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**REPUBLIC OF SOUTH AFRICA
IN THE HIGH COURT OF SOUTH AFRICA
GAUTENG DIVISION, JOHANNESBURG**

Case Number: 2017/30819

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

In the matter between:

N[...] M[...] obo

N[...] U[...] M[...] H[...] A[...]

Plaintiff

and

**MEC FOR THE DEPARTMENT OF HEALTH,
GAUTENG PROVINCIAL GOVERNMENT**

Defendant

JUDGMENT

Mpofu, AJ

Background Facts

[1] Mrs M[...] N[...], the natural guardian and mother of U (**minor girl child**) born on 19 February 2013, is suing the Defendant (**herein referred to as MEC for the Department of Health**) in order to recover damages allegedly suffered by the child as a consequence of Kernicterus due to the untreated presentation of a high bilirubin level after her birth. The cause of action is located in delict, amidst an allegation that the staff working at the hospitals and medical facility under the authority, administration and control of the Department of Health meted out substandard care to U resulting in the injuries she suffered.

[2] The plaintiff was a G4P3 mother (*viz* she had three children and was pregnant with the fourth). She had an uneventful gestational period. She gave birth to U who weighed in 3,490g at birth, her Apgar scores were normal at 9/10 and 10/10 after birth. Both mother and baby were discharged in good health within six hours, after a doctor had assessed them.

[3] On 21 February 2013, the Plaintiff returned to the South Rand Hospital for a routine check-up, additionally due to the fact that the baby was crying and unwell. They were both admitted at 13:55 by Dr Graca who made a provisional diagnosis of neonatal jaundice. It has been established through expert evidence that 70-80% of all babies develop visible yellow discolouration of the skin/eyes in the first week of life, this is normal (physiological), the yellow pigment (bilirubin) results from the breakdown of excessive red blood cells after birth, hence the inability of the newborn's liver to excrete the excessive load. Bilirubin is potentially toxic to brain cells. Since the bilirubin is bound to albumin in the blood, if and when the binding sites on the albumin are exceeded, free bilirubin can enter the brain and cause permanent damage *viz* Kernicterus.

[4] Dr Graca ordered double phototherapy for baby U on admission, her blood was drawn to ascertain the bilirubin level in her blood, i.e. a total serum bilirubin. A decision was taken that baby U would be transferred to Charlotte Maxeke Johannesburg Academic Hospital (CMJAH). At 15:00, while still in South Rand District Hospital, U was admitted to ward 12, whereby phototherapy was started, an infusion was inserted, the baby was stable and breastfeeding. Phototherapy light works by changing the fat-soluble bilirubin into water-soluble bilirubin to be excreted

by the patient. Intensive phototherapy in the form of double lights is used worldwide in the treatment of severe **neonatal hyper-bilirubinaemia**.

[5] At 17:45 the condition of the baby was recorded in the hospital records as “not well, crying and irritable”. The intravenous infusion was in place. The serum bilirubin level of the baby was recorded as 517. Dr Graca requested that the baby be kept “**NIL PER OS**” meaning that the baby not be breast fed in preparation for a **blood exchange transfusion (BET)** which would need to be performed at CMJAH.

[6] The Emergency Medical Services, (EMS) arrived at the South Rand Hospital at 23:00 to transfer the baby to CMJAH, a delay of 5 hours. The admission records allude to an admission at 23:33. Ultimately the **serum bilirubin** was checked at CMJAH and was found to have slightly reduced from 517 to 482 which was still above the exchange transfusion level. The baby’s condition was described as “awake, active and had a soft fontanelle”. Professor Bolton, the Defendant’s expert, testified that there is no absolute level of bilirubin above which brain damage will be inevitable, but that tables exist which aid caregivers to decide on appropriate management. He further testified that extremely high levels of bilirubin at which brain damage becomes more likely are at about **425 UMOL/L**. In cases of marked raised levels, an exchange transfusion is undertaken.

[7] Professor Smith (the Plaintiff’s expert) stated that when you have a jaundiced baby, irritability should alert you that the effect of bilirubin that is at play, and that the longer you expose the baby to high levels of bilirubin, you are putting it at risk. He contended that the cerebral irritation at 17:45 was a missed opportunity between **jaundice and encephalopathy**, this according to him is supported by the crying U, according to the mother. He went on to explain that irritability, excessive crying, poor ability to suck and a raised temperature were some of the symptoms for the **onset of encephalopathy**. He particularly emphasises that encephalopathy can be subtle at onset but certain symptoms and outcomes may be reversed by a BET although at a bilirubin of 517 the baby may have incurred a degree of injury. Prof Smith further testified that the first transfusion was delayed and performed with blood which was haemolysed. He denied that it was the baby’s reaction to the exchange, stating that

in his career spanning over 30 years he had not seen this phenomenon that he described as a health system's failure.

Emergency Medical Services

[8] After a diagnosis of jaundice by Dr Graca at the South Rand District Hospital and a decision was made at 13:00 on 21 February 2013 that the child should be transferred to CMJAH for a blood exchange transfusion, there was an inordinate delay by the Emergency Medical Services (i.e. ambulance services) to transfer the baby to undergo the procedure which could have reduced the permanent damage suffered by baby U. There are unconfirmed reports by the mother that around 18:00 she overheard Dr Graca over a speaker phone enquiring if the ambulance had arrived to transfer the baby, and that she would sue the ambulance service should anything go wrong. This assertion was denied by Dr Graca who admitted that the child was quite yellow hence she was admitted. She went on to say that she had to knock off at 16:00 and that is why, once Dr Modise became aware of the 517 raised level, he again logged a call to the EMS.

