IN THE HIGH COURT OF SOUTH AFRICA

(GAUTENG DIVISION, PRETORIA)

DELETE WHICHEVER IS NOT APPLICABLE

(1) REPORTABLE: YES/~~NO~~.

(2) OF INTEREST TO OTHER JUDGES: YES/~~NO~~.

(3) REVISED.

24/6/2016

DATE SIGNATURE

Case Number: 17386/2014

DATE: 24/6/2016

In the matter between:

S S 1ST PLAINTIFF

S S OBO C 2ND PLAINTIFF

And

ROAD ACCIDENT FUND DEFENDANT

JUDGMENT

Fabricius J,

1.

On 10 December 2012, the First Plaintiff, being pregnant with a child subsequently born on 28 February 2013, was involved in a head-on collision. The mother, S, did not sustain serious injuries and the child C, was born with cerebral palsy some seven weeks after this collision. Her parents sustained severe injuries. The occupants of the other vehicle were killed. The only question in this tragic case that I need to decide is whether or not the condition of cerebral palsy was caused by the high-velocity impact, or whether it is a congenital defect. Brief evidence was led by the mother as to what had occurred and during the trial it became common cause that the said collision could be described as a "high-velocity impact". The other vehicle that crashed into the vehicle in which the Plaintiffs were passengers was almost totally destroyed as is evident from the photographs presented to me. In my view, even a layman can see that a collision at high speed had occurred. The agreed upon high-velocity impact is in my view a relevant consideration as will become apparent when I deal with the medical evidence presented during this trial.

Viewed differently, as Defendant does, can it be reasonably said that C's condition was not at all related thereto?

2.

The mother can be glad, to put it mildly, that she escaped this horrendous accident with relatively minor injuries. According to the Particulars of Claim she sustained soft tissue injury to the hand and neck, blunt chest contusion, abrasions on the left shoulder, and multiple scarring. The Particulars of Claim further allege that the birth of C was normal but, subsequent development milestones confirmed that she was brain damaged. The clinical presentation was that of cerebral palsy and quadriplegia. It was pleaded that these injuries were caused by the collision and that the "accelerated deceleration" during the point of impact "caused bleeding" in C's brain. Damages were claimed in respect of both Plaintiffs, but the quantum issue is not before me.

3.

Defendant denied that the said condition was caused by the collision and in the alternative pleaded that the injuries were the result of pre-existing condition. Alternatively, it was averred that the injuries referred to, resulted post-accident.

4.

The mother's evidence can be summarised as follows:

She was 28 weeks pregnant with C at the time and on 10 December 2012, was a passenger in a Toyota Hilux Bakkie. A BMW drove into this vehicle at high speed which she estimated to have been 180km per hour. She also testified that the Hilux, after the impact, tipped over onto its side. I will refer to this evidence again when I deal with the medical opinions, because as innocuous as this may seem at this stage, it becomes an important consideration. Her parents were critically injured in the accident and the occupants of the BMW were killed. The mother also testified that at the time she felt as if her whole body "wanted to climb through the windscreen".

5.

Mrs S was taken to hospital and she informed the paramedics that she was 28 weeks pregnant. She was examined for lacerations and an x-ray was done to determine whether there were any other orthopaedic injuries. A few hours after the accident, she was transferred to the maternity ward where a sonar scan was performed. She was told that she had gone into early labour which needed to be arrested. The bedside sonar scan that was done showed that the foetus was “fine”. She was discharged from the Welkom Medi-Clinic on 11 December 2012. On the next day, she again experienced pre-term contractions and again consulted Dr Myburgh at the hospital. Another sonar scan was done and medication was prescribed to stop the contractions. Mrs S also testified that during the rest of her pregnancy with C she experienced less foetal movement and again consulted with her general practitioner and also requested a further sonar scan.

6.

Dr Coleman was Mrs S's gynaecologist/obstetrician. She consulted him three or four times during the pregnancy. The first of these consultations was on 8 August 2012, during which the routine blood-tests were performed. No abnormalities with the foetus were detected. The last visit to Dr Coleman prior to the accident was in early December 2012, during which she was told that the foetus' development was normal, that its growth patterns were normal and that the foetus was positioned correctly in utero.

7.

Post-accident, Mrs S attended further consultations with Dr Coleman. A further sonar scan was done and the test on the umbilical cord, i. e. to test whether the oxygen levels were within acceptable parameters, showed no abnormalities. Dr Coleman's clinical notes do not show that she reported reduced foetal movement. This is not of a particular concern inasmuch as the later medical evidence made it clear that foetal movement varied from time to time, and from person to person. C was born on 28 February 2013 and no abnormalities with the birth were

experienced. After the birth, the mother noted that C was particularly restless, did not sleep as expected, had dietary problems and would become spastic for certain periods. As a result, she consulted a paediatrician, Dr Kriel, who after about four months advised her of the possibilities of neurological problems. She then consulted Dr De Witt, a specialist paediatrician of Arwyp Clinic in Kempton Park. He admitted C to hospital and following an examination, noted that her legs had a tendency to "scissor". He advised the mother that C needed occupational therapy. C remained in this hospital for three days during which several tests were performed on her, including:

7.1 A MRI-scan on 3 July 2013;

7.2 A barium-swallowing test;

7.3 A test for the presence of bowel-obstruction;

7.4 Tests for allergies.

After these tests, Dr De Witt informed C's parents that she had "static brain damage". The mother was told that C would have to see a neurologist, Dr Wilson at the Sunninghill Hospital. C's formal diagnosis is called "mixed cerebral palsy with

spastic dystopia and limp upper body". The mother testified that C cannot sit, stand, walk, feed herself or hold her head upright without difficulty. Mrs S also testified that there was no history of cerebral palsy in her family. During cross-examination, the question arose whether or not Mrs S had sustained cracked or broken ribs, but I do not believe that this is a significant factor in these proceedings. It is clear that she assumed that ribs were broken on her right side due to the pain she felt there. She also testified that at the time of the accident, the safety belt was not resting over her abdomen, but rather across her pelvis. There was also a debate about the absence of foetal distress and it is clear from the relevant clinical notes that no such distress was noted. First Plaintiff was re-called by myself on 13 May 2016 and I will deal with that evidence and why I deemed it proper to re-call her. C accompanied her and I noted certain observations pertaining to asymmetry of her face.

8.

Dr Coleman:

He treated Mrs S both before and after the accident and all the routine tests that were done by him resulted in a finding of no abnormality. He saw First Plaintiff on 10 January 2013, about a month after the accident and at that stage the mother did not raise any concerns. Routine sonars also showed no abnormalities. He also mentioned that he delivered C on 28 February 2013 after a normal and uncomplicated birth.

9.

Dr Myburgh:

She treated Mrs S on the day of the accident and did sonar scans which resulted in her finding no abnormality and no bleeding. Nothing of any particular significance, apart from what I have said, emanated from her evidence.

10.

Dr Pistorius:

I will deal with his evidence in some detail as major parts thereof were challenged by Defendant's expert witness Dr Okoli, a neurosurgeon. He has an impressive *Curriculum Vitae*. It is necessary that I refer to this in some detail as well. He is the holder of a PhD degree in foetal brain imaging from the University of Utrecht in the Netherlands. He is also the holder of a diploma in foetal medicine from the Foetal Medicine Foundation in London. The PhD was obtained in 2008, and this diploma in 2000. His initial MBChB degree was obtained *Cum Laude* in 1986 from the University of Pretoria and the MMed degree from the Stellenbosch University in 1993. He has authored and co-authored more than 50 scientific publications. At present, and since July 2013, he is a maternal and foetal medicine specialist at the Tygerberg Hospital and the University of Stellenbosch. From 2004 to 2013, he was a gynaecologist, maternal and foetal medicine Fellow and later maternal and foetal medicine specialist at the University Medical Centre Utrecht.

His medico-legal report on the child C dated 7 June 2015 said the following:

"Available information:

- Mrs S's first pregnancy was uncomplicated under the care of dr Johan Coleman. Routine blood tests, screening tests for Down-syndrome and neural tube defects as well as routine ultrasound examinations were uneventful.
- Mrs S was involved in a head-on collision on 10 December 2012 (at a gestational age of 30 weeks) in the Free State and sustained minor injuries to the upper body. Other occupants of the same vehicle were transferred to hospital by helicopter. Ultrasound examinations on the day of the accident showed no foetal abnormality.
- Subsequent antenatal care did not demonstrate any abnormalities.
- A normal vaginal delivery resulted in a female infant with good Apgar scores at a gestational age of 39 weeks 6 days.
- MRI scan (performed on 5 July 2013) shows substantial brain pathology probably caused by previous injury and bleeding.

Discussion:

There are no other known antenatal, intra-partum or postnatal factors present which would conceivably cause the brain injuries as described by Prof Lotz in his report. The pattern of brain damage would correspond to ante natal injury (see, for example, *Prameela Karimi, Ronald Ramus, Jill Urban and Jeffrey M. Perlman: Extensive Brain Injury in a Premature Infant Following a Relatively Minor Maternal Motor Vehicle Accident with Airbag Deployment. Journal of Perinatology (2004) 24, 453 –457*). As such, the most likely cause of the brain damage is the motor vehicle accident occurring at 30 weeks of gestational age.

Signed at Parow on Sunday, 7 June 2015".

