She was sedated with Atarax and Pethidine which was given immediately.

[16] At 08:30 on 22 March 2010, there is a CTG tracing of the plaintiff and her foetus for a period of 5 (five) to 6 (six) minutes. The baseline on the CTG tracing is 140/minute (normal). There were no accelerations of the foetal heart rate and 2 (two) decelerations of the heart rate (120/minute). There was no tracing of the plaintiff's contractions.

[17] From the time of this CTG tracing at 08:30 until 12:20 on 22 March 2010 when the plaintiff was admitted to the labour ward for the delivery of her baby, there are no records of any monitoring of the plaintiff by the nursing staff or that the plaintiff was seen or examined by a doctor in this period of 3 hours and 50 minutes.

[18] There is also no indication in the records that the foetal heart rate or maternal contractions were monitored, either intermittently or continuously after the plaintiff's labour was induced by means of the Prostin tablets at 08:00 on 22 March 2010 until the birth of O at 12:45.

[19] According to the labour records from the labour ward, the plaintiff was

admitted to the labour ward on 22 March 2010 at 12:20. She was restless+++ and bearing down with each contraction, and the nursing staff were unable to obtain the foetal heart rate (FHR). Her cervix was 7 to 8 centimeters dilated and 80% effaced, and her membranes were still intact at that stage. At 12:30, the plaintiff's membranes ruptured and it is recorded that the second stage of the labour started at 12:30. O was born by normal vaginal delivery at 12:45 with Apgar Scores of 2/10 at 1 minute, 3/10 at 5 minutes and 4/10 at 10 minutes, ie there was no spontaneous respiration.

[20] The doctor noted at 13:20 that O was being ambubagged, was pale, her heart rate was >100 b/m and there was no spontaneous respiration. His examination revealed an Apgar of 2 at 10 minutes. The doctor further noted:

20.1 no improvement after 2(two) minutes of him performing face mask ventilation and he intubated O. He commenced endotracheal tube (ETT) ventilation;

20.2 five(5) minutes after intubation and ETT bagging, spontaneous respiration commenced. O's respiration was still irregular but "deep", her heart rate "good", and her colour pale. O weighed 2.51 kg at birth, was floppy++ and did not cry at birth. She had to be resuscitated after birth.

[21] Nothing was recorded with regard to O's clinical condition from birth until 13:20 when the doctor recorded his resuscitation. O's birth weight was 2510g, her length was 45cm and head circumference 33cm. According to the Progress Report, the nurses noted on admission to the Special Care Unit (SCU) at 13:55 that O was *"ill and had difficulty in breathing on oxygen per nasal prongs. Fitted ± 3 seconds".* At 23h00 O had another seizure which was treated with Rivotril.

[22] According to the attending paediatric doctor's note on 23 March 2010 (day 2), O was *"floppy+++with no response to stimulation".* Azithromycin (AZT) was commenced to prevent mother to child HIV transmission. On 24 March 2010, O had another convulsion. She had to be tube fed. A speech therapist was consulted to stimulate sucking and swallowing. A physiotherapist was also consulted for her

"floppiness" (hypotonia).

[23] O had ongoing feeding problems for which cup feeding had to be commenced before discharge. She was discharged on 5 April 2010 at 14 days of age. At long-term follow-up she was diagnosed with spastic quadriplegic cerebral palsy epilepsy and global retardation.

Expert Evidence and Joint Minutes

[24] The plaintiff filed summaries/reports of the following experts:

24.1 Prof G F Kirsten, neonatologist;

24.2 Prof R Solomons, paediatric neurologist;

24.3 Dr GS Gericke, genetics;

24.4 Dr C Harris, nursing expert;

24.5 Prof J W Lotz, radiologist;

24.6 Dr C Sevenster, obstetrician and gynaecologist;

24.7 Dr J Snyman, pharmacologist.

[25] The defendant filed summaries/reports of the following experts:

25.1 Dr ET Opai-Tetteh, obstetrician and gynaecologist;

25.2 Dr N Duma, paediatrician;

25.3 Prof PA Cooper, paediatrician and neonatologist;

25.4 Prof D Du Plessis, nursing expert;

25.5 Dr D Pearce, paediatric neurologist;

25.6 Dr T Kamolane, radiologist;

25.7 Dr L Bhengu, geneticist.

[26] The experts of similar expertise for the plaintiff and the defendant held joint meetings and furnished the Court with joint minutes. In the joint minutes the corresponding experts reached agreement regarding the facts dealt within their joint minutes and several issues in dispute. The aspects on which the experts agreed in their joint minutes constitutes further evidence on which I can rely in determining the

issues in this case[7].

Issues in dispute that require leading of expert evidence

[27] The central issues in *casu* are the following:

27.1 Whether the administration of 4 Prostin tablets to the plaintiff to induce labour was an overdose or too high dosage;

27.2 Whether the defendant's medical and nursing staff were negligent in administering such overdose and failing to monitor the plaintiffs induced labour up until the birth of O;

27.3 Whether the administration of an overdose of Prostin tablets probably caused hyperstimulation of the plaintiff's uterus, which probably resulted in hypertonic contractions, foetal distress, and an acute profound hypoxic-ischaemic insult to baby O's brain, leaving her with permanent brain damage;

27.4 Whether there was negligence on the part of the medical and nursing staff of the defendant in resuscitating baby O after her birth, and whether such negligence compounded or contributed to the acute profound hypoxic-ischaemic insult suffered by O;

27.5 Whether, but for the negligence of the medical and nursing staff of the defendant, O's permanent brain damage would probably have been prevented.