[9] The clinical manager for South Rand, Dr Letoaba, was not part of the staff in 2013, he told the Court in his testimony that there is a service level agreement signed on behalf of the Department of Health to the effect that the National Health Service is an authorised agent of Gauteng. He explained that EMS should ordinarily be available within an hour when summoned to a rural area and thirty minutes for urban areas. He was at pains to explain that the EMS system works on priority patients. He accepted that baby U was a priority patient. It has not been disputed that calls were made to EMS at 20:30 and 21:45. Dr Letoaba admitted that there was indeed a delay in waiting for EMS from 18:30 until 23:30.

[10] The abovementioned sentiment was equally conveyed by Prof Bolton who, in his own words, stated as follows: **“that there was some delay in the provision of an ambulance for the transfer of the patient to CMJAH. It has been accepted by all role players that guidelines within Gauteng are that an ambulance should be available urgently, plus that mother and baby cases are a priority in medical services. Here it took about five hours (5) to take a baby to a hospital located twelve (12) kilometres away”.**

[11] Matron Tasnim Loghat was an operational manager responsible to maintain quality and standards in the two paediatric wards of CMJAH. She testified that baby U was admitted in ward 286 on 21 February 2013 and that she would have to be moved to ward 287 for the blood exchange procedure and that, ideally ward 286 does not accommodate patients with medical conditions for example, patients waiting for a kidney/liver transplant as opposed to oncology and haematology patients.

The Actual Blood Exchange Transfusion

[12] The actual procedure was performed by Dr Mammen, who in 2013 was a registrar (in the process of being trained to be a paediatrician specialist). He was assisted by one Dr Hunt under the supervision of a qualified paediatrician, Dr Sheba Keresa Varughese, in charge of the unit. Dr Varughese testified that she had been on call, according to her Dr Nadia was a registrar on the night of 21 Feb 2013, however and of particular importance is that none of the witnesses alluded to the role of Dr Nadia during the BET although her name was thrown around. Ms N[...] says on arrival at CMJAH the baby was not attended by a doctor until the next day.

[13] According to Prof Bolton the procedure is usually performed in an intensive care unit under a radiant heat source for removal of bilirubin in a double volume exchange.

13.1 That a large bore catheter is inserted into a central vein via the umbilical vein.

13.2 That warmed partially packed blood is used to replace the baby's own blood utilising a dilutional technique and small amounts of the baby's blood are removed and replaced by the donor blood.

13.3 That this procedure is performed under sterile conditions with the baby being closely monitored and which process could take up to an hour to complete.

[14] There is a clash of opinion between Prof. Bolton and Prof Smith about the handling of blood prior to the exchange. Prof Bolton espouses for adding heparin (a blood thinning product) and **calcium gluconate** to the blood before the exchange

and gently tilting the blood pack to mix the products. Prof Bolton also alluded to warming the blood by using a coil. The temperature should be closely monitored to avoid overheating or causing the baby hypothermia.

[15] Prof Smith vehemently denies this state of affairs by saying, “[y]ou’re not supposed to add anything to the blood, as adding may change the PH of the blood.” He goes on to state that blood for transfusion has coagulants.

[16] The addition of **calcium gluconate** and **heparin** can cause clotting if it comes into contact with red blood cells. Prof Smith is adamant that if blood is warmed to more than 37 degrees Celsius, it haemolyses. Haemolyses means red blood cells were broken down and extrudes haemoglobin and potassium. He went on to say that **specimen “JTR 0084/13” was badly haemolysed according to records.** He does not agree with the stance of Matron Loghat and Dr Varughese that the only practical way to warm the blood is with water. He also does not agree that replacement of the baby’s blood within 15 minutes is allowed, he says it goes against norms and standards.

[17] The evidence of Dr Mammen, according to him, was drawn from hospital notes and the protocol book. He testified there had been a doctor who worked at night. None of the witnesses alluded to the night doctor or their role when the baby landed at CMJAH on 21 February at 23:33. Dr Mammen carried out the exchange at 14:45 on the 22 February 2013. He describes the child as crying but stable, temperature was 36.4, and that blood sugar was 2.5 which was on the lower end of the scale. The bilirubin reading was measured to be 482 - still above the exchange level.

[18] Dr Mammen testified further that he followed the protocols of Rahima Moosa Mother and Child Hospital and Chris Hani Baragwanath Hospital in Gauteng, which is to add 400ml of **heparin** and 4ml of **calcium gluconate** for such procedures to the donor blood. He testified that he wrote “shake well to mix all the added products and to distribute them well together”. Dr Mammen was taken to task about the shaking and mixing of the products because both Prof Smith and Dr Maponya (a

paediatrician) testified that shaking blood will surely haemolyse it. Dr Mammen agrees blood may have been haemolysed but not by adding the items added.

[19] Dr Mammen does not know how or by who the blood was warmed as per protocol and tends to hazard a guess that it may be the nurses. He further testified that, it took him 15 minutes to transfuse 120ml of blood, at that time the baby stopped breathing (suffered an apnoea). Saturation dropped to a certain level, they then performed a mouth to mouth resuscitation and put nasal oxygen, the baby responded well with saturation reverting to 90, the heart rate 135 and the blood sugar at 6.6.