It is common cause that the reference to "30 weeks" is an error and that it should read "28 weeks".

11.

Dr Pistorius said that he was a maternal and foetal specialist, which was a sub-speciality of obstetrics and gynaecology. He has specialised in this particular field for the last eight years. It involves the detection of foetal problems and ultrasound scans

are often performed to determine whether these problems or abnormalities exist. His daily practice involves primarily diagnostic work with occasional surgical procedures. He also regards himself as a specialist at foetal brain imaging. He testified that he considered the following when formulating his opinion:

11.1 Photocopies of the clinical notes emanating from the Welkom Medi-Clinic;

11.2 The clinical records of Dr Coleman and his delivery report;

11.3 The MRI scans of C's brain and Prof Lotz's report thereon. (Prof Lotz was the next witness).

12.

It will serve no useful purpose if I simply repeat all the evidence of Dr. Pistorius. The essential parts of his evidence will however be referred to and analysed in the light of the later evidence of Prof Lotz and Defendant's expert witness Dr Okoli, who is a neurosurgeon.

13.

Dr Pistorius testified that C's injury most likely occurred during the second part of the pregnancy and not before 20 weeks gestation and not after her birth. The reason for this was the pattern of damage to C's corpus callosum, as observed on the MRI images of C's brain. The corpus callosum consists of nerve fibres connecting the structures of the left hemisphere of the brain with the right. C's corpus callosum was fully formed which occurs after the 20 weeks gestation. Nevertheless, whilst fully formed, it was thinned. If this condition had occurred prior to 20 weeks gestation, the corpus callosum would be partially or even totally absent and certainly not fully formed. As a result, this condition could not have occurred prior to 20 weeks gestation. In addition, this condition was unlikely to have occurred after C's birth. This was so because C's brain damage had affected her right basal ganglia whereas an "insult" to the brain following directly after delivery of a baby usually affects more superficial areas of the brain. The condition of the corpus callosum, which is thinner, is a secondary result of the damage to the right side of her basis ganglia. The basal ganglia are a set of nuclei controlling muscle movement and muscular development. There are left and right counterparts. The damage to the

right basal ganglia therefore resulted in reduced communication between the brain hemispheres, and the corpus callosum therefore thinned as a result thereof. Accordingly, Dr Pistorius was of the view that because of the focalised destruction of the various structures of C's basal ganglia, the condition resulted from reduced blood supply (ischemia) to that region of the brain.

14.

Dr Pistorius was also referred to the expert summary report of Prof Lotz. He testified that no other known factors could have led to the pattern of damage in question. He testified, also again in the context of Dr Lotz's report that "severe spasm was the most likely pathology". Any "placental insufficiency" would have presented a more global presentation in the brain. 'Cord' damage, would have led to blood shunting to the cerebellum and brainstem and would then also have exhibited a more global presentation. "Direct injury to the carotid vessels" would most likely also have exhibited a more global presentation and in addition thereto there was no clinical

evidence of any bleeding to support this pathology. He said that when a foetus is involved in a high velocity impact the foetus experiences rapid deceleration. Different body parts of the foetus decelerate at different rates and the foetus' head, as a relatively smaller object, decelerates more rapidly than the foetus' body. This rapid deceleration also resulted in sudden flexion of the foetus' neck. A lateral flexion would most likely have been involved since foetuses typically lie with their spines to the side at 28 weeks gestation. It must in this context also not be forgotten that Mrs S testified that her vehicle flipped over after the impact. Blood vessels near the skull then experience stretching and or twisting i. e. flexion/ torsion forces. As a result, arteries become stretched across the skull base. When this happens they go into spasm. There was no need for an extremely strong contraction. This resulted, in the present case, in the localised reduction of blood flow to the right basal ganglia. This led to destruction of brain tissue in C's right basil ganglia which as a result was deprived of oxygen and waste products build up in the tissues. When cells are deprived in this way, they usually die after about five minutes. The cysts that are present in C's brain and are observed on the MRI images are build-ups of cerebral-

spinal fluid, resulting from dead cells having been removed and replaced. The cerebral-spinal fluid is therefore present where the basal ganglia would have been in C's brain, but for the trauma. Dr Pistorius testified that this is a well-documented pathology in medical literature. The corpus callosum of C subsequently thinned as a result of the damage to the basal ganglia.

15.

Dr Pistorius also mentioned the following other important aspects:

15.1 There was no specified threshold of severity of an accident required to cause trauma to a foetus. There was however a positive correlation between the severity of an accident and the likelihood of foetal trauma;

15.2 Part of his training involved research into the causative links between maternal trauma and foetal neurological trauma;

15.3 The high-velocity impact was definitely of sufficient degree to have caused the relevant trauma;

15.4 There was no direct link between reduced foetal movement and foetal abnormalities;

15.5 Ultrasound tests, both prior to – and post – accident would not likely have revealed brain damage;

15.6 Dr Okoli, the neurosurgeon, to be called by Defendant was not suitably qualified or experienced to testify about what the clinical effect of what was found on the MRI image ought to be, and he had no expert knowledge of embryology;

15.7 In the context of the clinical notes of Drs Coleman and Myburgh, he confirmed that there was no evidence of placental problems in utero, nor of any umbilical cord problems or of blood on the brain;

15.8 Any blood products would not likely to have remained in the time span between the accident (10 December 2012) and the date of the first MRI scan (3 July 2013);

15.9 The corpus callosum was fully formed in length by gestational week 20;

15.10 As far as the interpretation of MRI images was concerned, the opinion of an experienced neuro-radiologist like Prof Lotz should be deferred to;

15.11 Dr Okoli lacked experience in foetal development and he therefore incorrectly applied his experience in adult brains to the field of embryology. His own expertise in pre-natal persons was more refined and focused than that of a neurosurgeon. His own daily practice involved the development of the foetus in uterus, and the medical literature supported his view;

15.12 The corpus callosum of a foetus is more susceptible to thinning as a result of right basal ganglia damage than of an adult, because the foetus has yet to develop myelin, a sheath of fatty tissue surrounding the nerve fibres. This phenomenon was well-documented in scientific literature;

15.13 C's clinical presentation, i. e. whole-body spasticity was reconcilable with the MRI evidence that only her right basal ganglia were compromised;

15.14 If the defect was congenital then both sides of the brain would have been affected. Here the MRI showed that the basal ganglia were partially absent on one side. Dr Okoli's scenario was therefore improbable. On his version

the relevant vessels would have had enough blood supply for a certain period of time, and would then have stopped functioning without an accident, which was improbable.

16.

Prof Lotz:

Prof J. Lotz has the following related qualifications:

MBChB in 1972. He obtained the MMed, Master of Medicine, in diagnostic Radiology (*Cum Laude*) in 1980. He is a Fellow of the College of Radiology of South Africa and London. In 1993 he obtained the MSc (Med) in Anatomy. (Unfortunately I did not ask him why he also obtained a Masters degree in History in 1997). He has vast professional experience and since 2005 has been Professor of Radiology (Academic) at the University of Stellenbosch. His medico-legal practice has been similarly impressive and I quote from his CV:

"2007 – 2014

- Specializing in medico legal assessment of MRI features of hypoxic ischemic injury in pre-term and term infants.
- On-going international correspondence with leading experts in the field of hypoxic ischemic injury in infants.
- Compilation of a database of more than (500) cases of hypoxic ischemic injury in infants in a chronic stage of evolution. Comparative figures not available, but assumed to be one of the largest in the world.
- On-going academic evaluation of database and development of didactic teaching material in the field of hypoxic ischemic injury in children.
- Support of research in the field of hypoxic ischemic injury by affording prospective PhD candidates access to the unique database.
- Active in addressing the unique issues and problems of hypoxic ischemic injury in children in the South African context.
- Active in propagating an ethical, scientific and financially sustainable national plan to address a major cause of significant mortality and morbidity in the neonatal population.

- Expert opinion on concluded and on-going civil actions relating to hypoxic ischemic injury in the neonate in an estimated 250 cases."

17.

Prof Lotz studied the relevant MRI images and observed the following:

17.1 The child had focalised brain damage in her right basal ganglia;

17.2 Three of the nuclei of the right basal ganglia were either damaged or destroyed. These were the caudate nucleus which was damaged, the lentiform nucleus – which is destroyed, and the thalamus, a portion of which was destroyed. This indicated that this condition could only have been a vascular problem which occurred at the same time. He could not think of a condition that would do this gradually at all angles over days. There was no embryological basis for the absence of these nuclei, and text books supported his view (Slide 3);

17.3 The corpus callosum was thinner than normal, and

17.4 There was evidence of border-zone infarction in C's brain;

17.5 If this was a congenital condition, it had to be asked why only three nuclei were destroyed, and all at the same time? He could not think of any condition that would do this gradually.

18.

The commonality between the three nuclei was that they shared a common blood supply. The portion of the thalamus which remained alive was however supplied by a different vascular system than the other nuclei. As a result, the only objective and logical explanation for the pattern of damage in C's brain was that a vascular problem had affected the vessels. An intra-uterine traumatic event was the likely cause of this pathology. He emphasized that his findings were based exclusively on his consideration and interpretation of the MRI images, in which he was a specialist.

19.

