REPUBLIC OF SOUTH AFRICA

IN THE HIGH COURT OF SOUTH AFRICA

GAUTENG LOCAL DIVISION, JOHANNESBURG

Case No: 2011/41603

(1)	REPORTABLE: YES / NO
(2)	OF INTEREST TO OTHER JUDGES: YES/NO
(3)	REVISED.
DATE	SIGNATURE

In the matter between:

M: B obo

M: K **PLAINTIFF**

And

THE MEMBER OF THE EXECUTIVE COUNCILFOR

HEALTH AND SOCIAL DEVELOPMENT OF THE

GAUTENG PROVINCIAL DEPARTMENT **DEFENDANT**

***Summary**: Claim for damages – arising out of injuries sustained by foetus at birth - negligence of employees of Defendant – test for negligence discussed – causation – absence of hospital records – Defendant negligent and found liable.*

JUDGEMENT

CELE AJ

Heard: 18 June 2018
Delivered: 01 October 2018

Introduction

[1] The plaintiff sues herein in her personal and representative capacity, as the mother and natural guardian of her minor child, K M (the child), who was born on […] January 2006 at the Chris Hani Baragwanath Hospital (The Hospital). The claim is based upon the alleged negligence of the medical and/or nursing staff of the hospital, resulting in the child suffering from cerebral palsy. By agreement between the parties Court ordered that the issues of liability and quantum be separated and the determination of quantum of damages was postponed *sine die*. The Defendant opposed the claim by denying that any of its staff acted negligently.

Factual Background

[2] A pregnant Plaintiff attended antenatal clinic for at least four times. There was no history of any deficiencies of the foetus. A private doctor she attended in August 2005 told her that she was carrying a baby girl, which was to be her first born. On […] January 2006 she woke up early in the morning, went to the bathroom, discovered that she was bleeding (spotting) and she reported this to her mother with whom she lived. According to her it was still early morning around 02h00 because it was still dark. She never checked the exact time. Transport was arranged by her mother to ferry her to the Hospital. Her mother, Ms Teboho Margaret Mbanda thought it was about 04h50 when the Plaintiff left home for the Hospital. The Plaintiff

proceeded to the labour department where she registered her particulars and she was offered a hospital night dress to change into. When asked what was wrong with her she reported that she was bleeding. She was made to sit on a bench and was offered porridge to eat.

[3] Urine testing was conducted on her by a nurse and she was taken into a cubicle with a bed which she was made to lie on. The Plaintiff said that a brown belt, referring to a cardiotocograph monitor otherwise often referred to as the CTG monitor, was connected on her. The CTG is used for monitoring the expectant mother and the foetal heartrate before, during and after contractions. Any deceleration picked up by the CTG monitor is a symptom of foetal destress and it must immediately be acted upon to alleviate the distress. The CTG depicts a graphic pattern and produces an audible sound which requires interpretation of patterns associated with foetal distress and it informs the decision to intervene and accelerate birth.

[4] By then the Plaintiff had spent more than an hour since her arrival at the Hospital. She was then moved to another cubicle, taking the monitor with her. A male Doctor attended to her by conducting some tests on her. He ordered a nurse to get an intravenous infusion (a drip) which was connected to her. She estimated the time to be 11h00 by then, though she had no watch. While the Doctor continued to check on her he spoke to another person in attendance about a Caesarean section but she could not hear what exactly was said. This happened at about 11h10. Thus far, the Plaintiff had not had any labour pains. She was then given medication to induce labour. Some 30 minutes later a further dosage was given to her to further induce labour. Sometime later she was moved to the delivery room where she was counselled about the delivery process and was told what to do at various stages. She thought it was then about 14h30.

[5] A Doctor attended to her and he broke her water as it had not yet broken and then left. Still she had no labour pains and the baby was not coming. The nurse repeatedly told her to push. Time passed and the nurse called for another Doctor who inserted his hands into her and appeared to feel something. The baby then presented herself. She had a nuchal cord around her neck. The Doctor called for a scissor which was brought by a nurse. He clamped the nuchal cord, cut it and he

pulled the baby out. Birth was accordingly by vertex delivery at around 15h30 on […] January 2006.

[6] The baby was then taken away from the mother and placed into a head box. Most of the records pertaining to the labour and birth periods of the child were lost at the hands of the Hospital staff. From the remaining birth records the newly born baby or neonate was born with normal anthropometrical measurements but did not immediately breath had a reduced muscle tone and a blue coloration requiring immediate resuscitation, hence the immediate placement into the head box. Saturation of the neonate then improved to normal, at 93%.

[7] It was only on the next day that the Plaintiff saw her newly born baby who was still in the cubicle in the neonatal unit. On 30 January 2006 the baby were still in Hospital as she was receiving treatment. The neonatal discharge summary notes of this baby contained the following information about the baby:-

1. APGAR scores were recorded at 4/10 at one minute of life and 7/10 at 5 minutes of life;
2. Birth weight was recorded at 3000g and the discharge weight (6 days later) at 3280g;
3. Gestational age was recorded as term;
4. Findings in respect of a cranial ultrasound were recorded as a bright brain, good pulsations and no bleeds;
5. Diagnosis of perinatal asphyxia;
6. Cord around the neck;
7. A PH was recorded as 7.23 and base excess recorded as - 8.2.

[8] When the baby was discharged, the Plaintiff was not told anything about the medical condition of her baby. She followed up the discharge with a number of clinical visits. The birth of her child was registered at Home Affairs offices and K was the name given to the baby. The Plaintiff never requested for an abridged birth certificate.

[9] The Plaintiff had to consult various doctors as time went on and it appeared something was wrong with her baby. The baby was diagnosed with cerebral pulse. One such Doctor consulted was Dr Basson, an obstetrician and gynaecologist. The Plaintiff could not remember the exact dates of such visitations. When K was 8 years 1 month a number of brain scans were taken of her and presented to Professor Jan Willem Lotz, a Specialist Radiologist to analyse and interpret. The Plaintiff had a one on one consultation with Dr Basson and later also met Dr Pearce, a paediatric neurologist on 23 June 2016. The case of K was then referred by the parties to their various experts who compiled then filed their reports. Some of the experts exchanged their notes and formulated joined minutes in respect of the aspects they agreed on. Where there were points of departure these were noted by the experts.