The Relevant Legal Principles

[28] The test for negligence was authoritatively formulated by the Supreme Court of Appeal (SCA) in *Kruger v Coetzee*[8] and the test was restated in *Mukheiber v Raath*[9] in light of subsequent developments. In *Kruger v Coetzee,* Holmes JA stated that the test for establishing the existence, or otherwise of negligence was as follows:[10]

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[7] Glenn M ark BEE v The Road Accident Fund 2018 (4) SA 366 (SCA) para s 66 and 73.
[8] 1996(2) SA 428 (A).
[9] 1999 (3) SA 1065 (SCA) para 31.
[10] Ibid FN 8 at 430 E-G.

"For purpose of liability, culpa arises if-

(a) *a diligent paterfamilias in the position of the defendant –*

(i) *would foresee the reasonable possibility of his conduct injuring another in his person or property and causing him patrimonial loss; and*

(ii) *would take reasonable steps to guard against such occurrence; and*

(b) the defendant failed to take such steps...

Whether a diligent paterfamilias in the position of the person concerned would take any guarding steps at all and if so, what steps would be reasonable, must always depend upon the particular circumstances of each case. No hard and fast basis can be laid down".

[29] Obviously when one is dealing with the possible negligence of a specialist medical practitioner or a professional nurse, as *in casu,* the test of the ordinary diligent *paterfamilias* or reasonable man alluded in *Kruger supra* cannot be applied.

[30] In *Oppelt v Department of Health*[11] the Constitutional Court summarised the position as follows:

" In simple terms, negligence refers to the blameworthy conduct of a *person who has acted unlawfully. In respect of medical negligence, the question is how a reasonable medical practitioner in the position of the defendant would have acted in the particular circumstances.*

…

The negligence of medical practitioners is assessed against the standards at the medical profession at the time."

[31] Whether the medical practitioners and the professional nurses who attended to the plaintiff and her baby were negligent, must therefore be assessed against the respective reasonable standards of the medical and nursing profession applicable at

[11] 2016 (1) SA 325 (CC) paras 71 and 73.

the time.

[32] In *Michael and Another v Linksfield Park Clinic (Ply) Ltd and Another*[12], the SCA approved the approach of the House of Lords in the case of *Bolitho City and Hackney Health Authority*[13] regarding the evaluation of expert evidence in a medical negligence case:

> *"That being* so, *what is required in the evaluation of such evidence is to determine whether and to what extent their (the experts') opinions advanced are founded on logical reasoning. That is the thrust of the decision of the House of Lords In the medical negligence* case *of Bolitho City v Hackney Health Authority [1998] AC 232 (HL(E)). With the relevant dicta in the speech of Lord Browne-Wilkinson we respectfully agree. . .*
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> *[37] The Court is not bound* to *absolve a defendant from liability for allegedly negligent medical treatment or diagnosis just because evidence of expert opinion, albeit genuinely held,* is *that the treatment or diagnosis in issue accorded with sound medical practice. The Court must be satisfied that such opinion has legal basis, in other words that the expert has considered comparative risks and benefits and has reached a defensible conclusion (at 241* G*-242 B).*
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> *…*
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> *[39] A defendant can properly be held liable, despite the support of a body of professional opinion sanctioning the conduct in issue, if that body of opinion is not capable of withstanding logical analysis and is therefore not reasonable. However, it will very seldom be right to conclude that views genuinely held by a competent expert are unreasonable. The assessment of medical risks and benefits* is a *matter of clinical judgment which the court would not normally be able to make without expert evidence and it would be wrong to decide a* case *by simple preference where there are conflicting views on either side, both capable of logical support. Only where expert opinion cannot be logically*

[12] 2001(3) SA 1188 at 1200-1201 paras 36 and 39.

supported at all will it fail to provide 'the benchmark by reference to which the defendant's conduct falls to be assessed' " *(at 243 A-E)."*

[33] In determining causation in a medical negligence case, Corbett JA set out the position as follows in *Blyth v van Den Heever*[14]*:*

> *"In determining what in fact caused the virtual destruction of appellant's arm, the Court must make its finding upon* a *preponderance of probability. Certainty of diagnosis is not necessary. If it were, then, in a field so uncertain and controversial* as *the one which I have thus far endeavoured to delineate,* a *definitive finding would become an impossibility. Bearing in mind that in this* case *the appellant bears the burden of proof, the question* is *whether it is more probable than not that large scale ischemia, coupled with sepsis caused the damage".*

[34] In *Minister of Safety and Security v Van Duivenboden*[15] the SCA held that:

> *"A plaintiff is not required to establish the causal link with certainty but only to establish that the wrongful conduct* was *probably* a *cause for the loss, which calls for a sensible retrospective analysis of what would probably have occurred, based upon the evidence and what can be expected to occur in the ordinary course of human affairs rather than an exercise in metaphysics".*

Negligence of the Medical and Nursing Staff

[35] The plaintiff's counsel submitted that in determining whether there was negligence on the medical and nursing staff who attended to the plaintiff and O and whether such negligence caused or contributed to the brain damage that O suffered, the two questions to be determined by this Court are the following:

35.1 Whether the administration of 4 (four) Prostin tablets to the plaintiff to

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[13] 1998 {AC) 232 (HL(E))
[14] 1980 (1) SA 191 (A) 207.
[15] 2002 (6) SA 431 (SCA) at par 25.

induce labour was an overdose or a too large a dosage;

35.2 If so, whether such an overdose probably caused hyperstimulation of the plaintiffs uterus which probably resulted in hyper-contractions and foetal distress.

The Overdose or Too Large Dosage of Prostin

[36] In this regard, the plaintiff relied on the evidence of Dr Chris Sevenster, specialist obstetrician and gynaecologist, and Professor Gert Kirsten, specialist paediatrician and neonatologist.

[37] Dr Sevenster's undisputed evidence was that 4 Prostin tablets were administered vaginally to the plaintiff at approximately 08h00 on 22 March 2010 to induce labour. This is apparent from Dr Maseko's clinical notes during his evaluation of the plaintiff at 08h00 that the induction of the plaintiff's labour was 'commenced' by means of 4 Prostin tablets. As aforementioned, the prescription and clinical notes of Dr Maseko are not in dispute.