When he compiled his medico-legal report, he was aware of the occurrence of the accident. In the report he stated that there was no evidence of genetic or congenital anomalies. The "discussion" part of his report dated 18 March 2015 and based on the MRI scan of 11 March 2015, read as follows:

- "The pathology is centred in the anterior vascular territory in middle and anterior cerebral arterial distribution area, more so on the right. Bilateral border zone infarction and basal ganglia involvement is consistent with a profound hypoxic ischemic event, most likely the result of hypotension;
- The pattern of destruction is not that of periventricular leukomalacia of prematurity or of hypoxic injury of the term infant;
- Anterior distribution territory suggests that the carotid arteries were involved. The scan fails to identify a definitive cause, but placental insufficiency, cord damage (both with posterior circulation redistribution), direct injury to the carotid vessels or severe spasm are reasonable and logical considerations;

- It is my considered opinion that on a balance of probabilities, an intra-uterine traumatic event to have been the most likely cause for this extensive brain injury, now in a chronic stage of evolution".

20.

It is clear from the third unnumbered paragraph of this discussion that he referred to four "reasonable and logical considerations" as to how the particular damage was caused. He testified that he mentioned these only for the purpose of a differential diagnosis. In the present instance, the clinical evidence ruled out three of these, and the only mechanism that he found to be the most probable cause was "severe spasm". Because a specific area of the brain was affected only, the event had to be vascular, and there was no other logical explanation. There was no embryological basis for the condition seen on slide 7 of the brain, i. e. the absence of certain nuclei. One could not find such a congenital condition in the text books.

21.

Prof Lotz further testified that it was highly improbable that the three nuclei in C's brain were damaged at three separate times. Damage caused on three separate occasions would then most likely be the result of an embolic disease, but this would have meant that three separate embolisms would have had to travel the same vascular pathway to the right basal ganglia, which was, to him, highly improbable.

22.

Explaining the reason for his conclusion as to the cause of C's damaged brain, he testified that one needed to consider the auto-regulatory system of the brain in this context. This was a system whereby adequate blood supply to various parts of the brain is ensured. Blood vessels had the ability to contract and dilate and influence blood flow to the brain. When a blood vessel was subjected to stretching and/or twisting forces, it had a tendency to go into spasm. Such spasms could last for short

periods, or a few hours up to some four days even. In C's case the spasm must have lasted for at least half an hour, but more likely even several hours.

23.

In C's case, the corpus callosum was fully formed at 20 weeks gestation. It had assumed its final shape, but lacked in thickness. Her brain must therefore have been injured between 26 and 34 weeks gestation, given this presentation. The condition of her corpus callosum could not have been caused by a congenital defect given that it is fully formed, but thin. This thinning was the consequence of damage to her right basal ganglia. He had never seen a pattern caused by a congenital defect where the nuclei on one side only were destroyed.

24.

Prof Lotz also referred to heterotopia. He defined this as a condition where cells did not migrate to their normal position in the brain, and concluded that he did not find any congenital, genetic or inflammatory pathology and no evidence of heterotopia.

25.

He also testified that C suffered border-zone infarction. This meant that there had been a transient drop in blood pressure in her brain. The damage to this border-zone was less severe than the damage to the nuclei in her basal ganglia. This border-zone infarction was of relevance in that it provided a larger picture of what had occurred in her brain. Where there had been a blockage of blood vessels that supply the basal ganglia, this resulted in a drop of pressure in the brain and affected the right border-zone. The pattern of injury which I have described and which he saw on the MRI images was not consistent with the presence of a congenital defect. He testified that there were over 120 000 congenital defects, and he saw no evidence for any of them. As far as the mechanism of an injury was concerned, he testified that indirect forces could cause injury to blood vessels, and added that a sudden deceleration of the foetus inside the womb led to it experiencing a multitude of indirect forces. He added that the two classical works on neonatal neurology (by *Wolpe and Markowitz*) did not contain reference to a single defect that could cause

such an unknown or unnamed congenital defect. The crux was that C's corpus callosum had been fully formed, although it was thin.

I must add at this stage that Defendant tendered the following formal admissions during the trial and before Dr Okoli gave evidence:

25.1 At 28 weeks gestation, cerebral arteries are capable of constricting and causing spasm;

25.2 The corpus callosum can further develop in thickness after 24 weeks gestation;

25.3 C's MRI images showed a border-zone infarction. It was initially put to Prof Lotz that there was no border-line infarction. Prof Lotz replied that Dr Okoli needed to explain the presence of white material on one MRI slide (Slide 6).

26.

The experience and expertise of Dr Okoli was put to him for comment. He testified that he had never heard of a neurosurgeon being involved in an ante-natal case.

Neurosurgeons were better suited to comment on post-natal brain conditions. As far as his own expertise and experience were concerned, he repeated that a radiologist had to undergo five years of training to qualify and that neo-natal radiology was an even further specialization. He added that even neurosurgeons specializing in paediatric cases generally defer to the opinion of a radiologist regarding brain imaging. Dr Okoli was not trained to interpret an image of a natal brain. He noted from Dr Okoli's CV a paucity of paediatric and neo-natal experience as well as radiological experience and their fields of expertise were widely apart. He obviously did not intend to negatively comment on Dr Okoli's experience or expertise as a neurosurgeon. It was put to him on behalf of Defendant that Dr Okoli would agree that in C's case a disruption of the cerebral artery had occurred. It was also put to Prof Lotz that Dr Okoli would not agree with his conclusion that "severe spasm" was a probable cause of C's brain condition, but he maintained that he saw no other reason for the pattern of the damage. He added that Dr Okoli could not form an independent opinion in a field that was highly specialised. A neurosurgeon would

have no knowledge of ante natal damage and he should not have given evidence on this topic.

27.

Dr B. Okoli:

Dr Okoli is the holder of a Bachelors of Medical Science degree (Human Anatomy), which he obtained in 1986 at the University of Port Harcourt in Nigeria. He also obtained a Bachelor of Medicine degree at that University in 1989 and it is a "Bachelor of Surgery". In 2000, he obtained the degree Master in Medicine (Neurosurgery) from the Medical University of South Africa. He has practiced as a specialist neurosurgeon since 2001.

28.

His medico-legal report is dated 15 March 2016. He examined C, and stated the following: She is obviously neuro-physically and mentally retarded. She has

dysmorphic facial feature. She has poor neck control, poor trunk control and poor limb control. She has spastic limbs. He stated that C's milestones have been delayed, and it is obvious that she suffers from cerebral palsy. Under the heading "The implications of the MRI reports are as follows", he mentioned the following:

- "There was no haemorrhage in the brain of C either intra-uterine or extra-uterine.
- There are radiological features that are suggestive of ischaemic phenomena.
- Almost all the ischaemic features are within the territory of blood supply of the anterior cerebral artery.
- The pathological findings in the brain have not involved the back part of the brain called the cerebellum, so it is not all of the brain that is involved in this pathology.
- There is evidence that not all the MRI findings can be explained on the bases of global ischaemic phenomena at 28 weeks of gestation.

- There is disorder of neuronal migration with grey matter heterotopia that has failed to reach their final destination from their place of birth which usually occurs between 6 to 16 weeks of gestation.
- The part of the corpus callosum that is particularly affected is the body of the corpus callosum.
- Embryologically the corpus callosum is fully formed and has assumed its final shape by 18 to 20 weeks of gestation.
- If only the body of the corpus callosum is hypoplastic, it cannot be explained on the bases of a global ischemia that occurred at 28 weeks when it is expected to have been fully formed."

29.

It is also important to refer to his "Deductions" in that report, which were the following:

- “The history of the motor vehicle accident does not suggest a direct blow to the abdominal wall.
- There were no abdominal bruises.
- The probable pathway of involvement of the abdomen is the tightening or traction of the seatbelt against the abdominal wall.
- This caused an excitatory irritation to the abdominal/uterine muscle with a resulting contraction.
- The degree of uterine contraction does not seem to have been severe and could not have caused hypo-perfusion injury through the placenta.
- A hypo-perfusion injury through the placenta should affect the foetus and is expected to elicit foetal distress.
- This was absent throughout her period of observation and care.
- The ultrasound examination after the MVA did not reveal placental injury or disruption.
- The ultrasound evaluations after the MVA also did not reveal a blood clot in the brain.

- C has neurodevelopmental abnormality in the MRI images of the brain that could only have resulted from an arrest prior to the motor vehicle accident in question and this specifically refers to the neuronal heterotopia and hypoplasia of the body of the corpus callosum.
- The region of blood supply from the left and right anterior cerebral arteries bear the brunt of the brain damage.
- It is highly improbable that this trauma has selectively damaged these arterial vessels with the exclusion of other arterial vessels in the brain.
- It is also all the more improbable that a global hyper-perfusion injury will selectively affect the left and right anterior cerebral artery.
- Most likely, there is a malformation involving the anterior cerebral arteries which are unrelated to the motor vehicle accident under considerations.
- Conclusion: I am unable to associate the radiological pathology in the brain to the motor vehicle accident of 10 December 2012 when C was intra-uterine aged 28 weeks".

30.