[10] As the parties were preparing this matter for trial, they attempted to narrow the issues in dispute through three pre-trial minutes. Some of the issues settled in the pre-trial minutes are that the Defendant admitted that:-

1. At all relevant times hereto and particularly during on or about January 2006:
    1. Defendant was and still is the responsible person in respect of any and all contractual and delictual liability of the Department of Health and Social Development of the Gauteng Provincial Government;
    2. The Chris Hani Baragwanath Hospital was and still is the a hospital which falls under the authority of and is controlled and operated by the Department of Health and Social Development of the Gauteng Province;
2. Defendant by reason of:
    1. The existence of the public Hospital and the fact that the Defendant, through his Department of Health and Social Development, held out to the public and particularly the Plaintiff, that the aforesaid Hospital was a facility where reasonable medical care, treatment and advise is rendered and/or

2. The Plaintiff's admission as a patient at the said Hospital and the undertaking by such Hospital to render medical care and treatment to her and her unborn child and/or
3. The provision of section 9 (the right to equality), section 10 (the right to dignity), section 11 (the right to life) and section 27 (the right to health and health care services) of the Constitution of the Republic of South Africa, Act1996 was under a legal duty to ensure the rendering of medical care, treatment and advise to Plaintiff with such skill, care and diligence as could reasonably be expected of medical practitioners and nursing staff in similar circumstances, obliging the Defendant to ensure proper, sufficient and reasonable health services are provided to members of the public, particularly those who were obliged to make use of such services of a public hospital;

3. The aforesaid legal duty of care extended to K Zoe (then as an unborn child)
4. Any and all medical practitioners and nursing staff (whose identities are to the Plaintiff unknown) who were involved or who were obliged to become involved in the admission to and the rendering of medical services and the examination, monitoring and/or treatment of the Plaintiff at the aforesaid Hospital were either permanent or temporary employees of Defendant who acted within the course and scope of their duties as permanent or temporary employees.
5. The main problems of birth were, inter alia, identified and recorded as perinatal asphyxia with a pH of 7.23 and a BE of 8.2. In addition it was noted that the baby was delivered with the cord around her neck.
6. Cranial ultra sounds of the child confirmed a bright brain, good pulsations and no bleeds.
7. The MRI distribution of changes when K was 8 years 1 month old, namely deffuse bilateral periventricular and subcortical cystic and non-cystic leukomalacia and bilateral T2 hyperintensity-pulvinar, lateral thalamic and PLIC was in keeping with hypoxic ischemic event/s, mixed pattern predominately of a partial prolonged type.

8. The findings of the MRI studies suggest that genetic disorder as a cause of the child's brain damage was unlikely;
9. The child's cerebral palsy does not have a genetic/syndromic origin as per Prof Christian's addendum report;
10. There was no evidence from the family history, clinical evaluation, MRI or cranial ultrasound suggestive of a genetic, hereditary or metabolic cause for K's medical condition;
11. According to Prof Christianson's addendum report, the child had the following:
    11.1 Mixed cerebral palsy – dyskinetic and spactic quadriplegia;
    11.2 Microcephatic;
    11.3 Epilepsy not controlled;
    11.4 Contractures.
12 Hyperekplexia – this is a clinical feature associated with cerebral palsy, as such it has no significance in this case – as per Prof Christianson's addendum report.
13. The brain injury was suffered during the intrapartum period as per Dr Magashoa's summary in medico-legal report. (Dr Mogashoa adds that the nuchal cord contributed to the intrapartum hypoxia).

Evidence

[11] The general onus of proof rested on the Plaintiff to satisfy Court at least on a balance of probabilities that her version was the truth. Six witnesses were called to testify at the instance of the Plaintiff. In its general nature the evidence of the Plaintiff and her mother is common cause and forms part of the factual background, save to note that such evidence is contradictory when it comes to the time when the Plaintiff left home for Hospital on […] January 2006. On the basis of documentary and *viva voce* evidence the other four witnesses are found to be experts in their related fields of training and practice over many years. A summary of the evidence of these four witnesses shall now be outlined.

Plaintiff's version

Professor Jan Willem Lotz [Specialist Radiologist]

[12] He made a step by step presentation of sixteen slides[1] through which he showed in general and in specific terms how brain injury occurs when a foetus is exposed to asphyxia at the commencement of and during delivery of a baby. This is determined through a process of Magnetic Resonance Imaging (MRI) which allows an examination of a nucleus of an atom of the tissue be it of a skin or muscle. This reveals a proton which has an electronic charge as it spins like the earth to create a magnetic field. It is the examination of this field, by scanning through different planes, such as the axio, corona and the surgical or midline planes, which produces data that is put together to reveal any injury or assault which might have occurred to the brain tissue.

[13] As a result of the evolution of the brain over millions of years there are three distinguishable parts of the brain, the Reptilian, Mammalian and Human brain. This is in slide 1 of exhibit 2. The Reptilian core was developed first and it has all vital centres which keep human beings, as mammals, alive. For instance the heart bit, breathing and swallowing processes are controlled in the Reptilian brain. At birth the Reptilian brain plays a vital role as it supports life and enables the ejection of the child alive. The Human brain on the other side is asleep at birth. The delivery of a live child from the mother requires a pressure wave of blood supply to the brain of the baby. Where there is much less oxygen in the blood noticed by too few rings this state is referred to as **hypoxia**. If there is a drop in the blood pressure this is referred to as **ischemia**. The brain injury caused by much less oxygen and a simultaneous significant drop in the blood pressure is then called **hypoxic ischemic assault or insult.**

[14] If there are problems such as mild asphyxia just when the child is ready to be born (**partum period**), blood supply to the Human brain will be shunted away first so as to preserve blood supply to the Reptilian brain. Where the problem is soon resolved, a recovery process takes place with blood going back to the Human brain. The longer the injury or insult takes place the more there are chances that the

[1] See exhibit 2 with 16 slides.

Human brain will suffer irreparable damage. The effect of a mild attack is that, while the Human brain is compromised, the Reptilian brain is protected.

[15] In the event of a prolonged mild blood shortage which is in the region of 3 to 7 hours with the Reptilian brain protected the Human brain will pay the price of this blood shortage. He referred to this type of damage as the **prolonged partial insult**. Where there is sudden severe assault because of severe blood shortage to the brain such that there is no time to shunt the blood from the Human brain the Reptilian brain is exposed to a deprivation of blood supply. The injury resultant from this shortage is referred to as the **acute profound insult**. This results, at best, in severe brain damage and at worst, in death of the baby to be born. The MRI helps to detect the extent of any such brain injury or assault.