[38] Dr Sevenster's undisputed evidence was that during 2010, Prostin E2 oral tablets were widely used in obstetrical units for the induction of labour. Many hospitals used the tablets "off label". This means that although Prostin E2 tablets were indicated to be administered orally for induction, they were usually administered vaginally. Dr Sevenster explained that the pharmacological function of Prostin E2 tablets is to stimulate the muscle of the uterus in an artificial way, which leads to contractions to artificially stimulate the start of labour.

[39] Dr Sevenster testified that in his experience of more than 30 (thirty) years, and based on the standards that were applicable at the time as set out in the *Guidelines for Maternity Care in South Africa, 2007*[16] Prostin E_2 tablets should be administered as follows: *"1mg intravaginally four hourly for four doses".* One Prostin

[16] Exhibit 'F', Guidelines page107.

E2 tablet contained 0.5 mg Prostaglandin. This means that 2 (two) tablets, ie 1 mg had to be administered intravaginally for four doses. Dr Sevenster testified that the 4 tablets administered to the plaintiff were equal to 2 mg of Prostin which was double the recommended dosage, and based *on* his experience and the Guidelines, this was an overdose.

[40] Regarding the establishment of Guidelines, Dr Sevenster agreed with the statement by Prof Buchmann, *(E J Buchmann and R C Pattison, Babies Who Die from Labour-related lntrapartum Hypoxia: A Confidential Enquiry in South African Public Hospitals (2006) Tropical Doctor 88),* in which Prof Buchmann states the following:

> "So to standardise care, we produced guidelines as a multidisciplinary group with obstetricians, anaesthetists, midwives, nurses, public health specialists and the Department of Health officials to get a consensus of what is feasible and best in South Africa with all its constraints, given the best evidence available and what we have in terms of the resources".

[41] Professor Gert Kirsten, based on his vast experience of more than 33 years as a specialist neonatologist, testified that the administration of double the dosage of Prostin E_2 tablets to that prescribed in the Guidelines constitutes a toxic dosage. His evidence in this regard was only disputed by the defendant's counsel that it was not recorded in the hospital records that the 4 Prostin tablets constituted an overdose. That is of course not sufficient.

[42] Professor Kirsten further testified that Prostin is a very potent labour inducing drug. A major concern when using Prostin for induction of labour is the possibility of excess uterine contractions leading to impaired utero-placental perfusion and foetal hypoxia. In response to my question, Prof Kirsten confirmed that the strength of Prostin E2 tablets was 0.5 mg during 2010 and that this was its standard strength at that time.

[43] Dr Emmanuel Opai-Tetteh, the defendant's obstetrician and gynaecologist agreed with Dr Sevenster that it was the correct decision to induce the plaintiff's labour. They also agreed that it was a high-risk induction because the foetus was small for gestational age, and the doctor and nursing staff were aware of this.

[44] In his evidence-in-chief Dr Opai-Tetteh stated that he would not say that the administration of 4 Prostin tablets was an overdose as it depended on the protocol and prescription which the doctor writes for the midwife to administer. He was not aware of the protocol of the Pholosong Hospital regarding the administration of Prostin E2 tablets.

[45] I agree with the submission by the plaintiff's counsel that the evidence of Dr Opai-Tetteh is not sufficient to refute the evidence of Dr Sevenster and Professor Kirsten, and their conclusion that the administration of 4 Prostin E2 tablets to the plaintiff constituted an overdose or too large a dosage. Although Dr Opai-Tetteh tried to argue that the Guidelines are not a protocol but just guidelines, they constitute the standards that were applicable at the time and should have been adhered to. There is no evidence placed before me that any other standard or protocol existed at the Pholosong Hospital at the relevant time. Furthermore, Dr Maseko was not called as a witness to justify his administration of 4 Prostin tablets in the face of the Guidelines that the administration of 4 Prostin tablets is double the prescribed dosage.

[46] In cross-examination, Dr Opai-Tetteh however agreed that when induction is commenced, especially a high-risk induction such as that of the plaintiff, it is prudent to start with a low dosage of induction agent (Prostin), and when careful monitoring does not indicate any problems with the foetus, then one can increase the dosage if necessary.

[47] I am therefore satisfied that based on the evidence presented to the court, the

administration of 4 (four) Prostin E_2 tablets to the plaintiff to induce labour constituted an overdose or a too large dosage.

Whether the Overdose of Prostin Tablets Probably Caused Hyperstimulation which Resulted in Hyper-contractions and Foetal Distress

[48] The plaintiff relied on the evidence of Dr Sevenster and Professor Kirsten to establish that the overdose or too large dosage of Prostin tablets probably caused hyperstimulation of the plaintiff's uterus, which probably resulted in hypertonic-contractions and foetal distress.

[49] Dr Sevenster testified that in his opinion, the double dosage of Prostin tablets probably caused the uterine hyperstimulation which probably resulted in hyper-contractions and foetal distress. Hypertonic uterine contractions are severe and refers to more than four (4) contractions in ten (10) minutes, or contractions lasting two minutes or more over a period of ten (10) minutes. He further testified that in the event of hyperstimulation and hypertonic-contractions there is not enough time for the foetus to re-oxygenate between contractions. When the uterus contracts there is virtually no blood flow from the mother to the foetus. Usually when the uterus completely relaxes, the blood flow between the mother and the foetus can resume. With hypertonic contractions, there is not sufficient time for the uterus to adequately relax so that the blood flow is restored.