[20] At 15:15 another BET was performed where 140ml of blood was transfused, this was after consulting by telephone with the specialist Dr Varughese. The baby developed a rash on her face according to Dr Mammen, although U's mother observed a red rash all over the tiny body. Everyone agrees that the baby passed a bloody stool. This, according to Professors Smith and Bolton, are complications of a BET. The BET process was then halted. One Dr Chaya performed another one on 23 February 2013, which according to all witnesses was uneventful.

[21] During Dr Mammen's evidence the ABO incompatibility was canvassed as a cause of the baby's collapse. The mother's blood group was O positive while the baby's was B positive. The ABO group's incompatibility between mother and child is an extremely common finding and does not warrant any intervention after birth. The plaintiff, in its particulars of claim, had alleged that the defendant was negligent in not predicting the development of jaundice on the baby. A **coombs test** was conducted on both mother and child, both had a negative test result. Dr Mammen confirmed during his cross-examination that ABO incompatibility was indeed not an issue since the **coombs test** came out negative.

Bet Complications

[22] Complications associated with the BET procedure were canvassed by all experts. Prof Bolton accepted that oxygen levels in the baby's blood dropped to unacceptable levels, she developed bruising (haematoma) or red marks on her skin

as alluded to by both the mother and Dr Mammen. By 23 February 2013 the baby had stabilised, seizures which she suffered had been controlled with phenobarbital. The above state of affairs was confirmed by the mother who is adamant that she did not meet Dr Nadia on arrival at CMJAH at 23:30, but she and her baby slept until the next day, 22 February 2013, when her baby was taken for the BET. Upon the baby's return, she saw blood spots on her skin, her baby was breathing by a tube and passed a bloody stool. She testified that a doctor told her, "**sorry mama we tried your child won't hear, sit or do anything without help**". On 23 February, the child suffered seizures.

[23] Prof Smith is adamant that whether the Court finds that there was an onset of **encephalopathy** at CMJAH does not exclude the fact that the child had increased bilirubin which, if managed correctly could have saved the baby's woes. He described that the baby's condition was at the first stage of encephalopathy. He describes it as a stage of irritability and extensive crying. He cautioned that one need not tick all the boxes and that crying was a common feature. He goes on to say that the picture became complicated but that at transfusion the picture of encephalopathy was there as the baby deteriorated in the morning hence the delay. Prof Smith noted the nurse's entry that the condition of the baby was not well and that the baby was crying and jaundiced. He deems these symptoms in keeping with cerebral irritation in that she could not console the baby. The mother also testified that she should have attended a routine check up on day three but went on day two since her baby cried continuously. He testifies that the hospital staff was slow to do a BET or send the blood for a cross-match whether they noticed symptoms or not because early onset is reversible with appropriate treatment. Prof Smith goes on to say that replacement of blood within 15 minutes as alluded to by Dr Mammen goes against protocols, plus blood can't be warmed which is why it haemolysed.

[24] Dr Maponya also simply described a 120ml exchange within 15 minutes as "too quick". She told the Court that if one does that, "**a heart will go into shock**". She attributes this to the oxygen saturation dropping to below acceptable levels. She also criticised the second failed attempt at a BET, describing a 140ml exchange in 15 minutes as "**way too fast**". The doctor suggested that it could cause a blood reaction if one exchanges too quick, a common complication. Both Dr Maponya and

Dr Kgabi agree that there was substandard management in treating the child. Over and above saturation which went below acceptable levels, the baby suffered an **apnoeic attack**, saturation that went low, and lastly, a bloody stool - all complications of BET. The total bilirubin serum of 517 was neurotoxic whether one saw the signs or not.

[25] Prof Smith puts the delay at the door of health officials because a nurse called an ambulance again at 20:30 as the baby's condition remained unchanged. Another call followed at 21:45, one Tebogo was spoken to, he promised an ambulance will be despatched, it only arrived at 23:00 when it was summoned at 18:30 already. According to Prof Smith, the transfer of information from doctor to doctor was poor. This, he says, because the baby's diagnoses were known by 17:00, the reason for the transfer to CMJAH was also well known to the medical staff. The baby was put on "nil per os" in anticipation of a blood exchange transfusion. Prof Smith says a body starved of nutrition for so long could cause the body to go into stress mode. He testified that by shaking the blood, the cells broke down and the capacity to carry red blood cells was reduced.

[26] Prof Smith asserts that the dual pathology causing brain damage was foreseeable and preventable. Prof Bolton, while he concedes that there were delays in getting the results and finding a venue for the exchange, states that the child looked good and damage was not foreseeable. He described acute bilirubin as a progressive condition and that if a baby has signs, they will be sedated and the condition will worsen. He says delays did not cause brain damage. Prof Bolton attributes the injury to a hypoxic event during blood exchange transfusion. This in his explanation means, if during the exchange bilirubin is let into the brain, an imperfect storm results in damages. According to him, U developed a multi-organ failure after the exchange. He says the contributing factors were, (1) severe neonatal jaundice; and (2) collapse during the exchange. Prof Smith on the other hand thinks the first collapse during the exchange was not enough to cause damage.

Screening for Sibling Jaundice

[27] Professor Smith alluded to sibling jaundice, he asserted that since the mother's two children had jaundice at birth, this patient should have been flagged. He places blame at the door of the pre-natal clinic which should have warned the mother of the high risk of baby U developing jaundice due to a history of sibling jaundice in the family.