[16] He used slide 6 of exhibit 2 to demonstrate a prolonged partial hypoxia-ischemia by showing normal blood supplied artery and a compromised blood supplied artery. The coronal view of the head has arrows, indicating the shunting process on the Human brain, thus the ischemic superficial hemisphere, leaving the Reptilian brain, in the shape of a butterfly, in the deeper central nuclei, protected from the injury as a result of a compromised blood supply. He also demonstrated a foetus in utero with blood supply to the foetus via an umbilical cord and a partial placental abruption. There is decreased blood pressure – moderately hypotension. Slide 6 is a clear indication of a prolonged partial hypoxic ischemic insult with Reptilian brain at the centre having a butterfly shape.

[17] Slide 7 was used to demonstrate a sudden profound hypoxia-ischemia with concepts of further decrease in oxygenated blood flow leading to cardiac arrest. There is an artery with compromised blood supply and another with no blood supply. The coronary view of the head shows ischemic cerebral hemisphere, meaning the Human brain with no injury and the ischemic deeper nuclei in the form of a butterfly, where the injury occurred. The foetus in utero shows a placenta separating from the uterine wall and a decrease in oxygenated blood supply to the foetus via the umbilical cord. The blood oxygen concentration has fallen - hypoxia. Slide 8 shows a clear acute profound hypoxic ischemic injury, that is injury to the Reptilian brain at the centre with a butterfly shape and a Human brain with no injury.

[18] He said that when there is an injury to the brain, as a result of which blood is shunted away from the area it is supposed to go to, those areas have cavities as a result of a loss of cells. These spaces fill up with water such that the area looks white. Blood in the brain is supplied by the anterior cerebral artery, the middle cerebral and the posterior cerebral arteries. The posterior arteries supply the Reptilian brain. The overlapping areas in between the places supplied by these arteries are called the watershed areas. It is in these watershed areas that the shunting of blood begins. If pressure drops further, the anterior and the middle arteries will constrict and the posterior arteries will dilate to allow more blood supply to the Reptilian brain. If things go really bad the Human brain can be destroyed in a process to protect the Reptilian brain.

[19] On 20 February 2014 he was presented with MRI brain scan of K with a request to report thereon. He was not presented with any clinical features of the child. He preferred it that way so as to avoid such features contaminating his investigations. He was not concerned with what happened, where it happened and how it happened as he was to give the most scientific and objective report. Slides 15 and 16 of exhibit 2 were of K. He said that both slides showed the anterior watershed infarction and the posterior watershed infarction, represented by red and yellow arrows, meaning that the injury was of the front and back of the Human brain. He said that this evinced a clear case of a **prolonged partial hypoxic ischemic insult** suffered by K.

[20] Reference was also made by Professor Lotz to blue arrows inside the circle on slide 15 indicated as **Pulvinars**. He explained that when children are being looked after in the high care, after birth, if the sugar level of a child is not properly monitored, the child may develop low sugar in the blood system, a condition called **hypoglycaemia**. Pulvinars may be found close to the Reptilian brain. He added though that the Pulvinars do not necessarily play any part in the acute profound injuries. It was important to get Hospital records for K to know if she had low sugar. If she had it, he said it would be very bad as she was at high care and she should have been looked after very well. He made reference to his filed report which he said was

last updated in 2016. He confirmed the report[2] and adopted it as his evidence. The discussion/Assessment according to the report of the two experts is:

> 'The MRI distribution of changes when K was 8 years 1 month old, namely deffuse bilateral periventricular and subcortical cystic and non-cystic leukomalacia and bilateral T2 hyperintensity-pulvinar, lateral thalamic and PLIC would be in keeping with hypoxic ischemic event/s, mixed pattern predominately of a partial prolonged type.'

[21] In terms of the joint minute both he and Dr Weinstein picked up the Pulvinars and the occipital damage to K. Both suggested that hypoglycaemia be excluded on K. He testified though that there were features that pointed to a hypoglycaemic injury on K but that did not mean that the MRI was the last word on this aspect. When asked about a contribution to the injury by a nuchal cord, he said that the issue fell outside the scope of his expertise. By a partial prolonged type injury he said this meant that the child had no reserves for a while due to circulatory collapse resulting in there being an acute profound injury.

[22] He then explained a **motor cortex** which he said was a strip around the cortex like an Elis band. Its function is to allow the baby to move her hands, lips, tongue etc. It allows a child to move her head, to suckle and swallow. An injury on the cortex is referred to as **mixed**. It has a little bit of profound features and is by far a **prolonged partial insult** or **injury**. A **mixed pattern insult** is acute profound with partial prolonged features. When he and Dr Weinstein agreed that there was a mixed pattern predominantly of partial prolonged type, he understood this to mean that 98 % of what they saw was prolonged partial type. But there was something in the area of the motor cortex which suggested that there may have been a point in the development of this situation when the baby had no reserves and she collapses. He said this was to come out in the neonatology.

[23] When asked if he could determine the probable cause and timing of the injury, he referred to the American Collage of Obstetricians and Gaenocologists (Acox

[2] This is a joint minute of two Radiologists Prof Lotz (for the Plaintiff) and Dr A Weinstein (for the Defendant) as per Bundle E pages 1 – 3 dated 15 August 2016.

Report) last report of 2014 which says that Radiologists' opinion must be placed on a big plane of reference, meaning all causes of this injury must be defined on further assessment by experts of various other fields such as Neonatologists. He noted that the occipital lobes were the areas where the injury was seen and it had to be checked if K suffered any visual damage or visual loss. He said that a hypoxic ischemic injury played out over a period of time and so he did not have to look at any earlier scans preceding her 8 years of age as the brain did not have any regenerative ability. He testified that monitoring the baby at birth was very important. When there were signs of destress these had to be attended to urgently lest the situation complicates with resultant irreparable harm or even death.

Professor Johan Smith [Neonatologist]

[24] Professor Johan Smith confirmed that he was specialising in Paediatrics.as a Head of Department at Tigerberg Medical Hospital, in Stellenbosch. He said that he was very familiar with the working of the cardiotocograph known as the CTG. In the main he relied on the joint minute of the Radiologists in respect of the type of brain damage on K. He identified two types of hypoxic ischaemic insults:-

24.1. Sudden or acute-profound insults which occurs as a result of an insult which does not allow for the auto regulatory shunting of blood to the reptilian brain;

24.2. Intermittent hypoxia which, in a period of 60 - 90 minutes results in decompensation. The rate of decompensation is determined by the quality of the foetal reserves, the duration of the insult and the degree of the insult. He was firm in saying that the injury on K occurred during the intrapartum period and presented as a result of partial prolonged hypoxic ischaemic events, which developed and occurred over hours resulting in a predominantly partial prolonged insult and eventual culmination into an acute profound injury which he said probably occurred after sufficient depletion of foetal reserves.