[50] Uterine hyperstimulation thus impairs utero placental perfusion, giving rise to foetal hypoxia (reduced delivery of oxygen), and foetal acidosis and acidaemia (high hydrogen ion concentration in blood and tissues of the foetus). This results in loss of foetal heart variability and decelerations (lowered foetal heart rate) due to withdrawal of the vagal tone. If a double dosage of Prostin E2 tablets is used to induce labour as in the plaintiffs case, the risk is even higher and it would therefore be expected that uterine hyperstimulation would have resulted. Furthermore, a hyper-stimulated uterus usually presents as severely painful, which causes increased restlessness, as

was recorded by the nursing staff at 12h20 that the plaintiff was severely restless *(" restless+++ ").*

[51] During cross-examination of Dr Sevenster, the defendant's counsel disputed the probability of hyperstimulation and hypercontractions resulting from the overdose on the basis that it was not recorded in the hospital records and therefore amounted to speculation. Dr Sevenster pointed out that since no CTG or other monitoring of the foetal heart rate or the contractions of the plaintiff occurred after the induction of labour from 08h30 to O's birth at 12h45, it was not possible for the medical and nursing staff to have picked up or record the presence of hyperstimulation. On the probabilities, with a mother being induced with 4 Prostin E_2 tablets. and in the presence of a small for gestational age foetus, it is highly probable that hyperstimulation of the uterus and hypertonic contractions were present in this case.

[52] Professor Kirsten testified that the toxic dose of 4 Prostin tablets which were administered to the plaintiff was absorbed by the receptors in the uterus, and probably caused hyperstimulation and strong contractions (tachysystole)[17], that grew progressively stronger. This was not identified because the plaintiff was not monitored by the nursing staff.

[53] During cross-examination, it was put to Professor Kirsten that there is no recorded evidence of hyperstimulation, and that his evidence in this regard is based on speculation. Professor Kirsten refuted this statement by giving these reasons for his opinion:

53.1 The plaintiff's first CTG that was performed before her labour started (at 05h00 on 22 March 2010) was normal;

53.2 At birth, baby O was severely compromised and depressed; MRI indicated an insult consistent with a profound hypoxic-ischaemic injury, which fits in with hyperstimulation and hypertonic contractions which led to foetal distress, culminating in the acute profound hypoxic-ischaemic injury and brain

damage;

53.3 There was placental insufficiency present and baby O was asymmetrically growth restricted. This means that her head and brain developed normally during pregnancy, and the sub-optimal oxygen and nutrient supply from the insufficient placenta only affected her weight and her length;

53.4 O's normal heart rate on CTG before the induction of labour, and again 30 minutes after labour, (at 08h30 when the last CTG was performed) is indicative that this asymmetrical growth restricted foetus was metabolically stable before labour started with the administration of the Prostin tablets.

53.5 If one considers the above-mentioned facts, the reasonable inference is that the Prostin overdose probably caused hyperstimulation and hypertonic contractions, which resulted in foetal distress and eventually the brain damage which baby O suffered. This is not mere speculation but based on all the available facts.

[54] While the plaintiffs experts gave reasoned opinions regarding the probable presence of hyperstimulation and hypertonic contractions during their testimony, the defendant's only response was that their opinions amount to speculation. It is important to take cognizance of the fact that the only reason why there is no direct evidence of hyperstimulation, and the court having to draw an inference that it was probably present, is the lack of monitoring from 08h30 until 12h45 after the induction of the plaintiffs labour. In my view, it is opportunistic of the defendant to submit that there is no evidence of hyperstimulation and to postulate that the presence of hyperstimulation is speculation when the defendant should have provided such evidence.

The Negligence of the Medical and Nursing Staff

[55] Having established that the medical and nursing staff administered an

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[17] Tachysystole is a condition of excessively frequent uterine contractions.

overdose of Prostin E2 tablet s, and that this probably resulted in hyperstimulation, hypertonic contractions and foetal distress, which were not detected and acted upon, I agree with the plaintiffs submissions and that the plaintiff succeeded in proving the two main grounds of negligence:

55.1 that it was negligent of the medical and nursing staff to induce the plaintiffs labour with double the Prostin dosage prescribed by the Guidelines for induction of labour; and

55.2 that failure of the medical and nursing staff to continuously monitor the induction of the plaintiffs labour by means of a CTG or other device such as a doptone or fetoscope from 08h30 to 12h45 was negligent.

[56] The neonatologists Professor Kirsten and Professor Cooper agreed in their joint minutes that:

56.1 baby O had asymmetrical growth restriction;

56.2 a foetus with asymmetrical intra-uterine growth restriction tolerates labour poorly due to reduced energy stores and oxygen supply during uterine contractions;

56.3 these infants are prone to developing foetal distress during labour, especially during augmented uterine contractions when labour is induced[18].

[57] Both Dr Sevenster and Professor Kirsten testified that the failure of the medical and nursing staff to monitor the plaintiff's contractions and the foetal heart rate during her induction until delivery, was a serious deviation from the standard which one would expect from a reasonable medical practitioner and reasonable nursing midwives in the circumstances of this ca e. Early recognition of excessive contractions and foetal distress would have enabled the medical and nursing staff to take appropriate steps to stop the excessive hypertonic contractions and the foetal distress relatively quickly, and that would have been sufficient time to deliver the baby either by means of assisted delivery (vacuum extraction or forceps), or

[18] Bundle 3 Index 7, Joint Minutes Prof Kirsten and Prof Cooper pages 2-3 paras 5, 6 and 7.

caesarean section, depending on the favourability of the plaintiff's cervix. This evidence was not disputed. Even Dr Opai-Tetteh testified that in the event that the plaintiff was too restless for CTG monitoring, such monitoring should have been done by a fetoscope or doptone, and would have been effective.

[58] Both Dr Sevenster and Dr Opai-Tetteh agreed that the plaintiff and her foetus were subjected to substandard care during labour, due to the fact that labour was not properly monitored, and concluded that the monitoring of the labour process of the plaintiff was substandard[19].

[59] Although Dr Opai-Tetteh conceded during cross-examination that the failure by the medical and nursing staff to monitor the plaintiff's contractions and the foetal heart rate deviated from the standard of care, which he would expect from reasonable medical practitioners and nursing staff in the circumstances, he denied that it was negligent.