Issues for Determination by Court

[28] The Court has to determine whether the brain damage which manifested in down syndrome on baby U was:

[a] Foreseeable; and

[b] Preventable

Is there a causal link between the catastrophic brain damage suffered by baby U to the management of jaundice by the health institutions?

Analysis

[29] All experts are in agreement with the report of Dr Bengu (a medical geneticist) that a genetic cause for the neurological condition is unlikely. She found that there was severe **hyperbilirubinemia** and a history of **acute apnoea and a hypoxic episode leading to acute ischemic encephalopathy**. Dr Mogashoa (a paediatric neurologist) was not privy to the MRI findings, she considered that the brain damage was probably caused by bilirubin encephalopathy (kernicterus). Similarly, Dr Mbhokota (an obstetrician) noted that the MRI report confirms that the child suffered an **acute profound hypoxic** brain injury.

[30] In *PriceWaterhouse Coopers Inc. and Others v National Potato Co-operative Ltd and Another*,¹ duties and responsibilities of expert witnesses in civil cases were outlined as follows that:

[a] Expert evidence presented to the Court should be seen to be the independent product of the expert, uninfluenced as to form or content by the exigencies of litigation . . .

¹ [2015] ZASCA 2; [2015] 2 All SA 403 (SCA) at para 98.

[b] An expert witness should provide independent assistance to the Court by way of objective unbiased opinion in relation to matters within his expertise ... An expert in the High Court should never assume the role of advocate.

[c] An expert witness should state facts or assumptions on which his opinion is based. He should not omit to consider material facts which detract from his concluded opinion . . .

[d] An expert witness should make it clear when a particular question or issue falls outside his expertise.”

[31] Observations made by the Court are that almost all experts agree on the outcome of baby U’s condition and what brought it about.

[32] Dr Kgabi (paediatrician) opines that there was a gross delay in making the decision and performing the blood exchange transfusion. She goes on to say that the delay may have contributed to baby U’s poor neurological outcome.

[33] Dr Bhengu (the medical geneticist), noted that there was severe **hyperbilirubinemia** and a history of acute **apnoea/hypoxic** episode leading to **acute ischemic encephalopathy**. She however rules out genetic causes for the neurological condition.

[34] Dr Mogashoa, (a paediatric neurologist) considered that brain damage was probably caused by **bilirubin encephalopathy (kernicterus)**. She was not privy to MRI findings. Dr Mbhokota (obstetrician) noted that the MRI report confirms that the child suffered an acute profound hypoxic brain injury and in the absence of any evidence of intrapartum hypoxia, the brain injury was therefore suffered in the post-natal period. There were no intrapartum hypoxic episodes.

[35] Professor Du Plessis (a nursing expert) notes that the baby was born healthy with normal Apgar scores after a normal vaginal delivery. Doctors Maponya and Kgabi’s joint report stated that the impairment suffered by baby U is due to substandard management of jaundice and a delay in performing a blood transfusion. There has been issue with the fact that the Defendant received Dr Kgabi’s report by

7 September 2020 and was satisfied with it. They never indicated an intention to repudiate it. The Plaintiff alleges that this move by the Defendant is in bad faith.

[36] Professor Bolton, while he admits that there was some delay in the provision of an ambulance for the transfer of baby U, he alleges that ambulance services often function under pressure and this may have been the case here. A five-hour delay cannot be justified. He accepts that the baby's bilirubin level although "substantially" reduced, this in reference to the 471 umol/L, he agrees it is still extremely raised hence the child was placed under double phototherapy. He testified that blood was requisitioned from the South African Blood Services and that a blood specimen for mother and baby for grouping and compatibility has to be transported to the central branch of the SA National Blood Service. The specimen was processed at 10:36 on the 22 February 2013 and the blood requested for the exchange was released at 12:00 noon. This was in justifying the delay for the procedure to commence. Professor Bolton deems the dominant injury on U's brain compatible with an **"acute profound hypoxic ischemic injury to the basal ganglia thalamus"**. According to him there is a less prominent pattern revealing bilateral injury to the Globus Pallidus and hippocampus which is suggestive of bilirubin toxicity. Professor Bolton says the turnaround time for blood results is not excessive at 3 hours although the results revealed a very high bilirubin of 512 umol/L and that this level can be corrected by a BET.

[37] Professor Smith is a neonatologist (with knowledge of new-borns) over and above being a paediatrician of 30 years and counting. He testified that when unconjugated bilirubin levels are high kernicterus will manifest. The baby was at 17:50 awaiting bilirubin results as double phototherapy was administered on the baby, also that 2:45 minutes later at 17:53 nurses recorded the baby as not well, "jaundice++", an indication of raised levels at 517 umol/l. According to Prof Smith, this level threatened the baby's brain. He further stated that a combination of changes in consciousness, muscle tone and seizures are all signs of cerebral irritation related to toxic bilirubin levels. The baby was said to be unwell and irritable at 17:45. When asked to explain bilirubin induced brain damage, he responded; **"irritability, excessive crying, poor ability to suck, raised temperature and the baby may suffer seizures"**. To a question on whether encephalopathy can be

reversed he answered, **“if it scores below a certain number you can reverse the symptoms and outcomes, but if they instituted a BET they would have reduced the levels but at 517 bilirubin the baby may have incurred a degree of injury”**. It was put to Prof Smith that Dr Mogashoa in her report spoke of slight stupor, slight hypertonia, paucity of movement, and a slight high pitch cry to which he responded, “that’s why I said this is an initial phase of irritability and extensive cry, initial stage viz (encephalopathy). Prof Smith stated that one does not have to tick all boxes and that crying was a common feature when bilirubin levels were high. As it was put to him during cross-examination that at 17:45 there was no description of a high pitch cry, he answered **“excessive crying has to be considered, you can’t play with this, it is a foregone conclusion the brain was damaged”**.