[25] He said that it would be of importance in respect of this matter to know whether, in fact, a CTG tracing was done in respect of the plaintiff during the birth.

He referred to the available birth records and confirmed that according to these records the neonate was born with normal anthropometrical measurements but, within an hour of birth, did not breath, had a reduced muscle tone and a blue coloration. As a result, resuscitation was performed, and the neonate commenced independent breathing after 4 minutes. The neonate was placed in a head box and saturation improved to 93% (normal). He testified that having regard to the physical features noted, the identification of encephalopathy, the decompensation, the lack of muscle tone and inability of primitive reflexes the neonate was clearly depressed and suffered from respiratory distress and was diagnosed with asphyxia.

[26] He said that because of the arterial blood gas obtained 42 minutes after birth and the assessed pH of 7.23, the neonate was clearly acidotic, which is associated with a lack of oxygenation during the birth process. The neonate presented with clinical features of metabolic acidosis, hyponatremia, raised lactate that can be caused by oxygen deficiency to cells, increased global muscle tone, eventual mild respiratory distress and ultimately excessive levels of oxygen in the blood caused by overventilation.

[27] He said that the arterial blood gas of 13h15 on 30 January 2006[3] indicated:

27.1. A PH of 7.46 indicative of alkalosis (too much alkaline in the blood due to overventilation) and still washing out too much carbon dioxide;
27.2. Neurogenic hyperventilation indicative of acute respiratory and neurological compromise;
27.3. The cranial ultrasound of the brain at 14h53 on 30 January 2006[4] revealed features in keeping with brain swelling (oedema).

[28] He concluded that K was a depressed baby at birth which required resuscitation and had clear clinical signs of additional organ injury and swelling of the brain. The hypoglycaemia may have exacerbated the intra-partum injury but he conceded that there may not be any brain damages as a result of hypoglycaemia and it appears as a result of the joint radiologists' minute that this can now be

[3] (Page 22, Bundle F)
[4] (Bundle F, Page 23).

excluded. The mechanism of the mixed MRI pattern in respect of K is best described due to the sufficient depletion of foetal reserves.

[29] In respect of an umbilical cord Professor Smith gave evidence that the cord consists of a vein and two arteries of which the arteries have muscular walls. Compression of the vein would normally result in an anaemic neonate. In any event any impairment of blood perfusion due to cord compression would have been identifiable through proper monitoring. The presence of a nuchal cord does not require diagnosis through a sonar and involvement of the nuchal cord causing blood compromise can and should be detected through monitoring as a tight nuchal cord might be associated with foetal distress. In reaching his opinion he did not exclude the nuchal cord around the neck and remains of the opinion that the injury sustained by the neonate probably occurred during the intrapartum period due to undetected foetal distress or a failure to react to foetal distress.

[30] When asked whether the presence of a nuchal cord around the neck can cause an acute profound brain insult, Professor Smith explained that it can occur only as a result of and following upon a partial prolonged insult process culminating in depletion of foetal reserves and then resulting in an acute profound brain insult. Heh explained that no sentinel event was associated with this birth and that a cord around the neck does not qualify as a sentinel event like a prolapse of the umbilical associated with falling of the umbilical through the vagina. Although a tight cord around the neck may be a medical emergency it does not qualify as a Sentinel event, as Sentinel events are associated with a sudden and severe regulatory collapse. He dismissed reasoning put to him that an acute profound insult to the brain can result in a subsequent partial prolonged injury, stating that same is based on incorrect reasoning and not clinically sound.

[31] In dealing with the ACOG criteria in respect of intrapartum hypoxic ischaemic encephalopathy Professor Smith explained comprehensively that the ACOG criteria are not available in the South African context and as a result the ACOG template does not fit a Third World base.

[32] In respect of the acidosis Professor Smith gave evidence that the base deficit and relevant predictable rates would have resolved within 4 hours and in addition the neonate presented with an abnormal drive to reduce carbon dioxide which is associated with asphyxia and part of neonatal encephalopathy.

[33] He furthermore testified that, with proper monitoring, the distress could and should have been detected at an early stage and that intervention would have led to a positive result for the neo-born. The process of Labour should be monitored through any of the known mechanisms of assessment to monitor foetal heartrate patterns and the increase and decrease thereof before, during and after contractions. Foetal heart rate monitoring is conducted by means of, either-auscultation, doppler assessment or CTG which depicts a graphic pattern and produces an audible sound which requires interpretation of patterns associated with foetal distress and informs the decision to intervene and accelerate birth. A partogram may also be used to monitor birth as it is designed to plot and to graphically display the maternal and foetal condition.

Dr Johannes Petros Hattingh Basson [Obstetrician and Gynaecologist]

[34] Dr Basson was in full time private Obstetrical and Gynaecological practice at MediClinic, Welkom. In 2003 to 2012 he was an assessor for maternal deaths in the Free State for NCCEMD appointed by the Minister of Health. In his lifetime he delivered about 28000 babies. He confirmed the contents of his report and a joint minute drawn by him and Dr Peter C Koli, also an Obstetrician and Gynaecologist (for the Defendant). Both Doctors agreed that:

34.1. No antenatal or intra-partum records were available;

34.2. Intra-partum records were of the utmost importance to evaluate the intra-partum management of the patient. It is an obligation in terms of the Health Act No61 of 2003 that patient records were safely kept and made available for evaluation;

34.3. K suffered a brain injury, most likely in the peri-partum period, resulting in severe handicap.

[35] His evidence and his reports were based on the available Hospital records he received, K's Road to Health Chat, Medico-legal reports by Professors Lotz and Smith and on information he received from the Plaintiff. He said that the intra-partum hypoxic ischemic injury of a predominantly partial prolonged nature resulting in K suffering from cerebral palsy should, as could have been detected through proper foetal monitoring. According to him the accepted protocol for foetal monitoring in low-risk pregnancies during the active phase of labour requires that the foetal condition should be assessed 30 minutes, before, during and after contractions.