[60] In my view, a medical doctor and midwives in their position would undoubtedly have taken steps to prevent such harm; firstly, by not inducing labour with an overdose of Prostin tablets, and secondly, by monitoring the plaintiffs contractions and by taking reasonable measures to stop excessive contractions and foetal distress, and/or timeously delivering the baby before the foreseeable harm could materialize. I am therefore satisfied that the plaintiff has. on a balance of probabilities proven the two grounds of negligence.

Causation

[61] The next question I have to determine is whether there was a causal link between the negligent conduct of the medical and nursing staff and baby O's brain damage, which left her with mixed cerebral palsy, microcephaly and profound Intellectual disability.

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[19] Bundle 3, In d e x 7, Joint minutes of the Obstetricians, page 25 paras 1.13, 1.15 and 1.20.

[62] The issue of causation requires the determination of two central issues:

62.1 whether the acute profound hypoxic-ischaemic injury which baby O suffered resulting in permanent brain damage was probably caused by hyperstimulation of the plaintiffs uterus, and resultant hypertonic contractions and foetal distress due to the overdose of Prostin in the first instance, and secondly, in addition thereto, by the failure of the medical and nursing staff to monitor the probable excessive contractions of the plaintiff and the foetus as well as the distress of her foetus; and

62.2 whether, but for the negligent conduct of the medical and nursing staff, the acute profound hypoxic-ischaemic injury which baby O suffered would probably have been prevented.

The Probable Cause of the Acute Profound Hypoxic Injury and Resultant Brain Injury

[63] There is no dispute that the MRI findings of O's brain is consistent with an acute profound hypoxic-ischaemic event. The neuro-radiologists, Dr Kamolane for the defendant and Professor Lotz for the plaintiff, agreed that the injury which baby O suffered is hypoxic-.ischaemic in nature, and exhibits a pattern of acute profound injury, the result of a severe incident in the peri-natal period.

[64] Both Dr Kamolane and Professor Lotz agreed that:

64.1 the findings of the MRI study suggest that genetic disorders as the cause of the child's brain damage are unlikely;

64.2 the MRI findings suggest that inflammatory or infective causes are unlikely as causes of the child's brain damage; and

64.3 a review of the clinical and obstetrical records by specialists in the field of neonatology and obstetrics Is essential in determining the cause and probable timing of this hypoxic" ischaemic injury.[20]

[20] Bundle 3, Index 7, Joint minutes by neuro-radiologists, page 16 paras 3-7

[65] Dr Thabo Kamolane in his evidence-in-chief testified that the acute-profound hypoxic-ischaemic insult to baby O's brain could have occurred before, during or post-delivery within the perinatal period, and that it occurred in a term brain (the baby was at term gestation). He further testified that it is impossible to determine on the MRI when the insult occurred within the perinatal period, but what is clear from the MRI scan is that the insult occurred in a term brain, which means a brain of a least 37 weeks gestation.

[66] During cross-examination, Dr Kamolane confirmed that there is no white matter injury apparent on the MRI scan. Therefore, it is most probable that an acute profound injury occurred. This indicates a severe incident of total cessation of blood and oxygen to the brain.

[67] In re-examination, the defendant 's counsel put it to Or Kamolane that there are other causes of hypoxia which could have resulted in reduced blood flow and consequent brain damage. Dr Kamolane responded that in this case, there is overwhelming evidence of an acute profound hypoxic-.ischaemic injury which necessarily implies decreased blood flow and oxygen to the foetal brain. He found that the Peri Rolandic Cortex was in fact affected in this case, which is futher confirmation that the injury was an acute profound hypoxic-ischaemic injury.

[68] Professor Regan Solomons a paediatric neurologist called by the plaintiff gave evidence about the probable cause of the acute profound hypoxic-ischaemic insult which baby O suffered, by giving the following national scientific reasons regarding the nature, mechanism and the timing of O's brain injury:

68.1 Baby O suffered moderate neonatal encephalopathy. (Both Professor Solomons and Dr D Pearce for the defendant agreed)[21].

68.2 He agreed with the definition of neonatal encephalopathy by the

[21] Bundle 3, Index 7, Joint minutes of the Paediatric Neurologists, page 11, para 6.

American Cellege of Obstetricians and Gynaecologists: *"A Clinically-defined Syndrome of disturbed neurologic function in the earliest days of life in an infant born at or beyond 35 weeks of gestation, manifested by* a *subnormal level of consciousness or seizures, and often accompanied by difficulty with initiating and maintaining respiration and depression of tone and reflexes".*

68.3 O fits the criteria for neonatal encephalopathy. According to the hospital records, and the 5 and 10/minute APGAR scores, O had difficulty initiating respiration, she was hypotonic and had frequent seizures.

68.4 Professor Solomons excluded all other possible causes for a neonatal encephalopathy, and concluded that O's neonatal encephalopathy is hypoxic-ischaemic encephalopathy (HIE)[22] due to a peri-natal hypoxia! insult. HIE may cause death in the new-born period or what is later recognised as developmental delay, mental retardation or cerebral palsy.

[69] Professor Solomons quoted and agreed with the learned author VOLPE[23] regarding the three features or criteria to make the diagnosis of the intrapartum asphyxia insult as being the likely cause of a neonatal brain injury. These features are:

69.1 evidence of a foetal distress (eg. foetal heart rate abnormalities). In O's case, it is not possible to determine whether there was foetal distress as the foetal heart rate was not monitored after the induction of labour. He however agreed with Professor Kirsten that the very low APGAR scores are indicative of intrapartum foetal distress;

69.2 depression at birth necessitating resuscitation. It was noted in the hospital records that O was resuscitated; and

69.3 an overt neonatal neurological syndrome during the first hours and days of life. According to the hospital records, O was dull, hypotonic (floppy) and had seizures.