[38] Professor Smith accepts that there was no evidence of kernicterus at CMJAH, he however says acute bilirubin precedes kernicterus. He says blood banks are handled by technicians, doctors as physicians should insist and be in communication with the blood banks. When, in para15.1 of the report, the nurse noted the condition of baby U as not well and that the baby was crying and had jaundice++, it was a sign she could not console the baby. He is adamant that the staff was slow to send blood for a cross match and to perform a BET irrespective of whether they noticed symptoms or not. At 13:30 on 22 February the baby was transferred from the ward in a “sick condition” with a drip inserted. Professor Smith states that “sick” means the baby has the signs and symptoms for a jaundiced baby and that furthermore, “it is worrying”. He says that it is not true that the reaction during the BET started an hour later, in fact, it started within 15 minutes of the exchange. He also alludes to the fact that a term baby has 80ml of blood per kilogram. Baby U was 3,2kg and during the exchange, Dr Mammen exchanged 40% of the baby’s volume in 15 minutes i.e. 260 ml.

[39] Professor Smith blames the heating of the blood by saying “blood can’t be put in warm water, the chance that you exceed 36 degrees Celsius is high”. He finds no other reason why the blood haemolysed. Similarly, the blood pack cannot be shaken. Lastly the JTR 0084/13 specimen of 1 March 2013 was recorded as “badly haemolysed”.

[40] Dr Sheba Keresa Varughese a factual witness (paediatric consultant) in charge of the unit at CMJAH testified that Dr Nadia was the registrar on call. She said a call came at midnight or the early hours about a child with a high bilirubin, she says Dr Nadia would have seen the child but she goes on to say that the notes were not available. She admitted blood was ready however there was a problem with the ward and baby U needed to be moved to another ward for the BET. She also states that how the blood was warmed is not documented and it was not recorded. During her cross-examination on a question of what level of bilirubin would be sufficient to penetrate the brain she answered **“we look at the baby’s weight, age, level of bilirubin” she went on to say “517 is quite high”**. When it was put to her that the baby suffered a double blow due to haemolysed blood according to the radiologist she answered, **“I don’t agree with haemolysed blood, the child had a high bilirubin. which if in my view managed correctly, the baby’s brain would have been saved.”**

[41] Although Prof. Bolton agrees there were some delays in getting results and finding a venue, he insists the child looked good and he does not believe the damage was foreseeable. This statement goes against the opinion of all other experts. The Court finds his opinion biased in favour of the Defendant. When he is confronted with the evidence and asked why he did not record a call made at 20:30, he says he did not deem it necessary as it would cause his report to have too much information. As long as there is some admissible evidence upon which the expert’s evidence is based, it cannot be ignored, but it follows that the more an expert relies on facts and not on evidence, the weight given to his opinion will diminish. For example, when it was put to Prof Bolton that the baby was crying, he dismissed it as babies cry for various reasons, not taking into account that the baby cried inconsolably and was described by Prof Smith as the onset of encephalopathy. An opinion based on facts and not on evidence has no probative value to the Court. The records pointed to crying, irritability and jaundice which cannot be styled as something other than what the nurses observed.

Radiology Reports of Doctor Weinstein and Prof Andronikou

[42] An MRI of the brain was performed on the child when she was six years and eight months' old. The joint minutes of the plaintiff and defendant's radiologists are largely in agreement. They agree that the MRI demonstrates features of a prior profound hypoxic injury with the **basal ganglia thalamus and peri-rolandic pattern or acute profound pattern**. In addition, the radiologists agree that there are MRI changes compatible with bilirubin toxicity mandating clinical and laboratory correlation.

[43] Dr Keshave, the paediatric neurologist noted that the child had **microcephaly and dystonic cerebral palsy** complicated by global developmental delay, intellectual disability, symptomatic epilepsy, **kyphosis and pseudobulbar palsy**. Dr Keshave concluded that baby U suffers from kernicterus secondary to delayed identification and management of **hyperbilirubinemia**. In this regard Prof Bolton states that it is not easy to distinguish between the clinical nature of the cerebral palsy caused by damage to the basal ganglia from hypoxia-ischaemia versus that caused by bilirubin toxicity. Dr Smith on the other hand accepts Dr Keshave's conclusion that baby U suffered from kernicterus secondary to a delayed identification of hyperbilirubinemia.

Common Cause Issues

[44] The following issues are common cause:

44.1 Baby U was born on 19 February 2013 at 12h40 at the South Rand District Hospital weighing 3,490 grams with good Apgar scores.

44.2 At 16:50 baby U was seen by a doctor who noted on day 0 of life at +/- 4 hours past normal vaginal delivery that baby U was asymptomatic, feeding normally, had passed stools and urine (meconium noted), pink hydration, respiration clear, cardiovascular system normal, abdomen soft and non-tender, central nervous system normal and good reflexes.

[45] The child was assessed; the plan was to discharge the minor child on breastfeeding. Both mother and child were discharged at 17:40 from the maternity register by a sister Ntshobane. She recorded that mother and baby were in a satisfactory condition. Both mother and child were collected by the mother's husband

at 18:40. This evidence dispels the assertion that the baby was discharged within two hours of its birth and it has been accepted by experts that discharge within six hours is standard worldwide.