[36] He said that foetal monitoring was to be conducted through determination of the foetal Heartrate and heartrate patterns defined by the influence of contractions on the foetal heartrate during the birth process. Any decelerations in foetal heartrate should have been aligned with contractions and any hypoxic ischaemic complications suffered by the foetus would present through various foetal heartrate patterns. These abnormalities do lead to distress and umbilical artery acidemia. These patterns were recognisable as problematic and if they persisted necessitated assisted birth and or caesarean section and or intrauterine resuscitation. He gave evidence that having regard of the factual evidence of continuous CTG monitoring, the medical staff did not react on the probable indications of foetal distress which would have been visible from assessment of the CTG results and failed to expedite delivery.

[37] In respect of the incident of the nuchal cord, he testified that nuchal cords were common and were not associated with adverse outcome in respect of births. The presence of a nuchal cord was associated with an outcome of intrauterine growth restriction, cord infarct/stroke, foetal death, meconium aspiration syndrome, increased rate of intrapartum foetal heartrate abnormalities. Patients with nuchal cords around their necks required close monitoring during labour, preferably by continuous foetal electronic heartrate monitoring as tight and multiple nuchal loops were associated with persistent variable or late decelerations. According to him the staff did not react to the expected indicators associated with foetal monitoring. He said that it is highly probable that foetal distress was present but missed as a result. Commenting on the scores in the patient ward control register on K he said that 6/10 – 7/10 taken in 5 minutes was an average score. 4/10 was a bad score even though

one was not to look at one score as the condition of the baby improves after every 5 minutes. The score according to him should have been 9/10 or 10/10. He said that a tight nuchal cord could cause asphyxia but was unlikely to lead to long term disfigurement. He said that the absence of meconium was not helpful as meconium was not the only source of asphyxia.

[38] Dr Basson and Dr Koli differed on the approach to be adopted in the absence of intrapartum records. Dr Basson said that intrapartum records were of utmost importance for the filing of a balanced report on patient care in cases of known adverse outcome where access to records has been denied. However, collateral information may be used, though with circumspection, to determine the standard of care.

Dr Debora Francis Anna Pearce [Paediatric neurologist]

[39] She testified that she met and consulted with the Plaintiff but her report was not limited to that consultation. She examined K who at that time was 10 years 5 months old. Her report has results of such examination[5]. She confirmed her report and adopted it as her evidence. She also met and consulted with Dr Mogashoa, also Paediatric Neurologist (for the Defendant). She confirmed their joint minute. Dr Pearce's evidence was brief. She said that, in her opinion, the insult was in the intrapartum period. The agreement reached by her and Dr Mogashoa was that there was normal foetal brain growth prior to birth, and that it is likely that peripartum or intrapartum hypoxic ischaemia contributed to the pathogenesis of neonatal encephalopathy. Dr Mogashoa added that the nuchal cord contributed to such intrapartum hypoxia and the final agreement is that the child's condition is most likely the result of intrapartum hypoxia.

Defendant's version.

Professor Keith Duncan Bolton [Paediatrician]

[5] See Bundle C p24 to p28.

[40] The defendant called only Professor Bolton to give *viva voce* evidence. In addition to Professor Bolton's evidence, the defendant obtained and filed the medical legal reports from the following experts' witnesses:

40.1. Dr Koll – an obstetrician and gynaecologist who file a joint minute with Dr Basson
40.2. Dr Mogashoa– a paediatric neurologist who filed a join minute with Dr Pearce
40.3. Dr Weinstein – a radiologist who filed a joint minute with Prof Lotz and
40.4. The nursing sister Smit.

[41] Professor Bolton testified that he was employed as a part time consultant Paediatrician at Rahina Moosa Mother and Child Hospital in the Gauteng Department of Health, after having been a Chief Paediatrician there. From his stated qualifications and experience he is indeed an expert in his specialised field. He was furnished with documentation on K by attorneys of the State Attorneys' office in Johannesburg. He confirmed and adopted as his evidence his initial and the subsequent reports he made in this matter. The subsequent report was necessitated by the discovery of new documents. Commenting on the Hospital's Midwife's notes he said that the Apgar score recorded was 6/10 – 7/10 and the nurse referred the baby from the delivery cubicle to the "sick bay nursery" in the labour ward.

[42] Commenting on the Doctor's notes he, *inter alia*, said that it was noted that K has the umbilical cord around her neck at birth. Her birth weight was 3000 grams and her crown-heel length was 53 centimetres with the head circumference of 36 centimetres. The Doctor's one minute Apgar score was 4/10 and the five minute Apgar score was 7/10. When K was brought to the sick bay, it was noted that she was not breathing, was cyanosed (blue) and was floppy. She was bagged and spontaneous breathing commenced after 4 minutes. The oxygen saturations increased from 78% to 94%. The neurological examination after resuscitation showed that the baby was lethargic, had poor reflexes and she was breathing at an abnormally fast rate (tachypnoea) . A blood gas analysis was performed 42 minutes after birth while she was in 15 litres head box oxygen. The important results showed pH – 7.232, pC02 – 44.2, pa02 – 65.4, B.D – 8.2. A cranial sonar performed in the

early post-natal period showed: “bright brain, good pulsation and no bleeds.” He took Court into various parts of his report, including probable/possible additional contributing factors to the cause of cerebral palsy in this case as partly being:

> ‘Nuchal cord (NC) one probable proximal contributing factor was the reported cord around the neck at birth. The definition of a NC is where the umbilical cord is wrapped 360 degrees around the foetal neck. While single, loose NC is common and has a benign outcome, the same is not true for multiple and tight loops. Non-reassuring CTG, meconium-stained liquor, 5 minutes Apgar Scores < 7 and NICU transfers are and the midwife had to cut significantly more common with tight NC.
> The nuchal cord was tight and the midwife had to cut the cord and clamp it to facilitate the second stage of labour.
> It is therefore probable that NC played a role in this child’s birth asphyxia. The current concept for the development of Neonatal Encephalopathy stresses the multifactorial and “broader perspective” that is necessary when considering causation. The nuchal cord was not of the defendant’s making and antepartum diagnosis of the NC (by ultrasound) does not justify a change in delivery management.’

[43] The conclusions reached by Professor Bolton were put into serious doubt during cross-examination when taking into consideration the concessions made by the Defendant in the third answers to the pre-trial minute and the agreed conclusions of the experts of both parties in their joint minutes. In his last remark on conclusion he said: -

> ‘However the lack of contemporaneous notes covering the confinement, labour and early new-born period makes it difficult to support or refute the Plaintiff’s version of events.’