Dr Okoli testified that he dealt with neo-natal cases with congenital malformations at least once every two weeks or so. From time-to-time referrals are made to him where the patient has a surgically correctable brain legion. It is common cause that C has cerebral palsy. Dr Okoli however testified that in his opinion a radiologist cannot diagnose cerebral palsy since it was a clinical diagnosis which emanated from an examination of the particular patient. He did however confirm that a MRI scan could provide a reason for such palsy. He was also capable of interpreting MRI images which he sees in practice every day. He did not agree with Plaintiff's case on the basis that C experienced trauma in utero. He gave evidence about the make-up of the basal ganglia, which are a collection of neurons primarily responsible for "supple movements". He also referred to the slides provided by Prof Lotz when he gave evidence, and testified what he saw on these images. He said that the tissues of the basal ganglia on the left side of C's brain were fully developed, but not the right side. The under-developed nuclei of the right basal ganglia were simply smaller than those on the left, and there was in his view no MRI evidence that the nuclei on

the right were ever damaged. The lacunae in the brain on the right basal ganglia region, where they should have been fully developed nuclei, are instead filled with cerebral spinal fluid and no gliosis is present. (Dr Pistorius testified that gliosis is a proliferation of microglia, which are “scavenger” cells in the brain that remove damaged or dead cells. Dr Okoli did not agree with that part of Dr Pistorius’ evidence in that brain tissue which is dead does not “disappear” in the brain, but instead gliosis remains present. This phenomenon could be seen, and was so seen by him, regularly in stroke victims, and he observed MRI evidence of gliosis every second day. He referred to a well-known text book read by every neurosurgeon, namely ***Neurological Surgery, 5th Edition by H. R. Winn***. This assessment was based entirely on his interpretation of the MRI images of C’s brain. He added that this pattern of damage was indicative of a congenital anomaly that resulted in the under-development of the nuclei of the right basal ganglia, and not that of ischemia. He also admitted that there was evidence of border-zone infarction in C’s brain, which Prof Lotz had described in some detail. He also agreed with Prof Lotz that the corpus callosum in C’s brain was fully formed in length, but not in thickness. I asked

Dr Okoli what, in his view, had caused the thinness of the corpus callosum and he stated that it was “under-formed”. Defendant’s Counsel, prior to Dr Okoli’s evidence, had conceded that at 28 weeks gestation, cerebral arteries were capable of constricting and causing spasm. During his evidence he qualified this concession by stating that blood vessels acquire “optimum ability” at a gestational age of 34 weeks, and that he did not think that a foetus’ blood vessels could contract optimally at 28 weeks gestation. It is not clear whether the reference to “optimally” was intended to detract from the previous concession made. Regarding the mechanism of the injury, he, as was common cause, referred to the high velocity head-on collision. In this context he said that the foetus experienced linear forces. A direct force on the foetus would be applied by an object such as the dashboard or safety belt. In the case of indirect forces, the foetus’ head would hit the wall of the mother’s womb and for a brain injury to follow from this, the brain would be “rattled” in the foetus’ skull. In both cases, he said, the result would have been brain bleeding and brain swelling. This would be evident on an ultrasound. No such evidence existed of this in C’s case. I must add at this stage that Dr Okoli was not

aware of the fact that Mrs S had testified that her vehicle had flipped onto its side after the collision. In the context of the "linear forces" that he referred to, he stated that a sudden deceleration force could not cause injury to the blood vessels in the brain without there being an avulsion from the brain of those vessels, i. e. that the vessels would have "pulled off" from the surrounding brain tissue. He further said that if the internal carotid artery experienced stretching or twisting, this would invariably result either in brain bleeding or an injury to the inner-lining of the blood vessels causing an embolism. There was no evidence of such an embolism.

31.

While he was giving evidence, Dr Okoli produced a copy of a passport photo of C and said that her eyes were asymmetric and that her forehead was irregular. In his report under the heading of "Examination", he had stated that C had "dysmorphic facial feature". Neither Counsel delved into this observation at the time, and as a result of Dr Okoli's production of the passport photo, I deemed it in the interests of justice and of the child, not to leave this topic hanging in the air as it were, but to

re-call First Plaintiff and also Dr Pistorius. It is clear from the provisions of s. 6 of the *Children's Act 28 of 2005* that all proceedings in a matter concerning a child must respect, protect, promote and fulfil the child's rights set out in the *Bill of Rights*. The relevant section in the *Constitution of the Republic of South Africa* is s. 28 which states in s. 28 (2) that a child's best interests are of paramount importance in every matter concerning the child. It seemed to me that the Defendant's Counsel regarded my decision in this context as "unusual", but it must be remembered that a Court has a discretion to re-call a witness for further examination or cross-examination, also in civil trials.

See: *Herbstein & Van Winsen, The Civil Practice Of The High Courts South Africa, 5th Edition, Vol 1, at 899*.

Quite apart from that, it is my view that where the interests of children are at hand, a Court should take the best interests of the children into account and, as in the present context, re-call a witness and ask pertinent questions to ensure that topics that may appear to be particularly relevant are not left hanging in the air, because of Counsel's oversight, inexperience or lack of appreciation of the importance of the

particular topic. For the same reason, I also deemed it in the interests of justice, and in the interests of the child, to re-call Dr Pistorius. I will deal with this evidence hereunder. At this stage Dr Okoli added that C's dysmorphic face, as he described it, could not be explained on the basis of trauma. It could only be explained on the basis of a congenital defect. Had this been otherwise, there would have been fractures to C's skull which there were not. He also said that congenital malformation of the occipital bone could have caused the asymmetry of the eyes. The foetus' face was fully developed at 28 weeks gestation and as a result the facial malformation could only have been caused either by a fracture or a congenital defect.

32.

Dr Okoli also was of the view that C's physical symptoms, namely spasticity on both sides of her body, did not accord with the condition of her right basal ganglia in that he would have expected her to have spasticity in only the left side of her body, given that only her right basal ganglia are abnormal. He was therefore of the view

that C presents with symptoms that accord with global generalised brain problem indicative of a congenital defect. Furthermore, having regard to the clinical notes of Drs Coleman and Myburgh, he testified, as they did, that there was no evidence of foetal distress and as a result he said the foetus could not have experienced trauma.

33.

Cross-examination of Dr Okoli:

Dr Okoli conceded that a maternal and foetal specialist was more qualified to comment on issues concerning foetal development than himself. He argued however that the issue at hand was more about the brain structure. It is common cause that Dr Okoli does not have a degree in radiology. He said however that radiology was part of the neurosurgical training and that he had looked at “uncountable” number of scans. He conceded at the same time that a radiologist who specialises in neonatal brain-imaging was more qualified than himself to provide an interpretation of MRI images of a neonatal brain. In the present context this concession was correctly

made and it is of importance, in regard to the qualifications and experience of Prof Lotz. He was asked about his practice in the context of diagnosis of congenital defects and he testified that obstetricians and paediatricians often approach him after they had made a diagnosis involving congenital defects and where a surgical correction was an option. In some of these cases however he was also involved in diagnosis.

34.

He considered the MRI images of C's brain during the process of compiling his mentioned medico-legal report. As far as C's corpus callosum was concerned, he conceded that it was fully developed in terms of its structure, but that it was thin. This, in his view, was an inherent condition that C was born with. The reason for the thin corpus callosum was exclusively a congenital defect which she would have had prior to 20 weeks, while in utero. The reason for this firm conclusion was that there was no evidence of intra-uterine trauma and no evidence of dead tissue on C's MRI images. He was then asked whether he considered that the thin corpus callosum

could have been a secondary effect, and answered in the negative. However, he added at the same time that this thinning could not have been caused by a loss of blood supply. Counsel for Plaintiff put to him the Plaintiff's actual explanation for C's thin corpus callosum, namely that it was a consequence of the damage to her right basal ganglia, and the subsequent loss of communication between her right and left basal ganglia. Dr Okoli was of the view that the thinning did not follow from basal ganglia destruction. He had often observed stroke victims who had suffered brain damage to one brain hemisphere without subsequent corpus callosum thinning. In essence, he remained of the view that the thinning was the result of a congenital defect, because he did not find gliosis on the MRI images and because he deemed the loss of blood supply to the corpus callosum highly improbable. His conclusion therefore was that C's corpus callosum had fully developed in its proper proportions and had then subsequently thinned and that he attributed this to a congenital defect. Prof Lotz had testified in this context that he did not know of any congenital defect that would have caused this particular result. Dr Okoli however was of the view that the defect was "hypoplasia" and that he had seen such frequently. According to him,

hypoplasia was an under-formation. Examples of congenital defects that caused corpus callosum hypoplasia could be found in medical literature and he had experienced this as well. Dr Okoli referred to an article in the 'Neurology' magazine of the American Academy of Neurology of 2011, headed "Distinguishing 3 classes of corpus callosal abnormalities in consanguineous families". He did not know of the authors and could say nothing about their expertise. This article had also not been put to the Plaintiff's expert witnesses for comment. The topic discussed therein is contextually also totally different. Dr Okoli should have been referred to the *Journal of Perinatology (2004)*, mentioned by Dr Pistorius for comment, but was not.

35.