[46] The Plaintiff's cause of action as pleaded in the particulars of claim is based on the legal duty owed to baby U arising from the negligent conduct of the medical practitioners and staff in the employ of the Defendant in that:

[a] They failed to timeously provide transport to transfer U from SRH to CMJAH;

[b] They failed to do a blood test to ascertain the compatibility between the Plaintiff's blood group - **O rhesus positive** - and the baby's **B rhesus positive**;

[c] They failed to timeously detect and avoid jaundice of baby U, by conducting the blood compatibility coombs test between her blood group and that of the plaintiff;

[d] They failed to appreciate that a high serum concentration of unconjugated bilirubin in the blood of baby U could cause permanent brain damage;

[e] They failed to transport baby U timeously to CMJAH for a blood exchange transfusion;

[f] They failed to timeously deliver baby U for treatment at CMJAH to be rendered by Dr Nadia or any other medical officer;

[g] They failed to timeously conduct a blood exchange transfusion;

[h] They failed to provide an **anti D immunoglobulin** after delivery;

[i] They failed to do an exchange transfusion when the total serum bilirubin approached dangerous levels.

[47] In their amended plea dated 20 November 2019, the Defendant denied all allegations and pleaded that, in the event that the Court finds that there was negligence, there was no casual connection between the negligence and the cerebral palsy. The Plaintiff alleged that the hospital staff employed the wrong procedure in performing the blood exchange transfusion because the transfusion blood was wrong and as a result the blood haemolysed, plus that the process was performed too quickly.

[48] The Defendant pleaded that blood was ordered as soon as baby U arrived at CMJAH, and that the delay in receiving the blood was not attributable to negligence

on the part of the hospital, but rather on the part of the South African National Blood Services, which is a separate entity to the Defendant and out of CMJAH's control.

[49] The Defendant further pleaded that baby U had no signs of neurological compromise on 21 February 2013 at 19:30 before her transfer from South Rand to CMJAH, however, at 17:45 on 21 February South Rand Hospital recorded **baby U's condition not well, crying, irritable with jaundice++**. This was the time when the laboratory called to say the serum bilirubin of baby U was 517.

[50] A doctor was called at 18:15 (Modise) to transfer U to CMJAH, the latter discussed with Dr Nadia whose notes are unavailable, nor was her role articulated upon baby U's arrival at CMJAH when the ambulance ultimately ferried U and the Plaintiff at 23:30. According to the Plaintiff, a factual witness, she and baby U were not attended by anyone until she was taken for the blood exchange transfusion on 22 February 2013 at 10:00. The above is against information that at 23:23 baby U was seen at CMJAH by a doctor whose name is not mentioned who noted the following, "**well jaundiced, vigorous, flat fontanelle, good cry, moving all limbs, normal tone, normal reflexes**". The doctor noted that there was no evidence of kernicterus and was diagnosed at day 3 with neonatal jaundice. The plan was to admit and administer strong phototherapy, bloods for FBC, urgent SBR, blood group coombs test for mother and baby and to administer intravenous fluids.

[51] On 22 February 2013 baby U was seen at 09:05 and it was noted that she was on day 4 of life with no kernicterus on examination, she was looking well, not dysmorphic, jaundice under phototherapy, not pale, spine normal, chest clear, abdomen contained no organomegaly, kernicterus was diagnosed as neonatal jaundice and there was ABO incompatibility. The plan was to follow up on bloods, to put the child on nil per OS (no feeding) and urgent SBR transfusion. The mother was extensively counselled, she gave consent, the plan was to fetch blood from the blood bank.

[52] Baby U was transferred from ward 286 to 287 since according to the nurses, the exchange could only be carried out in 287. The expiry date on **blood pack number 22315183** was 23 February 2013.

[53] At 10:21 on 22 February 2013, the baby was recorded as stable but crying, pulse 120, respiratory rate 30, saturation 100%, temperature 36.4, hgt 2.5, central nervous system awake, soft fontanelle, chest clear, cardiovascular system normal with no murmurs, abdomen soft not distended, **umbilicus venus line (UVL)** under sterile technique, and intravenous line (IV) inserted on the left hand.

[54] Prof Smith stated that 517 bilirubin is associated with 50% of cerebral palsy babies. He is adamant that the excessive delay in the transfer of U was grossly substandard practice, and has contributed to bilirubin-induced encephalopathy and brain injury as seen on the MRI. He was critical of the 10-hour delay by the South African National Blood Service in processing the blood which he branded excessive and blamed CMJAH for failure to inform the blood bank that blood was urgently required. He described the blood staff as technicians and that physicians (doctors) are the ones who should convey the urgency of a transfusion on a new born whose bilirubin stood at dangerous levels.

[55] Doctors Kgabi (Defendant's expert) and Maponya stated in their report that baby U had a mixed type cerebral palsy, which is associated with global development delay with hearing loss, malnutrition, mental impairment, epilepsy that is caused by **bilirubin encephalopathy** that resulted from substandard management of **severe jaundice (hyperbilirubinemia)** and the delay in conducting a blood exchange transfusion. In their heads of argument, the Defendant criticises the evidence of Dr Maponya as not being supported by objective evidence. It is argued that her evidence contradicts her own report. The Defendant asserts that there is no causal link between the conduct of its employees and the clinical condition and outcome of baby U as the Plaintiff failed to prove causation on the part of Defendant's employees.