[44] Conclusions reached by the experts of the Defendant who did not give *viva voce* evidence were by and large in agreement with the conclusions reached by Plaintiff’s experts with the exception of Dr Mogashoa who differed on three aspects from conclusions or observations of Dr Pearce. The three situations are in respect of:-

44.1 All of the essential criteria necessary to consider intrapartum hypoxia are present and that a diagnosis of intrapartum hypoxia ischemic encephalopathy can therefore be made in terms of the evidence of, inter alia:

44.1. 1. Cerebral palsy of mixed cerebral palsy, predominantly dystonic. Dr Mogashoa said she found mixed cerebral palsy, predominantly dyskinetic and spastic quadriplegic;

44.1.2 Exclusions of other identifiable causes: infection CPR noted to be 26. Blood cultures negative. LP not done. No infection confirmed or disproved. Dr Pearce: the current literature suggests that CPR may be elevated in some non-infectious conditions (prolonged rupture of membranes, maternal fuver during labour, foetal distress, perinatal asphyxia, shock, intraventricular haemorrhage, pneumothorax, pneumothorax and meconium aspiration pneumonitis). Dr Mogashoa agreed but deferred to Neonatologists to comment on CPR.

44.1.3 Having regards to ACOG 2014 it is likely that peripartum or intrapartum hypoxic ischemia contributed to the pathogenesis of neonatal encephalopathy. Dr Mogashoa agreed but added that it was her opinion that nuchal cord contributed to the intrapartum hypoxia. However both agreed that K's condition was most likely the result of intrapartum hypoxia.

Evaluation

[45] As already alluded to the civil onus rests on the Plaintiff to satisfy Court that its version is more probable and consists of credible evidence as compared to that of the Defendant. In AA Onderlinge Assuransie Assosiasie Beperk v De Beer[6] the Court dealt with and indicated that the balancing of probabilities means:

[6] 1982(2) SA 603 (A).

45.1 To select a conclusion which deems to be the more natural, or plausible, conclusion;

45.2 From amongst several conceivable ones;

45.3 Even though that conclusion may not be the only reasonable one.

Negligence

[46] This claim is based on the negligence of the medical and/or nursing staff of the Hospital, resulting in the child suffering from cerebral palsy. Proof of negligence depends on whether conduct, in the circumstances of each case, falls short of that of a reasonable man. The test for negligence appears in the following dictum of Holmes JA in Kruger v Coetzee:

> 'For the purpose of liability culpa arises if-
>
> (a) A diligens 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.'

[47] Following this judgment, there were a number of Supreme Court of Appeal judgments reformulating this requirement. Thus, OLIVIER JA, said this in Mukheiber v Raath and Another[7]:

> 'For the purposes of liability culpa arises if –
>
> (a) A diligence paterfamilias in the position of the defendant –
>
> (i) Would have foreseen harm of the general kind had actually occurred;

[7] 1999(3) SA 1065 (SCA), para [31] (emphasis added)
This was apparently applied in *Sea Harvest Corporation (Pty) Ltd and Another v Duncan Dock Cold Storage (Pty) Ltd and Another* 2000(1) SA 826 (SCA), paras [21] and [22]
In *Mkhatswa v Minister of Defence* 2000(1) SA 1004 (SCA), paras [19] to [23], SMALBERGER JA referred, with approval, to the point SCOTT JA articulated in *Sea Harvest* (paras [21] and [22])

(ii) Would have foreseen the general kind of causal sequence by which that harm occurred;

(iii) Would take reasonable steps to guard against it; and

(b) The defendant failed to take those steps.'

[48] In Standard Chartered Bank of Canada v Nedperm Bank Limited[8] the principle was stated as follows:

> 'In delict, the reasonable foreseeability test does not require that the precise nature or the exact extent of loss suffered or the precise manner of the harm occurred and should have been reasonably foreseeable for liability to result. It is sufficient if the general nature of the harm suffered by the plaintiff and the general manner of the harm occurring was reasonably foreseeable.'

[49] NEETHLING and POTGIETER [9] state, with reference to these decisions:

> *"*From this it can be concluded – and this is important – that because both the concrete and abstract approaches require foreseeability of the general nature of the consequences and the general manner in which it occurred. Both approaches should as far as negligence is concerned, produce the same result.*"* [10]

Inferential reasoning

[50] The case of the Plaintiff is largely reliant on circumstantial evidence in deciding whether any Hospital personnel acted negligently as a result of which K suffered cerebral palsy. The direct evidence of the Plaintiff is limited. When an inference of negligence would be justified and to what extent expert evidence would be necessary would depend on the facts of the particular case. A court is not called upon to decide the issue of negligence until all of the evidence is concluded. Thus, any such explanation as may be advanced by a defendant forms part of the

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[8] 1994(4) SA 747 (A), at 65

[9] *op cit* Chapter 4, footnote 130

[10] The concrete approach originated in BOBERG, "*The Law of Delict*", p. 390
See, also, *Barnard v Santam Bpk* 1999(1) SA 202 (SCA), 213-215

evidential material to be considered in deciding whether a plaintiff has proved the allegation that the damage was caused by the negligence of the defendant.[11]

[51] In Ratcliffe v Plymouth and Torbay Health Authority[12] at paragraph 48 Lord Justice Brooke made the point that:

> '…surrounding a procedure which led to an unexpected outcome for a patient. If such a case should arise, the judge should not be diverted away from the inference of negligence dictated by the plaintiff's evidence by mere theoretical possibilities of how that outcome might have occurred without negligence: the defendants' hypothesis must have the ring of plausibility about it. .
> It is likely to be a very rare medical negligence case in which the defendants take the risk of calling no factual evidence, when such evidence is available to them, of the circumstances.'

[52] It suffices for plaintiff to convince the court that the inference that he or she advocates is the most readily apparent and acceptable inference from a number of possible inferences.[13] In Caswell & Powell Duffryn Associated Collieries[14] at 169 – 170, Lord Wright remarked: -

> 'Inference must be carefully distinguished from conjecture or speculation. There can be no inference unless there are objective facts from which to infer the other facts from which it is sought to establish. In some cases, the other facts can be inferred with as much practical certainty as if they had been actually observed. In other cases, the inference does not go beyond reasonable probability. But if there are no positive proved facts from which the inference can be made, the method of inference fails and what is left is mere speculation or conjecture.'