In his report, Dr Okoli referred to an abnormality in the MRI images of the brain "that could only have resulted from an arrest prior to the motor vehicle accident in question". During cross-examination he elaborated on that by stating that he made this observation because of the mentioned under-development of the corpus

callosum which by the gestational age of 20 weeks would have been fully formed. The “arrest” must have occurred prior to 20 weeks gestation. It was then put by Plaintiff’s Counsel that his report and his evidence were inconsistent in this respect inasmuch as in his report he made mention of an “arrest” that must have occurred prior to 20 weeks gestation, whilst in his testimony he attributed the thinness to an inherent congenital defect. He denied that this was an inconsistency and suggested to Counsel that the whole picture be considered inasmuch as the particular malformation could not be explained on the basis of global ischemia. He was of the view that when a foetus experienced brain damage at 28 weeks, gliosis would be apparent on MRI images. He then further testified that he had observed heterotopia on the MRI images. This was a neural migration problem and was related to malformation. This would not have been apparent on a routine sonar scan. He regarded his observation of heterotopia on the MRI images as very important regarding his conclusion that C had a congenital defect, since it was indicative of the fact that not everything had developed. He however later on testified that the presence of heterotopia was only one factor that he considered in concluding that C

had a congenital defect. I may mention at this stage that both Dr Pistorius and Prof Lotz had seen no evidence of heterotopia. He also excluded the possibility of a vascular injury, because in such an instance the pattern of brain damage would have been more generalised and less focal than it was. Prof Lotz's explanation of the pattern was put to him, namely that the single unifying commonality that the damaged or destroyed nuclei in the basal ganglia share, is that they are all supported by the same vascular system. Dr Okoli however persisted in his view that the pattern of brain damage could not have been induced by trauma.

36.

Prof Lotz had testified in some detail about the presence of a border-zone infarction on the MRI images, and Dr Okoli conceded that there was evidence of such. Prof Lotz said in this context that the presence of a border-zone infarction provided a bigger picture of what had happened in C's brain, more particularly that there had been a transient loss of blood pressure. Dr Okoli's view in turn was that the

particular infarction could not be explained on the basis of a right middle side artery involvement.

37.

As far as the mechanism of the injury was concerned, he conceded that blood vessels could react to indirect forces. Dr Pistorius' opinion in this context was put to him, but Dr Okoli disagreed therewith for the following reasons:

37.1 The foetus would not have experienced any complex vertical or twisting forces;

37.2 The force exerted on the foetus would most probably have been linear in a head-on collision, which would have impacted directly on the wall of the womb.

I have already mentioned the undisputed evidence of Mrs S that her Toyota had flipped onto its side after the high impact collision. Dr Okoli was, and had been, unaware of this evidence, but did not offer an opinion whether this fact would change his view relating to linear forces. Prof Lotz had testified that no-one could

describe the mechanics after the impact exactly. He favoured a scenario including tortion after an indirect force which would not all have been on the same plane.

38.

As far as spasm in blood vessels was concerned, he had conceded that at 28 weeks gestation these vessels had the ability to contract. He also conceded that Dr Pistorius' evidence that smooth muscle developed in the vessels in the foetus' brain by 22 weeks gestation was correct. He agreed that most babies born prematurely from 20 weeks gestation onward survived. He stated that this was so, because their organs were sufficiently developed. He stated that he was not sure that this capability to survive must also be due to sufficient smooth muscle development in the brain's blood vessels, and because they had a functional auto-regulatory system. I have already mentioned that it was put to him that smooth muscle at 28 weeks gestational age could contract sufficiently to prevent blood flow. His view was that vessels at this stage could not "maximally" contract. He then proceeded to qualify this concession by stating that there was a degree of development and that

smooth muscle development was maximally optimal at approximately 34 weeks gestation. Prof Lotz's opinion was put to him, namely that a vessel spasm could cause injury to the inner-lining of the vessel which could result in an embolism which would subsequently block smaller vessels in the brain. According to Dr Okoli this was unlikely, because any embolism would flow upward.

39.

It was also put to him that the presence of a congenital defect in C was never mentioned in his medico-legal report. He conceded this, but said that the word "malformation" indicated his consideration of a congenital defect. This congenital defect "may" involve malformation of the anterior cerebral artery. Dr Pistorius' evidence was put to him, namely that a congenitally malformed artery would cause on-going brain damage whilst the pattern of C's brain damage was indicative of a transient event. It was therefore highly improbable that such a congenital condition would cause transient vascular problems. Dr Okoli testified on this topic that the

condition was one of under-development of the brain structures and that supply of blood was sub-optimal at the outset of C's development. He drew this inference on the basis that there was no evidence of basal ganglia damage. He conceded that he could not name the particular congenital defect, but stated there were in excess of a 120 000 possibilities in this context. He did not comment on Dr Pistorius' evidence that signs of foetal distress do not necessarily accompany foetal injury.

40.

C's "dysmorphic" facial feature (facial deformation):

Reference to this dysmorphic facial feature appeared in Dr Okoli's report, but it was scarcely dealt with, either in examination, or cross-examination, until Dr Okoli produced C's passport photo which he had obtained from his secretary as a matter of routine. He testified that the facial asymmetry of C's eyes was a result of a generalised muscle imbalance. A bone fracture, if there had been one, could have healed in deformity, as he put it. He conceded that only a mild deformity was

present. Dr Pistorius, who was re-called by me, gave a different opinion. He said that C's eye-asymmetry was merely a squint of the left eye because of the muscular imbalances in her entire body, including her face. When Mrs S was re-called by myself, she testified that she had not observed any such facial deformity at the time of C's birth and I need to note that neither Dr Coleman, nor Dr Myburgh made any reference thereto. At the time of this evidence C was sitting on Mrs S's lap and I made my own observations. I did note a squint in the left eye and I also noticed that when C smiled at me repeatedly, such smile was not uniform. I could not see any irregularity in C's forehead and when this was put to Dr Okoli he stated that there was only a slight or mild deformity in this context, something that I could not see. Dr Pistorius' view was that C's "lazy" eye and the boney facial asymmetry was a particular feature of cerebral palsy and that there was no evidence that there had been any boney malformation in her face or skull which could have healed in deformity as Dr Okoli had said.

41.

Plaintiff's argument:

41.1

In summary, it was contended that it stood to reason that Dr Pistorius' and Prof Lotz's reasoning should be preferred to that of Dr Okoli. The former considered two parts of the brain in their commonality, whereas the latter only considered two "obstacles" that were not relevant to Plaintiff's explanation of C's thin corpus callosum.

41.2

Dr Okoli's submission that the thinning of C's corpus callosum was the result of a congenital defect should be rejected. When Prof Lotz said that there was no known congenital defect that would produce this result, Dr Okoli contended that the defect was "hypoplasia". This however, is not a defect, but a symptom. Dr Okoli himself defined it as "under-formation or sub-optimal formation which results in something being incompletely formed". Essentially therefore, what Dr Okoli stated, was that the congenital defect that caused C's corpus callosum to thin, was "thinning". When this

was pointed out to him during cross-examination, so it was contended, he could not name a single congenital defect that causes part of the corpus callosum to develop fully, and another part to under-develop. Even when it was put to him that Dr Pistorius and Prof Lotz had testified that they had never heard of such a congenital defect, he was unable to provide a single name to describe such a defect, even as a mere possibility. In this context, it was therefore contended, that his answers were vague, evasive and non-committal.

41.3

Prior to Dr Okoli's testimony, Defendant conceded that muscles were sufficiently developed at 28 weeks gestation to cause the arteries to go into spasm. This was a contradiction to what was put to Dr Pistorius during cross-examination and was indicative of the fact that Dr Okoli changed his opinion in this regard during the cause of the trial. Despite this concession, Dr Okoli further testified that he did not believe that muscle spasm could have caused loss of blood supply in C's case, because at 28 weeks gestation, the vessels could not have "maximally" contracted.

Dr Okoli held this view despite having conceded the following points emanating from Dr Pistorius' testimony that:

41.3.1 Smooth muscle develops in the vessels in the foetus' brain by 22 weeks gestation;

41.3.2 Dr Pistorius regularly measures the blood flow in foetuses using ultrasound scans, from which he can infer that smooth muscles in the blood vessels are developed;

41.3.3 Most babies born prematurely from 26 weeks gestation onward survive.

41.4

Despite making the above concessions, Dr Okoli resisted the ineluctable conclusion that at 28 weeks gestation, it was indeed possible for a foetus' blood vessels to go into spasm and arrest blood supply. It was contended that this was indicative of both bias and paucity of experience and knowledge in the field of brain development.

41.5

Dr Pistorius and Prof Lotz also testified that a rupture of the blood vessels in the foetus, as well as the damage to the tissue inside the blood vessels, will only occur

automatically in case of direct force applied to the vessels. They also explained that vessels reacted differently to indirect forces. These were the basic principles of medicine taught at the third and fourth year of medical school.

41.6

Prof Lotz testified that in C's case, the spasm probably lasted for several hours, given the extent of her brain damage. There was no collateral/factual evidence that contradicted this view if one takes into account the time when the accident occurred and the time when the first ultrasound was done in hospital, some hours after the accident.

41.7

It was therefore submitted that the logical medical explanation was that spasm in the mid-cerebral artery caused the loss of blood supply to C's right basal ganglia.

41.8

Dr Pistorius and Prof Lotz provided two reasonable and logical explanations on how the mechanics of the spasm injury or "intra-uterine traumatic event", would have

operated following the foetus' sudden deceleration caused by the high-velocity impact. Prof Lotz testified that the exact specifics of an indirect force can be countless, and that there was a multitude of possible indirect forces that could influence the reconstruction of the mechanics of injury. Dr Okoli considered only a forward movement as the most probable indirect force, but seemed to change his opinion after he was informed that the motor vehicle had turned to its side after the impact. He however had no firm view about this and did not deal with it. Dr Pistorius also said that head and body react differently to deceleration, because of their different weight.