[56] Dr Mammen reiterated in his viva voce evidence that when the BET commenced, the child was awake. He used the protocol of adding heparin and gluconate and shook to mix the products well. The baby was 3,2 kg so he needed to exchange the baby s blood at 180ml per kilo which works out to 576mls that should be given over 1 hour. This is divided by 4, meaning 144ml should be given in 15

minutes. Dr Mammen is adamant that the blood pack was not haemolysed. This is against Prof Smith's evidence that the pack had in fact haemolysed.

[57] Dr Mammen is also of the view that even though the coombs test was negative, the mother's blood can react to baby's blood and a reaction can manifest regardless. When asked why there was a delay to do a BET, he answered as follows, “[t]he blood was ordered when the baby arrived at CMJAH, and as soon as the blood was ready we got it and we tried our best, given the circumstances and despite the challenges to do the exchange, to protect the baby and despite the initial concern, we said we have to do the exchange now.”

[58] This answer does not inspire confidence in a practitioner who is about to perform a life-threatening procedure on a three-day old neonate. When a question was posed to him about who warmed the blood, Dr Mammen had no clue who did or what means were employed. The Court find this very sloppy as he was supposed to have managed this complicated process from A to Z. Equally when a question was posed about the words written by Dr Mammen in reference to the adding of heparin and gluconate and shaking well, he conveyed a reply which was less than satisfactory that he did not mean a vigorous mix but just to get the products to gel well together.

[59] Dr Mamman breached the duty owed to baby U when instead of fully supervising the blood warming process, he allowed the nurses to carry out the important duty. In *Life Healthcare Group (Pty) Ltd v Suliman*,² Dr Suliman manifested his responsibility by giving instructions to a nurse to allow labour to proceed, and to sedate if necessary and prescribe medication. The SCA held that, “[i]n our law a negligent omission is only unlawful if it occurs in circumstances that the law regards as sufficient to give rise to a legal duty to avoid negligently causing harm”.

² [2018] ZASCA 118; 2019 (2) SA 185 (SCA) at para 10.

[60] The Court finds that substandard care was meted out to baby U because if baby U and her mother landed at CMJAH at 23:30 and blood was ordered at that time only for it to be fetched by Dr Hunt at 12 midday on 22 February (the next day) and for the process to commence later on for a life-threatening condition is excessively sloppy and substandard especially because the bilirubin level of 517 had an insignificant decline to 482 which is still above the exchange level. This coupled with the knowledge by staff at CMJAH that ward 286 is not suited for a BET, when the decision to move the child to 287 was made, surely damage was manifesting on the baby. Prof Bolton for the Defendant agrees to a note in the hospital records that described the baby as “sleepy”.

[61] Prof Smith espoused that the hospital entry of irritable and jaundice ++ meant the baby was inconsolable and he deemed this the early onset of brain damage, which with appropriate treatment, would have been reversed. Clearly double phototherapy was not successful and the only route was a BET, which was delayed with haemolysed blood. All experts are **ad idem** that shaking the blood vigorously and subjecting it to too much heat/cold can haemolyse it. Dr Maponya also described “irritable” as meaning inconsolable no matter what you do to the baby. She said the brain now has neurotoxins. Prof Smith goes on to say that the staff was slow to send the blood for cross-matching and performing a BET whether they noticed symptoms or not.

[62] In her testimony, Dr Varughese stated that Dr Mammen had performed several BETs, during Dr Mammen’s cross examination he said this was his second. He had only been one year and seven months into his training as a paediatrician. **Dr Nadia was not called to testify about her role, she was the doctor communicated with to effect the transfer, the fact that her notes are unavailable does not assist the Defendant’s case.** How does an academic hospital such as CMJAH leave a neonate whose bilirubin level stubbornly refuses to lower from 23:00 on 21 February until 22 February after midday? This clearly displays a nonchalant attitude.

[63] On the other hand, Prof Bolton presented a graph and mentioned 427 as a level at which unconjugated bilirubin penetrates the brain cells to cause kernicterus.

When a question is posed about this level he responded, “[t]his is the level at which kernicterus becomes more likely”. He, however, contradicted himself when he said, “[t]he child did not have kernicterus at the time of exchange, despite delays this was not cause of kernicterus on baby U”. Asked if irritability was a sign that something was terribly wrong with a child whose bilirubin was 517 he responded, “crying and irritable is not a sensitive or specific for a very ill baby, most babies who are taken to hospital and who are being treated while their mothers are anxious and being moved around are crying and irritable.” The mother should have returned to South Rand on day three for a routine check-up, but her baby was crying and unwell hence she went before the scheduled time.

[64] Prof Bolton also referred to a book by Josef Volpes regarding initial and intermediate set in of encephalopathy. He explained moderate stupor as a semi-coma where the child is not arousable, its level of consciousness is below normal. Although not readily, he admitted to an entry of the records that the child was sleepy but denies it was due to kernicterus, instead attributing the complexity of baby U’s case to **hypoxic ischemia** and that this opened the door for **hyperbilirubinemia damage**. He says the baby had an unexpected and probably unpredictable reaction during the exchange.