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[11] Goliath v Member of the Executive Council for Health, Eastern Cape (085/2014) [2014] ZASCA 182; 2015 (2) SA 97 (SCA) (25 November 2014).
[12] [1998] EWCA Civ. 2000.
13AA Onderlinge Assuransie-Assosiasie Bpk v De Beer 1982 (2) SA 603 (A); see also Cooper & another NNO v Merchant Trade Finance Ltd 2000 (3) SA 1009 (SCA).
[14] [1940] AC 152.

Negligence and causation

[53] Patients of public health institutions are entitled to be treated in the same way as patients in private medical institutions. What is required is a public health delivery system that recognises the dignity and rights of those who are compelled to use its facilities. It is that basic sensitivity that the Constitution demands.[15] It needs only be foreseeable that the plaintiff will suffer damages and the precise nature of the damages need not be precisely foreseeable at that stage.[16] Although the onus of proving negligence is on the plaintiff, the plaintiff does not have to adduce positive evidence to disprove every theoretical explanation which is exclusively within the knowledge of the defendant, however unlikely, that might be devised to explain in a way which would absolve the defendant and his employees of negligence.[17]

[54] Sometimes, however, a plaintiff is not in position to produce evidence on a particular aspect. Less evidence will suffice to establish a *prima facie* case where the matter is peculiarly in the knowledge of the defendant. In such situations, the law places a shifting evidentiary burden upon the defendant to show what steps were taken to comply with the standards to be expected. The general *onus* nevertheless remains with the plaintiff.[18] In Vallaro obo Barnard v MEC[19] which is full bench decision of this division Court held, with reference to McIntosh v Premier, Kwazulu-Natal and Another[20] in para 12 , inter alia, that:

> 'The second inquiry is whether there was fault, in this case negligence. As is apparent from the much-quoted dictum of Holmes JA in Kruger v Coetzee 1966 (2) SA 428 (A) at 430E-F, the issue of negligence itself involves a twofold inquiry. The first is: was the harm reasonably foreseeable? The second is: would the diligens paterfamilias take reasonable steps to guard against such occurrence and did the defendant fail to take those steps? The answer to the second inquiry is frequently expressed in terms of a duty. The foreseeability requirement is more often than not assumed, and the inquiry is

[15] Premier, KwaZulu-Natal v Sonny and another 2011 (3) SA 424 (SCA) in paras 33 and 34.
[16] Botes v Van Deventer 1966 (3) SA 182 (AD) at 191A – G.
[17] Naude N.O. v Transvaal Boot and Shoe Manufacturing Co 1938 AD 379 at 392-3).
[18] Monteoli v Woolworths (Pty) Ltd 2000 (4) SA 735 (W) at [27].
[19] Appeal case no A5009/16, Gauteng Local Division, Johannesburg.
[20] 2006 (6) SA 1 (SCA).

said to be simply whether the defendant had a duty to take one or other step, such as drive in a particular way or perform some or other positive act, and, if so, whether the failure on the part of the defendant to do so amounted to a breach of that duty. But the word 'duty', and sometimes even the expression 'legal duty', in this context, must not be confused with the concept of 'legal duty' in the context of wrongfulness which, as has been indicated, is distinct from the issue of negligence.

[14] The crucial question, therefore, is the reasonableness or otherwise of the respondents' conduct. This is the second leg of the negligence inquiry. Generally speaking, the answer to the inquiry depends on a consideration of all the relevant circumstances and involves a value judgment which is to be made by balancing various competing considerations including such factors as the degree or extent of the risk created by the actor's conduct, the gravity of the possible consequences and the burden of eliminating the risk of harm. …'

[55] Commenting on an approach to factual causation as stated in Lee v Minister for Correctional Service[21] Mogoeng CJ in Mashongwe v Prasa,[22] *inter alia*, said:

'[65] Lee never sought to replace the pre-existing approach to factual causation. It adopted an approach to causation premised on the flexibility that has always been recognised in the traditional approach. It is particularly apt where the harm that has ensued is closely connected to an omission of a defendant that carries the duty to prevent the harm. Regard being had to all the facts, the question is whether the harm would nevertheless have ensued, even if the omission had not occurred. However, where the traditional but-for test is adequate to establish a causal link it may not be necessary, as in the present case, to resort to the Lee test.'

[56] With all these legal principles in mind, I turn to the evidence adduced by the parties on the issue of negligence. The Plaintiff testified that she spent more than an hour waiting in the first cubicle where urine testing was done and the CTG monitor

[21] 2013 (2) SA 144 (CC). The point of departure is to have clarity on what causation is. This element of liability gives rise to two distinct enquiries. The first is a factual enquiry into whether the negligent act or omission caused the harm giving rise to the claim. If it did not, then that is the end of the matter. If it did, the second enquiry, a juridical problem, arises. The question is then whether the negligent act or omission is linked to the harm sufficiently closely or directly for legal liability to ensue or whether the harm is too remote. This is termed legal causation.
[22] 2016 (3) SA 528 (CC).

was connected on her apparently by the first nurse she came into contact with. It was at this cubicle that some doctors spoke about a Caesarean section. At that stage she had not felt labour pains yet. She was, for some time, left in that cubicle as she said she was not taken to theatre. Another nurse came to her to give her the first doze of medicine to induce labour. That nurse returned later to give her the second doze, still to onset labour. She was thereafter moved to the delivery room where a doctor broke her water. The next person to attend to her was a different nurse who told her when and how to push. Still the Plaintiff was feeling no labour pains and the baby was not coming and it took quite a while. Then that nurse called for a doctor. It was a different doctor from the one she met before. The doctor called for a pair of scissors. The nuchal cord was clamped, cut and the baby was pulled out. The rest is the history testified to by various witnesses which history includes that:

> 'When K was brought to the sick bay, it was noted that she was not breathing, was cyanosed (blue) and was floppy. She was bagged and spontaneous breathing commenced after 4 minutes. The oxygen saturations increased from 78% to 94%. The neurological examination after resuscitation showed that the baby was lethargic, had poor reflexes and she was breathing at an abnormally fast rate (tachypnoea).'[23]

[57] The estimated time when the Plaintiff met the first doctor was around 11h00 and K was born at 15h30. While an attempt was made to challenge times taken during delivery, no counter evidence was adduced on this aspect by the Defendant. I accordingly accept the times estimation given by the Plaintiff on events from 11h00 to 15h30, notwithstanding a discrepancy on the Plaintiff's version of when she left home for the Hospital. I have already found that most of Plaintiff's evidence became common cause during the trial.