41.9

It was therefore contended that the evidence of Dr Pistorius and Prof Lotz on the probable mechanics of the injury, took cognisance of the multitude of ways in which indirect forces could have operated. There was no reason why their explanation could not be regarded as logical and probable. From a medical perspective, these indirect forces could indeed have caused vascular spasm, and that spasm could indeed have caused constriction of blood supply to C's right basal ganglia.

41.10

The evidence of Drs Coleman and Myburgh did not change the conclusion of Dr Pistorius that the brain damage, as seen on the MRI scan, was in all probability caused by the accident that occurred at 28 weeks gestation.

41.11

As far as the alleged congenital defect relied upon by Dr Okoli was concerned, it was submitted that he could not name any particular defect in this context, but merely referred to a congenital defect that caused malformation of an artery which caused loss of blood supply and border-zone infarction. He also mentioned a congenital defect that caused partial under-development of the corpus callosum, whilst denying it to fully develop in form. He also said that it could be a defect that only affected basal ganglia on one side of the brain, and further mentioned a congenital defect that caused facial dysmorphia. It was put to him that the characteristics of such congenital defects were unknown to Dr Pistorius and Prof Lotz, and were excluded by the two “Bibles” of neo-natal neurology. Nevertheless, Dr Okoli could not identify a single congenital defect from a text book even as a

possibility. This was so even after it was put to him that his evidence implied that there could be more than one type of congenital defect that affected C. It was therefore submitted by Plaintiff's Counsel that one would reasonably have expected Dr Okoli, if he was an expert on this topic, to at least have identified one probable congenital defect. The result was that Dr Okoli's assertion in this context should be rejected on the basis that it was vague, impossible and at best forms part of a variety of any one out of 120 000 possibilities.

41.12

As far as facial deformity is concerned, it was submitted that Dr Okoli greatly exaggerated the extent of this deformity. During his evidence-in-chief, he created the impression that the deformity was substantial and that, other than a congenital defect, a bone fracture would have been required to cause such deformity. When C was seen in Court, during Mrs S's re-call, it became apparent that C's forehead was not irregular. These observations were made, both by myself and Dr Pistorius, and were a far cry from the "dysmorphic facial features" that Dr Okoli had referred to. It was also contended that once C had been seen by myself in his presence, and

in the presence of Dr Pistorius, and C's mother, he contradicted his evidence-in-chief. During that evidence, he had said that "a congenital defect of the occipital bone" was the likely cause of the asymmetry of C's eyes. During cross-examination he said that generalised muscle imbalance and not the boney problem might be the cause of the alleged deformity. He also stated that a bone fracture could have "healed in deformity". This contradicted his evidence-in-chief in which he testified that the accident could not have caused the alleged deformity, because there was no evidence of a bone fracture. During cross-examination he also conceded that the milder the deformity, the more other explanations for the alleged deformity may exist. Dr Pistorius' opinion that C's "lazy" eye and expressive facial asymmetry, both of which were attributable to muscular problems, was a feature of her cerebral palsy, and that this was a far more likely explanation for any asymmetry in C's face, since there was clearly no evidence at all that there had been any boney malformations in her face or skull.

41.13

Dr Okoli's explanation that the clinical diagnosis of whole body spasticity does not match the findings on the MRI scan was illogical. The argument implied that there must be something further than what one sees on the MRI scan that caused this whole body spasticity. Dr Okoli then resorted to the congenital defect as being an explanation, without explaining how that would operate in the body of C in order to cause spasm on both sides. In this context it was contended that Dr Pistorius' evidence was more logical. He testified that the damage to the side of the basal ganglia would cause spasticity to the left side of the body, but that this spasticity would cross over to the other side of the body and would cause less severe spasticity to the right side. It was submitted that this is by far the more probable and logical explanation that reconciles C's clinical diagnosis with the damage one saw on the MRI scan.

42.

In summary it was therefore contended that Dr Okoli's evidence, that C's face was deformed, was entirely exaggerated, illogical and improbable. The discrepancies

between his evidence-in-chief and his responses during cross-examination, led ineluctably to the impression that he sought further reasons to justify his opinion that C had a congenital defect. He therefore assumed the role of advocate and was intent to convince the Court that his theory on congenital defect was correct. Once independent observations of C's facial features were recorded, he was forced to change his opinion and make certain concessions. This brought his objectivity into question.

43.

As I have said herein above, my own observations in Court of C's face, in the presence of Dr Okoli and Dr Pistorius, do not show a clear asymmetry. I could also notice no malformation of the skull or forehead.

44.

Defendant's argument:

The crux of Defendant's argument is that C's whole body spasticity and paraplegia are neurologically not compatible with a one sided basal ganglia injury and a limited injury to the corpus callosum. It was contended that Plaintiff's case advanced no relevant explanation in respect thereof and in fact, ignored it. It was also contended that in asking whether Plaintiff discharged the onus resting on it on a balance of probabilities, it failed to present an objective and acceptable description of the probable manner in which C might have been injured in the motor vehicle collision. Plaintiff's experts were also criticized for failing to place an objective consideration of all relevant facts before me. It was said that Plaintiff's experts had prepared only limited expert summaries, had deviated from them extensively and had only given selective evidence regarding the topic of arterial spasms. No probable mechanism of the brain injury was allegedly put forward and it was contended that a number of probable explanations were tendered by them.

Defendant's Counsel presented a very detailed written Heads of Argument for which I am grateful, as I am for Plaintiff's written Argument, and the following material submissions were made:

45.1

C's actual condition, as I have said, namely that of whole bodied spasticity, paraplegia and mental retardation, does not fit in with the one-sided basal ganglia injury. Dr Okoli had said that the basal ganglia controls opposing sides of limb movement. Whole body spasticity can only fit in with a more generalised brain injury than that described by Plaintiff's experts;

45.2

Dr Pistorius, according to Defendant's Counsel, expressed an immediate bias towards Dr Okoli, especially relating to the question whether he was capable of making a diagnosis of the cause of C's cerebral palsy. Prof Lotz in turn, so it was contended, expressed himself outside the field of his own expertise on numerous occasions. (It must however be remembered that Dr Okoli himself had conceded

during cross-examination that Prof Lotz was more qualified than him to consider radiological examinations and investigations.);

45.3

Despite this concession, Dr Okoli was experienced enough to have relied on his own interpretation of the relevant MRI imaging, and his conclusion was that the structural problem identified on such images could not be explained simply by reference to "trauma";

45.4

Dr Okoli had also conceded that in respect of neo-natal development, Dr Pistorius was better qualified than himself, but nevertheless, because of his practical experience, he was qualified to come to the conclusions that he did;

45.5

Exhibit "J" contained various images of C's brain and also depicted normal brain sketches. The crux of Dr Okoli's opinion in this context was that there was no scarring present in the brain which indicated that the relevant parts never developed. In the absence of gliosis (scarring), the relevant nuclei were under-developed;

45.6

The under-developed middle section of C's corpus callosum related to hypoplasia due to a congenital defect. Dr Okoli said in this context that he saw hypoplasia probably twice a year only. The crux really was, as I have already said, that because there was no gliosis present, malformation was indicated. If, according to Plaintiff's thesis, a reduction in blood flow due to a spasm that had been present, more areas of the corpus callosum would have thinned out;

45.7

The probability of an arterial spasm having occurred could not explain the very focal brain injury, the whole body spasticity, paraplegia as well as mental retardation;

45.8

Dr Okoli had also conceded in cross-examination that in respect of foetal development, Dr Pistorius was better qualified than he was;

45.9

Despite these concessions it was contended that Prof Lotz lacked sufficient expertise in the field of neurosurgery and neurology, and that he had refused to

objectively assist the Court. I must say at this stage that I find this a rather strange submission and I will deal with it when I analyse the argument;

45.10

As I have said, Dr Okoli was of the view that a decelerating effect would most probably have caused a generalised injury and not a focal injury as C presents. It must be remembered in this context that Dr Okoli had not been aware of the fact that the Toyota in which Plaintiff was a passenger had flipped over after the high-velocity impact, and also had not considered that C's head had been shown to have turned downwards at the last gynaecological examination;

45.11

The fact that I re-called Dr Pistorius, thereby interrupting the cross-examination of Dr Okoli, was according to Defendant's Counsel "a somewhat strange development in the course of the trial". I will deal with this topic hereunder, but I must state at this stage that Dr Pistorius is based in Stellenbosch and obviously arrangements had to be made for him to return to this Court. I deem this not only in the interest of justice in general, but also in the interest of the child, that the topic of dysmorphia that Dr

Okoli had introduced himself by producing C's ID-document, not be left hanging in the air as it appeared to me that it could be an important factor to be considered in general;

45.12

Defendant's Counsel did not wish me to make any credibility finding although he did criticise First Plaintiff's evidence relating to the presence of foetal distress, which was not supported by clinical observations at the time. This however is not a relevant topic and I will not deal with it, nor with First Plaintiff's evidence relating to less numerous foetal movements. Prof Lotz was however criticised because his evidence went beyond the contents of paragraph 3 of his expert summary, which referred to a number of "reasonable and logical considerations", relating to the cause of C's condition. He had also not examined C, had never consulted medical text books in that context, and did not consider congenital defects, so it was contended. It will be convenient to add at this stage that during cross-examination Prof Lotz was asked how a congenital defect would present itself on MRI imaging. On the one hand Prof Lotz said that the MRI did not show any congenital defect, but also added

that he could not answer the question, because he did not know which congenital defect was being referred to, because there were thousands upon thousands;

45.13

Upon analysis of Defendant's Heads of Argument, it appears that the major part of the criticism levelled against Prof Lotz, is that he did not consider other probable causes apart from his arterial spasm theory. In that context he failed in his duty as a supposedly objective witness, so it was said.