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[22] Hypoxic-ischaemic encephalopathy is defined as damage to cells in the central nervous system (the brain and spinal cord) from inadequate oxygen.

[23] Volpe is the World Authority on Paediatric Neurology

[70] Based on the available records and the criteria of VOLPE, Professor Solomons concluded that the acute profound hypoxic-ischaemic encephalopathy injury to O's brain was probably caused by an intrapartum hypoxic ischaemic insult, which is defined as an extreme intrauterine event that resulted in acute profound asphyxia. He further testified that in the absence of close monitoring of the labour process, the cause of such intrapartum hypoxic insult is not identified or recorded.

[71] The aforesaid evidence of Professor Solomons was not disputed. The defendant's counsel questioned Professor Solomons' timing of the hypoxic injury to the intrapartum period in light of the placental insufficiency and asymmetric growth restriction that was present. Professor Solomons conclusively refuted the postulation by the defendant's counsel that baby O's asymmetrical intra-uterine growth restriction possibly caused brain injury. He explained that in the case of IUGR the baby is usually born premature, and if there is a hypoxic injury, it would occur at an earlier stage during gestation (around thirty weeks.) O was not born premature, had no signs of peri-ventricular leukomalacia, and have no signs of spastic diplegia but rather a mixed cerebral palsy pattern. In the unlikely event of the asymmetrical IUGR causing hypoxia in a term foetus, the auto-regulation mechanism of the brain would preserve the deep grey matter of the brain, and the pattern of injury on the MRI scan would be indicative of damage to the white matter of the brain, which is not the case with O, whose grey matter has been damaged.

[72] Professor Solomons has no doubt for all the reasons stated above, that the cause of O's acute profound hypoxic-ischemia was an extreme sentinel event which occurred in the intrapartum period. The only plausible extreme event which probably caused the insult was the hyperstimulation, which probably occurred as testified to by Dr Sevenster and Professor Kirsten.

[73] Professor Kirsten testified that the most probable explanation in this case is

that baby O suffered an intrapartum acute profound hypoxia! insult due to the Prostin induced tachysystole (hypercontractions), which caused a severe mixed metabolic and respiratory acidosis. This resulted in a foetal bradycardia during the last (30)thirty to (40)forty minutes before O's birth, which reduced her cardiac input, caused hypotension, (low blood pressure), reduced brain perfusion, and eventually an acute profound (near-total) hypoxia and cerebral palsy.

[74] As a result of the failure of the medical and nursing staff to monitor the plaintiff's contractions md the foetal heart rate, the probable outset of hyperstimulation, tachysystole and foetal heart rate decelerations were not detected and therefore the nursing and medical staff did not intervene.

[75] Professor Kirsten further testified that an identified cause of an acute profound hypoxic injury is referred to as a sentinel event. In this case, the toxic dose of Prostin that was administered to the plaintiff whilst she was giving birth to an asymmetrical growth restricted baby, who did not have the reserves to cope with the hyperstimulation of the uterus and consequent hypertonic contractions, together with total lack of monitoring of the induction of labour constituted such a sentinel event.

[76] The above evidence by Professor Kirsten regarding the mechanism and nature of the hypoxic injury which O suffered, was not disputed in cross-examination.

The Defendant's Case of Possible Other Causes for the Brain Injury

[77] During the cross-examination of Dr Sevenster, Professor Kirsten and Professor Solomons, the defendant's counsel raised with them the possibility that there were other factors which may have compromised O's and the plaintiff's pregnancy, and may have been the cause of the hypoxic-ischaemic injury to O's brain. The defendant's counsel raised the hygienic problems with a reduced immune system since the plaintiff presented with genital warts at her first ante-natal visit, and her treatment having been deferred for her pregnancy, her positive HIV status, a

recording of urinary tract infection, and O's asymmetrical growth restriction caused by placental deficiency.

[78] It must be noted that only one of the defendant's witnesses, Or Opai-Tetteh touched upon these factors. The defendant's experts who raised the possibility (and not probability) in their reports ie Dr Duma and Professor Cooper were not called to substantiate this defence of the defendant.

[79] The crux of Dr Opai-Tetteh's testimony was that it is a possibility that asymmetrical IUGR, placental insufficiency, genital warts, the plaintiff's HIV status and the urinary tract infection, may have played a role in O's eventual brain damage. There is however no evidence before me that any of these factors, either in themselves or in combination was a probable cause of O's brain damage.

[80] In my view, the evidence of Dr Opai-Tetteh was insufficient to upset the probable cause of O's brain damage as testified by Dr Sevenster, Professor Kirsten and Professor Solomons. Furthermore, Dr Opai-Tetteh conceded in his evidence that the results of the blood tests performed on O indicated that infections could be excluded as a cause for the hypoxic injury. He further agree that the effect of the asymmetrical IURG and placental insufficiency made O more vulnerable than an uncompromised foetus to handle the pressures of the birth process.

[81] In the absence of any evidence regarding the factors raised by the defendant being probably linked to O's brain damage, and merely raising these factors as possibilities, amounts to no more than speculation. With regard to HIV, the undisputed evidence of Professor Solomons was that although the plaintiff was HIV positive, O tested negative, and that it is very rare to transfer HIV through the placenta. If it is so transferred , multi-organ injury usually occurs, and not only an injury of the brain as is the case of O. Based on the undisputed evidence of Professor Solomons, HIV can be excluded as an ante-natal factor that caused hypoxia and brain damage to O.

[82] Regarding the genital warts raised by the defendant as a possible contributing cause for O's brain damage, either on its own or in conjunction with the other factors, Professor Solomon's undisputed evidence was that in his experience he has never encountered genital warts as being associated with brain damage in the form of cerebral palsy. He referred the court to and agreed with the peer review article, *(Streja et al 2013, Congenital Cerebral Palsy and Pre-Natal Exposure to Self-Reported Maternal Infections, Fever and Smoking---American Journal of Obstetrics and Gynaecology).* The authors of this article evaluated children with cerebral palsy and the study excluded association of genital warts with cerebral palsy.