[58] There is no direct evidence of when intrapartum commenced. Yet the first doctor to attend to the Plaintiff at around 11h00 already had a reason to be concerned about her health status such that a consideration of a caesarean section was made. At this stage, the only source for concern would be the recording on the CTG monitor whose records have gone missing. The Plaintiff testified about nurses and doctors who came to her and what they did on her. Her evidence clearly

[23] See Prof Bolton's evidence.

indicates that there are moments when she was left alone. In the absence of labour pains she was well orientated to see what was happening around her. One can also glean from the patients ward control register that between 24 January 2006 and 25 January 2006 no less than 13 baby deliveries were made and attended to by the labour ward staff.[24] I note that the dates and times of birth were not registered sequentially and some of the required information is lacking from the register. Understandably, the labour ward staff might have been fairly busy moving from one patient to another. Yet certain minimum standards had to be observed and followed.

[59] I find that the consideration of a caesarean section was either as a result of warning signs depicted on the CTG monitor or from the condition of the Plaintiff at the time, such as might be the dilation of her vertex. None of the attendant doctors or nurses came to testify to negate this irresistible conclusion. In my view, had facilitated methods of delivery, such as the caesarean section, been resorted to around 11h30, K would have been born much earlier than 15h30. Time of birth could have been around 12h15 to 12h30. Among such facilitated methods, a caesarean section, would then have had the advantage of avoiding K presenting herself with a nuchal cord. It remained common cause that K was a healthy baby just before the delivery process commenced. The probabilities of a child born without suffering asphyxia in this case are very high, had early interventions, such as caesarean section, been resorted to, in response to those early warnings depicted from the CTG. The consideration of the caesarean section was a manifestation by the doctors that they foresaw a complicated delivery for which a caesarean section was the solution. They however, and without any given explanation, failed to carry out the reasonable procedure to alleviate the impending harm. It has to be borne in mind, as testified by Professor Smith, that with proper monitoring, the distress to the foetus could and should have been detected at an early stage and that intervention would have led to a positive result for the neo-born. The CTG monitor produces an audible sound which requires interpretation of patterns associated with foetal distress and informs the decision to intervene and accelerate birth. Clearly this information was either missed out by the attendant doctors and/or nurses or was picked up but no reacted to. By failing to resort to that early intervention procedure, they failed to do

[24] See pages 73 – 74 of bundle F. We do not have information on preceding and succeeding pages. There could be deliveries during this period.

what a reasonable person, possessed with information they had, would have done in the circumstances. Their omission was therefore negligent.

[60] The failure of the doctors to intervene as soon as a need for it arose had the effect of prolonging the birth of K by not less than 3hours. After such a delay, K was lethargic, had poor reflexes and she was breathing at an abnormally fast rate (tachypnoea). K had bright brain, good pulsation and no bleeds. She was not breathing and was cyanosed (blue). She could only breathe on her own after 4 minutes of resuscitation after birth. She was diagnosed with cerebral palsy. Professor Bolton testifying for the Defendant said this condition was probably due to a tight nuchal cord around the neck of K at birth. This could have been avoided by an earlier intervention. In any event, the patients ward control register refers to the cord being slightly tight. Besides this evidence no witness came forward from the Defendant to testify on the degree of tightness of the cord. To therefore rely on a cord that was slightly tight, without giving more details of this, is rather conjecture.

[61] Professor Lotz was the most impressive of all witnesses who gave *viva voce* evidence in this matter. In much simpler terms he explained how blood in the human brain was shunted to protect the reptilian brain and the significance of the injuries in the two areas of the brain. With slides he showed the coronal view of the head of K having arrows, indicating the shunting process on the Human brain and the resultant ischemic superficial hemisphere, leaving the Reptilian brain, in the shape of a butterfly, in the deeper central nuclei, protected from the injury as a result of a compromised blood supply. He said that the blood shunting process he observed through the MRI on K would have endured for a minimum of about 3 hours but not more than 7 hours. He referred to the process as prolonged partial insult as opposed to a sudden severe assault because of severe blood shortage to the brain such that there is no time to shunt the blood from the human brain, resulting in the acute profound insult. The 3 hours delay of birth caused by a failure to implement a caesarean section co-insides with the minimum period of a prolonged partial insult.

[62] Professor Lotz said it repeatedly that the loss of oxygenated blood to the human brain is inevitably at a price. The areas where the blood is stolen from in the human brain control various sites. I accept the undisputed evidence of Professor

Lotz and Dr Weinstein that in the brain injury of K there was a mixed pattern predominantly of partial prolonged type, meaning that 98 % of what they saw was prolonged partial type. But there was something in the area of the motor cortex which suggested that there may have been a point in the development of this situation when the baby had no reserves and she might have collapses.

[63] Further I accept the uncontroverted evidence of both Drs Basson and Koli who agreed that, while there were no antenatal or intra-partum records available; such records were of the utmost importance to evaluate the intra-partum management of the patient and it was an obligation in terms of the Health Act No 61 of 2003 that patient records were safely kept and made available for evaluation, K did suffer a brain injury, most likely in the peri-partum period, resulting in severe handicap. The brain handicap suffered by K was of a cerebral palsy. On the total probabilities of this matter, I find that the negligent conduct of the employees of the Defendant caused K to suffer from the cerebral palsy.

Findings

1. The neo-natal signs were consistent with an intrapartum event predominantly of the partial prolonged type with features associated with acute profound insult and that this resulted from a failure to intervene in time. if the birth was properly managed the stressful situation facing the foetus could and should have been recognised and reacted upon.
2. Court finds for the Plaintiff – the Defendant acted negligently causing K to suffer from cerebral palsy.
3. Defendant is ordered to pay the costs of two counsel.

Order

[64] As the issue of quantum was made to stand over by agreement of the parties, the Draft order is made an order of Court.

_____

H Cele
Acting Judge Gauteng
Division of the High Court

Appearances

For the Plaintiff:	Adv.N Van Der Walt SC
Assisted by:	P Uys and R Andrews
Instructed by:	Renefouche Attorneys
For the Defendant:	Adv. N Makopo
Assisted by:	Adv. B Shabalala
Instructed by	State Attorney