45.14

Dr Pistorius gave detailed evidence, but this was rather inappropriately described by Defendant's Counsel as being a "miraculous theory". In essence his opinion had been that the destruction of the basal ganglia by a loss of blood supply for an undetermined period was the cause of the thinning of the corpus callosum. If there had been any other cause for this injury to the corpus callosum, a more global destruction of the brain would have been expected. It must be remembered at this stage that Dr Okoli agreed that at some stage poor blood supply to the cerebral arteries must have been present, because both the basal ganglia and the corpus

callosum were involved. The cause of the reduced blood supply in this context is the essential difference of opinion between that of Dr Pistorius and Dr Okoli. It was submitted that Dr Pistorius' evidence was "an incredulous explanation". This was his submission in the context of his evidence as to how the sudden deceleration would have caused a flexion in C's neck. It would in fact have been most likely a lateral flexion, which would be supported by Mrs S's evidence that the vehicle had flipped onto its side after the impact. It is true that Prof Lotz had said that no one knew exactly what happened to C's body at the time of the impact and immediately thereafter. Defendant's Counsel however, criticised Dr Pistorius who presented his theory without even testifying as to the position of C in the womb. Dr Coleman however had said that C had already turned in the womb;

45.15

Plaintiff's witnesses were furthermore criticised on the basis that neither Dr Pistorius, nor Prof Lotz, could say with any certainty as to how long the blood supply had been interrupted. Counsel's conclusion in the context of the expert evidence was that not even Plaintiff's experts could agree as to what had happened to C. According to him

the blind led the blind, which is a rather inappropriate observation in this sad case having regard to the experience and expertise of the medici. Lastly, Defendant's Counsel made a surprising submission for which there was no evidence whatsoever, and which had never been put to any witness at all, namely that C's face did not present the asymmetry initially testified to by Dr Okoli, because she had received "botox treatment". The less I say about this contention, the better, but it is totally without foundation.

46.

Recalling of witnesses and observations in Court:

It is not that unusual in civil trial proceedings that the Court recalls a witness who has already given evidence for further examination or cross-examination. A Court does have this discretion, and, in the interests of justice, it may be necessary where a glaring uncertainty has not received the necessary attention. By this I mean that

such uncertainty, when it appears, ought at least to be of a material nature. Contextually, see: *Oosthuizen v Stanley 1938 AD 322 at 333*.

It is obviously not a Court's duty to recall a witness to promote or defeat a party's claim where Counsel intentionally or inadvertently failed to adequately address a particular issue. Prejudice to the opposing party must be considered, and it should be made clear on which topic the additional evidence will be focussed on, and limited to.

See: *The South African Law of Evidence, 2ND Edition, D. Zeffert and A. Paizes, Lexis Nexis, at 927*, and

Hladhla v President Insurance Company Ltd 1965 (1) 614 AD.

Apart from these considerations, I considered it in the best interests of the child that Dr Pistorius be recalled after Dr Okoli had deemed it fit to produce C's identification document from his records. No one had seen this before and the topic of asymmetry of her face had received only scant attention. A child's interest remains of paramount importance in every matter concerning a child. Section 28 (2) of the *Constitution* is abundantly clear as are the provisions of s. 6 of the *Children's Act*

28 of 2005. The question of what she looked like to a specialist physician and why, was a material consideration when I made this decision. Dr Okoli was still being cross-examined at the time, and Dr Pistorius' evidence on this topic was interposed so that the former could also put his observations on record, and I my own. I have said what these were. Obviously I cannot say what the cause of C's "squint" in the left eye was and the way her smile differed each time, but it seemed to me that Dr Okoli had over-stated his observation in this regard, and that the factual observation of Dr Pistorius accorded more with my own observation, which I was entitled to make and note it on the record. I did not see an obvious deformity in C's face, such as initially referred to by Dr Okoli. I saw what Dr Pistorius saw.

See: *The South African Law of Evidence, supra at 859.*

How does a Court evaluate conflicting medical expert evidence?

This topic has been dealt with in a number of foreign jurisdictions and also in South Africa, including dicta by the Appellate division and the Supreme Court of Appeal. I do not intend repeating the well-known dicta relating to the principles that need to be applied in great detail, but certain basic observations in this regard are apposite. Before dealing with conflicting evidence and attempting to arrive at the most probable finding in this particular context, it is necessary to refer to the classic English judgment in *National Justice Compania Naviera SA v Prudential Assurance Company Ltd ("The Ikarian Reefer") [1993] 2 Lloyds Rep 68 at 81*, where the duties of an expert witness were set out, and these duties have been confirmed by various judgments of the South African Courts since then. In the present context, the most important duty of an expert witness is that he should provide independent assistance to a Court by way of an objective, unbiased opinion in relation to matters within his expertise. In the same vein, an expert witness should make it clear when a particular question or issue falls outside his expertise. The crux really is in any given case that an expert comes to Court to give the Court the

benefit of his or her expertise. An expert does not assume the role of an Advocate, nor gives evidence which goes beyond the logic which is dictated by the scientific knowledge which that expert claims to possess. A further important consideration is the following: an expert's bold statement of his opinion is not of any real assistance to a Court. Proper evaluation of the opinion can only be undertaken if the process of reasoning which led to the conclusion, including the premises from which reasoning proceeds, is disclosed by the expert.

See: *Coopers SA Ltd v Deutsche Gesellschaft Für Schädlingsbekämpfung MBH 1976 (3) SA 352 (A) at 371 F –G*.

In *South African Law of Evidence supra at 328*, the learned authors say, and this also appears from a number of important reported decisions, that ultimately the determination by the Court depends on the examination of the opinions and the analysis of the reasoning behind them.

An expert witness is also expected to state the facts or assumptions upon which his opinion is based. He should not omit to consider material facts which could detract from his concluded opinion. Stubborn dogmatism is of course of no benefit to the Court. In this context it is important to remember that in civil cases, in finding facts and making inferences, the Court may decide upon a mere preponderance of probability, even although its so-doing does not exclude every reasonable doubt.

See: *Govan v Skidmore 1952 (1) SA 732 N at 734 A*.

This dictum was approved by the Appellate Division in *AA Onderlinge Assuransie Bpk v De Beer 1982 (2) SA 603 at 614 H – 615 B*. The Appellate Division also approved the *Govan* dictum at 734 C, where it was said that "It seems to me that one may, as Whigmore conveys in his work on *Evidence (3RD Edition, par. 32)*, by balancing probabilities select a conclusion which seems to me the more natural, or plausible, conclusion from amongst several conceivable ones, even though that conclusion may not be the only reasonable one."

The same passage was referred to with approval by Holmes JA in *Ocean Accident and Guarantee Corporation Ltd v Koch 1963 (4) SA 147 (A) at 159 C to D*, where

the following was said in respect of the word “plausible”: “I need hardly add that “plausible” is not here used in its bad sense of “specious” but in the connotation which is covered by words such as acceptable, credible, suitable…”

49.

In *Michael and Another v Linksfield Park Clinic (Pty) Ltd and Another 2001 (3) SA 1188 SCA at 1201*, two important dicta appear: the one is that when dealing with expert opinion, a Court must be satisfied that such opinion has a logical basis. I may also add my humble opinion to this admonition, namely that the importance of logical thought in any given context is not new, and it is undisputed that Greek philosophers laid the basis for logical thinking and deductions more than 2400 years ago. Aristotle (384 322 BC) was the leading thinker on this topic.

See: *Think, Simon Blackburn, Oxford University Press 1999, Chapter 6.*

The most important dictum of the Supreme Court of Appeal in the *Linksfield* case supra, is, for present purposes the following (at par. 40): “Finally, it must be borne

in mind that expert scientific witnesses do tend to assess likelihood in terms of scientific certainty. Some of the witnesses in this case had to be diverted from doing so, and were invited to express the prospects of an event's occurrence, as far as they possibly could, in terms of more practical assistance to the forensic assessment of probability, for example, as a greater or lesser than 50% chance and so on. The essential difference between the scientific and the judicial measure of proof was aptly highlighted by the House of Lords in the Scottish case of *Dingley v The Chief Constable, Strathclyde Police 2000 SC (HL) 77*, and the warning given at 89 D to E, that "one cannot entirely discount the risk that by immersing himself in every detail and by looking deeply into the minds of the experts, a Judge may be seduced into a position where he applies to the expert evidence the standards which the expert himself will apply to the question whether a particular thesis has proved or disproved – instead of assessing, as a Judge must do, where the balance of probability lies on a review of the whole of the evidence"."

I certainly do not intend to be seduced in that manner and I will apply the judicial measure of proof to the evidence that I have referred to.

50.