[83] Professor Solomons stated that if the warts caused infection that crossed the placenta, there would be multi-organ damage, chorioamnionitis and infection of the placenta and amniotic fluid. The baby would be born prematurely, which was not the case with O.

[84] Kirsten in his evidence corroborated the evidence of Professor Solomons, and testified that he has never in all his years of practice in State Hospitals, encountered vaginal warts as being associated with a compromised immune system. In his opinion, the genital warts in the case of the plaintiff played no role whatsoever in the brain damage which baby O suffered. There is also no evidence that the plaintiffs positive HIV status compromised the immune system of the foetus during pregnancy, and the urinary tract infection had no impact in this case.

Whether the Acute Profound Hypoxic-Ischaemic Injury and Permanent Brain Damage of baby O was probably preventable

[85] Dr Sevenster testified that the Prostin tablets would probably have been absorbed by 09h00, and hyperstimulation would probably have been detected on CTG soon after 09h30 if appropriate monitoring occurred. Soon after the hypertonic contractions started, there would have been a change in the foetal heart rate, which would have been evident on CTG. As soon as the excessive contractions started

due to hyperstimulation, these would also have been detectable on CTG monitoring or by means of a doptone or fetoscope.

[86] Professor Kirsten agreed with Dr Sevenster that as soon as signs of the hyperstimulation and hypertonic contractions were detected, the nurse should have called the doctor. The nursing staff and/or doctor should have immediately removed the remaining tablets from the plaintiff's vagina and administered Tocolysis (medication to suppress contractions). The probability of stopping hypertonic contractions with this medication was excellent. The midwives are trained to perform intrauterine resuscitation, and they should have turned the plaintiff on her left side and given her oxygen. In the unlikely event that the Tocolysis did not have an effect, the baby should have been delivered immediately by means of forceps or vacuum extraction in the event that all the criteria for safe delivery were met. If the plaintiff's cervix was not favourable for such a delivery, a caesarean section could have been performed. A caesarean section would have taken longer, reasonably within an hour in a State hospital. This evidence was not disputed by the defendant.

[87] On the undisputed evidence of Dr Sevenster and Professor Kirsten, I am of the opinion that the onset of hyperstimulation of the plaintiff's uterus, hypertonic contractions and foetal distress, and later brain damage which baby O suffered was foreseeable and preventable in this case.

Resuscitation of Baby O

[88] The plaintiff relies on a further ground of negligence which contributed to or compounded the acute profound hypoxic-ischaemic brain injury which baby O suffered, in that the medical and nursing staff were negligent in their resuscitation of baby O after her birth.

[89] The undisputed evidence of Professor Kirsten was that O's hypoxic brain damage due to hypoxic-ischaemic insult was compounded by a further postpartum

hypoxial insult due to suboptimal resuscitation[24]. He gave the following reasons for his opinion, which were not disputed during cross-examination;

89.1 a paediatric doctor was not proactively called to the labour ward to effectively resuscitate O at birth;

89.2 the midwife continued with inappropriate resuscitation consisting of face mask bagging despite there being very little improvement. This continued for at least 10 minutes before a doctor was summoned to take charge of the resuscitation;

89.3 the doctor also inappropriately continued with face mask ventilation for at least another 2 minutes before O was intubated;

89.4 spontaneous, though irregular respiration was only established another 5 minutes after intubation, ie 17 to 20 minutes after birth;

89.5 the fact that baby O's oxygen saturation was only 90% on 100% oxygen (normal >92% in room air, i.e. 21% oxygen, at 35 minutes of age), that she was still pale and had deep irregular respirations indicate that she was severely acidotic and hypotensive, and required the administration of inotropes;

89.6 the prolonged hypotension would probably have caused reduced brain perfusion and oxygenation resulting in further hypoxic-ischaemic brain damage;

89.7 her blood pressure was not measured and she did not receive any inotropes;

89.8 a blood gas should have been obtained after the resuscitation to guide further management. Professor Kirsten testified that in his opinion the blood gas would probably have confirmed hypoxia, a severe metabolic acidosis and a compensatory respiratory alkalosis.

[90] Although Professor Cooper, neonatologist for the defendant disagreed in the joint minutes with Professor Kirsten regarding the negligent resuscitation, he was not

[24] Bundle 2.2, Addendum report pages 278-280.

called to testify and his opinion on the aspects in respect of which he disagrees with Professor Kirsten is not before Court. I have also noted that the basis for Professor Cooper's disagreement that baby O was not intubated and properly ventilated is that *' the relevant pages that would contain the details of resuscitation were missing, and thus this conclusion cannot be drawn'*[25]*.* The evidence of Professor Kirsten in this regard therefore stands unchallenged.

[91] I am satisfied that the plaintiff has established on a balance of probabilities that O's hypoxic brain damage due to the intrapartum insult to her brain was compounded by a postpartum hypoxic-ischaemic insult due to suboptimal resuscitation and poor post-resuscitation assessment and treatment.

[92] On the totality of the evidence, the plaintiff has succeeded in proving on a balance of probabilities that:

92.1 there was negligence on the part of the nursing and medical staff of the defendant, their management, monitoring and assessment of the induction of her labour and the delivery of her baby O, as well as in their resuscitation of baby O after her birth; and

92.2 the negligence of the nursing and medical staff of the defendant probably caused or contributed to the intrapartum acute profound hypoxic-ischaemic insult to O's brain which she suffered before birth, and was probably compounded by negligent resuscitation, which resulted in O being left with permanent severe brain damage manifesting as mixed cerebral palsy, microcephaly and profound intellectual disability.