In *Louwrens v Oldwage 2006 (2) SA 161 SCA at 174 H*, with reference to the *Linksfield Park* decision supra, the following was said: "What was required of the trial Judge was to determine to what extent the opinions advanced by the experts were founded on logical reasoning and how the competing sets of evidence stood in relation to one another, viewed in the light of probabilities." Logical reasoning can lead one to the probabilities and to the judicial standard of proof that I have mentioned. I have some philosophical reservations whether, on the other hand, it can be said that one can arrive at a probable answer on the basis that it is logic. Either a process of thought is logic or it is not logic. Can one say that something is probably logic?

See: *Mutual and Federal Insurance v SMD Telecommunications [2011] 2 All SA 34 SCA at 45 par. 27*.

In "Evaluation of Divergent Expert Opinions" by L. Meintjies-Van der Walt in *The South African Journal of Criminal Justice 2011 at 213*, she referred to a passage in an article which pointed out the limitations of logic: "Logic, or any other formalism, will not determine what is and what is not true. Logic determines the consequences of believing certain things to be true; logic is concerned with arguments, not truth. It provides an analysis of arguments that are in some formal sense justifiable, e.g. valid arguments which are sound with respect to the formal semantics. But it does not tell you what things you should believe to be true in the first place". The author of that article also advises that a number of other factors should be kept in mind which may not always show specific features of logical reasoning, for instance the question whether the experts' methods and reasoning enjoy general acceptance in the relevant scientific community?

In *Buthelezi v Ndaba 2013 (5) SA 437 SCA at 442 G*, with reference to opposing views of expert witnesses, the Court said that its determination must depend on the analysis of the cogency of the underlying reasoning which led the experts to their conflicting opinions. The relevant dictum in the *AA Onderling Assuransie* decision

supra was also recently approved by the Supreme Court of Appeal in *Golias v MEC for Health 2015 (2) 1997 at 107 par. 19*, where the following was said: "... it is important to bear in mind that in a civil case it is not necessary for a Plaintiff to prove that the inference that she asked the Court to draw is the only reasonable inference; it suffices for her to convince the Court that the inference that she advocates is the most readily apparent and acceptable inference from a number of possible inferences."

The mentioned dicta in the *Govan v Skidmore* case supra, the *AA Onderlinge Assuransie* supra and *Ocean Accident v Guarantee Corporation Ltd* supra, were again referred to with approval by the Supreme Court of Appeal in *Cooper and Another N.O v Merchant Trade Finance Ltd 2000 (3) SA 1009 at 1028 A to D*.

The approach to the evaluation of medical evidence as stated in the *Linksfield* decision supra, was also recently endorsed by the Constitutional Court in *Oppelt v Department of Health 2016 (1) SA at 339 par. 36*. The crux really is that a Court must not assess the cogency of scientific evidence by scientific standards, but by the legal standard of the balance of probabilities (par. 38).

51.

I therefore intend deciding this action on the basis that I must accept the inference that is the most readily apparent and acceptable inference from the other possible inferences that emerged from the evidence and that in doing so I must apply the judicial standard of proof and not the scientific standard. Logical thinking in this process will assist in arriving at the final result, but philosophically speaking, it cannot determine it.

52.

An interesting article on the topic of the evaluation of scientific evidence appears in the *South African Law Journal, Vol 120, 2003 at 352*, by the mentioned L. Meintjies-Van der Walt, titled "*Expert Odyssey: Thoughts on the Presentation and Evaluation of Scientific Evidence"*. In the present context, having regard to the qualifications and expertise of the three main witnesses, namely Drs Pistorius, Lotz

and Okoli, I deem the reference to *Menday v Protea Assurance Company (Pty) Ltd 1976 (1) SA 565 (ECD)*, of particular importance: “However eminent an expert may be in a general field, he does not constitute an expert in a particular sphere unless by special study or expertise he is qualified to express an opinion on that topic. The danger of holding otherwise – of being overhauled by a recycle of degrees and diplomas – is obvious; the Court then has no way of being satisfied that it is not being blinded by pure “theory” untested by knowledge or practice. The expert must either himself have knowledge or experience in the special field on which he testifies (whatever general knowledge he may also have in pure theory) or he must rely on the knowledge or experience of others who themselves are shown to be acceptable experts in that field.”

On the topic of logic, the learned author said that the theory on which the expert bases his or her evidence must exhibit internal consistency and logic. The premises of the theory, along with the observations and data, must lead to conclusions through logically valid reasoning. I agree with that approach.

53.

Analysis of the crux of the evidence and findings:

At the time of the high-velocity impact and sudden deceleration, C's corpus callosum was fully formed. There is no dispute about that. Dr Pistorius is a highly qualified and experienced specialist in the field of foetal medicine and foetal brain imaging. Prof Lotz has vast relevant experience in radiology and imaging. Both agree that C's right basal ganglia were damaged, and that this caused the thinning of the corpus callosum. This could not have occurred prior to 20 weeks gestation and not after her birth. There was thus focalised destruction and an intra-uterine traumatic event was the likely cause. No other known factors could have caused this.

Dr Okoli is an experienced neurosurgeon. He does not remotely have the qualifications and experience of the other specialists in ante-natal radiology, imaging and diagnosis. I agree with Prof Lotz's comment that it is obvious that their fields of expertise were wide apart, and that there is merit in his suggestion that Dr Okoli

should not have accepted this instruction or brief to express an opinion and to give evidence. This comment was not intended to reflect on his knowledge or expertise as a neurosurgeon. It was also admitted by Defendant that at 28 weeks gestation, cerebral arteries are capable of constricting and causing spasms. The corpus callosum can further develop in thickness after 24 weeks gestation. C's MRI image showed a border-zone infarction. These facts are supportive of Plaintiff's experts' view that severe arterial spasms resulting from likely flexion of the foetus' neck would have caused a localised reduction of blood flow to the right basal ganglia. This then caused the thinning of the corpus callosum and C's present condition. Dr Pistorius and Dr Lotz saw a positive correlation between the severity of the accident and the likelihood of trauma. Dr Okoli saw none. He fought valiantly to overcome superior forces of expertise, supported by medical literature, but valiance, and some Judges would have called this stubborn dogmatism, cannot withstand the process of logical reasoning based on particular expertise. The dictum in ***Menday v Protea*** supra, is particularly apposite. Dr Pistorius was also of the view that if C's condition was congenital, both sides of the brain would have been affected, and damage

would not have been so localised. The fact that only a specific area of the brain was affected, pointed to a traumatic vascular event. There was no other logical explanation. Prof Lotz was of the same opinion. Added to this was the fact that Dr Okoli had agreed that there had been a border-zone infarction (but after initially denying it). The vastly experienced Prof Lotz, and I refer particularly to his expertise in ante-natal imagery/radiology, had seen no evidence of any congenital, genetic or inflammatory pathology and also no evidence of heterotopia. The congenital defect relied upon by Dr Okoli did not exist, it could not be found in any literature, and Dr Okoli himself was unable to identify it. I asked Dr Okoli specifically what had caused the thinness of the corpus callosum and he replied that it was "under-formed". He gave no specifics as to why, when and how. At no stage did Dr Okoli testify that at 28 weeks gestation the particular foetal arteries could not go into spasm and therefore arrest the blood supply. At best, according to him, they could only do so "optimally" at 34 weeks gestation. There was no reason indicated by him why an "optimal" spasm was required in the present case. Dr Okoli was very firm in his view that C's whole body spasticity did not support the opinion of Dr Pistorius and Prof

Lotz of focalized brain damage. He had however agreed that at some stage poor blood supply to the cerebral arteries must have been present because both the basal ganglia and the corpus callosum were involved. No details of this “stage” were given or its cause. The cause of this reduced blood supply is the essential difference of opinion between that of Drs Pistorius and Okoli. Dr Pistorius’ view in this context was that the damage to the side of the basal ganglia would have caused spasticity to the left side of the body, but that this spasticity would cross over to the other side of the body and would cause less severe spasticity to the right side. Dr Pistorius was not challenged on this particular point at all during cross-examination, which I deem important in the over-all mosaic of this challenging case.

I have already dealt in some detail with the topic of C’s “dysmorphic facial features”. Dr Okoli’s evidence in this regard is not consistent, and also does not accord with my own observations in Court, nor those of Dr Pistorius.

Applying therefore the judicial measure of proof that I have referred to, keeping in mind also the particular experience and expertise of the experts, and considering where the balance of probability lies on a review of the whole of the evidence, I have no hesitation in finding that the most natural and plausible conclusion is that the relevant accident on 10 December 2012, was the cause of C's present condition, as well as the injuries sustained by the First Plaintiff.

The following order is therefore made:

1. It is declared that Defendant is liable to compensate Plaintiffs for the injuries sustained by them as the result of the collision on 10 December 2012;
2. Defendant is ordered to pay the costs of the action;
3. The parties are given leave to present to me, within 14 days from date hereof, a more specific draft order relating to costs, should they deem it fit to do so.

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JUDGE H.J FABRICIUS

JUDGE OF THE GAUTENG HIGH COURT, PRETORIA DIVISION

Case number: 17386/2014

Counsel for the Plaintiff:	Mr Uys Jordaan
	of Uys Jordaan Attorney
	Attorney for the Plaintiff

Counsel for the Defendant: Adv M. Hugo

Instructed by: Maponya Inc

Date of Hearing: 28 April; 3, 4, 5, 9, 10, 13 & 17 May 2016

Date of Judgment: 24 June 2016 at 10:00