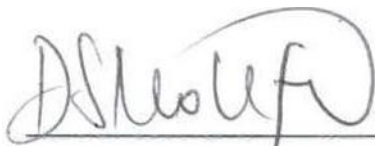
[93] I thus make the following order:

1. The defendant is liable for payment of 100% of the proven or agreed damages the plaintiffs minor daughter, O, suffered as a result of the monitoring, assessment and management of the plaintiff's labour and delivery

[25] Bundle 3, Index 7, Joint minutes of Neonatologist, page 6

of O on 22 March 2010, as well as the neonatal management of O, by the nursing and medical staff of the defendant at the Pholosong Hospital, resulting in O suffering severe brain damage manifesting as inter alia mixed cerebral palsy and spastic quadriparesis, visual impairment and global developmental delay;

2. The defendant is liable for the payment of the plaintiff's taxed or agreed costs of suit on the High Court Scale; and
3. The draft order annexed hereto and marked "A" is hereby made an order of Court.



DS MOLEFE J

JUDGE OF THE HIGH COURT

APPEARANCES

Counsels on behalf of Plaintiff	:	Adv. J H StÖh SC and Adv. F Pauer
Instructed by	:	O Joubert Attorneys
Counsel on behalf of Defendants	:	Adv. Shakoane SC and Adv. C Lithole
Instructed by	:	The State Attorneys, Pretoria
Dates of hearing	:	9 March 2020 - 20 March 2020
Date of Judgment	:	22 May 2020

ANNEXURE "A"

IN THE HIGH COURT OF SOUTH AFRICA
(GAUTENG DIVISION, PRETORIA)

CASE NUMBER: **83019/2016**

In the matter between:

M M obo
O M Plaintiff

and

THE MEMBER OF THE EXECUTIVE COUNCIL FOR HEALTH
OF THE GAUTENG PROVINCIAL GOVERNMENT Defendant

COURT ORDER

It is ordered that:

1. the Defendant is liable for payment of **100%** of the proven or agreed damages the Plaintiff's minor daughter, **O,** suffered as a result of the monitoring, assessment and management of the plaintiff's labour and the delivery of **O** on 22 March 2010, as well as the neonatal management of **O,** by the nursing and medical staff of the Defendant at the **PHOLOSONG HOSPITAL,** resulting in **O,** suffering severe brain damage manifesting as *inter alia* mixed cerebral palsy end spastic quadriparesis, visual impairment and global developmental delay.
2. the Defendant is liable for the payment of the Plaintiff's taxed or agreed costs

of suit on the High Court scale, such costs to include:

2.1 the costs occasioned by the employment of 2 {two) counsel by the Plaintiff, including their cost of preparation for, and attendance of all pre-trial conferences that were held and attended by them, as well as the drafting and settling of the pre .. trial agendas and minutes;

2.2 the Plaintiff's costs of obtaining the medico-legal reports and addendum reports of the Plaintiff's experts relating to the issue of liability, including the cost of counsel of drafting the Plaintiff's expert summaries in respect of the issue of liability of whom notice has been given in terms of Rule 36(9)(a) and (b);

2.3 the cost of preparation, qualifying and reservation fees, and fees for testifying, (if applicable), in respect of the liability trial of 9 to 18 March 2020 of the experts of the Plaintiff In respect of the issue of liability of whom notice has been given In terms of Rule 36(9) (a) and {b), including the cost of consultations by the Plaintiff's legal representatives with these experts, and the costs of these experts in preparing for and holding Joint meetings with their respective counterparts, and preparing joint minutes;

2.4 the costs of the MRI investigation of **O'S** brain performed by Koen Radiologists for purposes of the report of Prof Lotz, expert radiologist;

2.5 the cost of preparing and updating 8 (eight) sets of trial bundles;

2.6 the costs and expenses of accommodation and of transporting the Plaintiff, the minor child, **O,** and d family member or helper, in attending all medico-legal examinations and consultations by the Plaintiff's and the Defendant's experts, (where applicable), for purposes of preparing their reports for the trial relating to the issue of liability, subject to the discretion of the Taxing Master;

2.7 the costs stipulated above shall be paid into the trust account of the Plaintiff's attorney, the details which are:

Olof Joubert Trust

Account Number […]

First national Bank

Brooklyn Branch, Branch code: 22-20-26

Reference: **O JOUBERT/JM1738**

3. the costs of the trial of 11 to 18 March 2019, so postponed on 18 March 2019, is declared to be cost in the cause on the High Court scale as taxed or agreed between the parties, which cost shall include:
    3.1 the costs occasioned by the employment of 2 (two) counsel, including their trial preparation fees and their costs for the consultation/s with the plaintiff's expert witnesses of whom notice was given in terms of Rule 36(9)(b); and their drafting of and preparation for the application regarding the defendant's entitlement to the medico-legal reports of Dr Harris and Prof Solomons that was heard on 18 March 2019;
    3.2 the preparation, qualifying and reservation fees (if applicable) of the Plaintiff ' s experts of whom notice was given in terms of Rule 36(9)(b):
    3.3 the Court attendance fees of **OR C SEVENSTER** and **PROF G KIRSTEN** for 11 March 2019;
4. The following provisions shall apply regarding the determination and payment of the plaintiff' s abovementioned taxed costs:
    4.1 the plaintiff's attorney shall timeously serve the notice of taxation on the defendant's attorneys of record;
    4.2 the plaintiff's attorney shall allow the defendant 30 (THIRTY) calendar days to make payment of the taxed costs from date of settlement or taxation thereof;
    4.3 should payment of the plaintiff's taxed or agreed costs not be effected timeously , the plaintiff will be entitled to recover interest at the rate of 10.25%, calculated from the 31st calendar day, after the date of the

Taxing Master's *allocotur,* or after the date of settlement of costs, up to the date of final payment.

5. The matter is postponed *sine die* for the determination of the issue of the *quantum* of the plaintiff's minor child, **O'S** claim.

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BY ORDER OF COURT