[65] On the issue of whether there had been negligence on the part of the pre-natal clinic having not outlined the issue of sibling jaundice, Prof Bolton conceded that he was not aware whether there was a guideline on this matter which dealt with whether history should be taken from the mother. Perhaps if the full history was taken about sibling jaundice, this would have alerted South Rand to be on the lookout. Be that as it may, on 21 Feb 2013, a bilirubin level of 517 was well known to the hospital staff, this was an emergency case for fear that once the bilirubin reached dangerous levels, it could cause irreparable harm. If operations can be done at night, why couldn’t this procedure be carried out when baby U arrived at CMJAH? Was it not negligent to let baby U sleep with a high level of bilirubin? According to the mother, they were seen by a doctor only around 10:00 the next morning. CMJAH is a provincial academic hospital and it has the capability to perform BETS within their facility, their laboratory operates 24 hours.

[66] How the procedure was conducted in terms of adding heparin and calcium gluconate and heating and shaking the items was not pleaded by the Plaintiff. Similarly, the Defendant attempted to introduce documents not discovered in terms of the rules. Courts should in civil matters be wary to accept a disregard of the rule

The Legal Position

[67] For the Defendant to be liable for a loss, the act/omission of the Defendant must be wrongful and negligent and have brought about loss. Negligent conduct giving rise to loss is not actionable unless it is wrongful. Where negligent conduct manifests in a positive act that causes physical harm, this is prima facie wrongful, however a negligent omission will only attract liability when the legal convictions of the community impose a legal duty as opposed to a moral duty to avoid harm to others by a positive act. While the Court expects medical practitioners to exercise reasonable skill and care in their respective field, the determination of negligence in a given case falls within the Court's purview.

[68] The correct approach to the assessment of expert evidence was nicely set out in ***Michael and Another v Linksfield Park Clinic (Pty) Ltd and Another (1)*** where the SCA stated:³

“It is perhaps as well to re-emphasise that the question of reasonableness and negligence is one for the court itself to determine on the basis of the various, and often conflicting, expert opinions presented.”

[69] What is required in the evaluation of such evidence is to determine whether and to what extent the opinions advanced are founded on logical reasoning. The Court is not bound to absolve a Defendant from liability for negligent medical treatment or diagnoses just because evidence of expert opinion, albeit genuinely held, is that the treatment or diagnoses in issue accorded with sound medical practice. The Court must be satisfied that such opinion has a logical basis.

³ [2001] ZASCA 12; 2001 (3) SA 1188 (SCA) at para 34.

[70] Expert witnesses involve the drawing of inferences from facts. The inferences must be reasonably capable of being drawn from those facts. If they are tenuous or far-fetched, they cannot form a foundation for a Court to make any finding of fact. Is the reasoning of the expert consistent with established facts? Just as an example, Prof Bolton conceded that it was misleading of him to say predominant lesions are those produced by acute profound ischemic insult as opined in his report as opposed to the subtle features suggestive of bilirubin damage. He also conceded that the danger level of 400 umol/l was where the neurological toxicity or kernicterus becomes likely, this is supported by his own graph. Prof. Bolton also speculated about the so-called blood reaction by baby U during the BET, this was not even pleaded.

[71] Only Prof Bolton randed the hospital records scanty whilst all other experts are **ad idem** about baby Us' outcome. He is the only one who says the cause of the collapse is speculative. See *Bee v Road Accident Fund* in which the SCA held that, "[w]here certain facts are agreed between parties in civil litigation, the court is bound by such agreement"⁴. Dr Kgabi opined that **"[t]here was a gross delay in making the decision to perform a BET and that the delay may have contributed to baby U's poor neurological outcome."** Dr Weinstein (a specialist radiologist) commented that **"[t]he pattern of the brain injury suffered by U was acute profound HIE and the MRI changes compatible with bilirubin encephalopathy"**. Dr Bengu, a paediatrician and geneticist, opined that the brain damage may be caused by excessively high unconjugated bilirubin due to delayed treatment.

[72] It is poignant that the Defendant did not present the testimony of Dr Nadia, who supposedly played a critical part in the treatment of baby U, given that she was the Dr whom baby U's condition was communicated to prior to being to CMJAH. Furthermore, Dr Nadia's notes, which would have played an absolutely critical role in determining the treatment being administered to baby U, conveniently and surreptitiously disappear and are unavailable for scrutiny by the experts and consideration by the Court. This is one of the facts that impacted negatively on the

⁴ 2018] ZASCA 52; 2018 (4) SA 366 (SCA) at para 64.

Defendant's case and the Court has no option but to accept the Plaintiff's claims as factual.

[73] In this regard, I am inclined to be swayed in the Plaintiff's favour. **See *National Employers' General Insurance Co Ltd v Jagers*** where the court stated that:⁵

"It seems to me, with respect, that in any civil case, as in any criminal case, the onus can ordinarily be discharged by adducing credible evidence to support the case of the party on whom the onus rests. In a civil case the onus is obviously not as heavy as it is in a criminal case, but nevertheless where the onus rests on the plaintiff as in the present case, and where there are two mutually destructive stories, he can only succeed if he satisfies the Court on a preponderance of probabilities that his version is true and accurate and therefore acceptable, and that the other version advanced by the defendant is therefore false or mistaken and falls to be rejected. In deciding whether the evidence is true or not the Court will weigh up and test the plaintiff's allegations against the general probabilities. The estimate of the credibility of a witness will therefore be inextricably bound up with a consideration of the probabilities of the case and, if the balance of probabilities favours the plaintiff, then the Court will accept his version as probably true. If, however the probabilities are evenly balanced in the sense that they do not favour the plaintiff's case any more than they do the defendant's, the plaintiff can only succeed if the Court nevertheless believes him and is satisfied that his evidence is true and that the defendant's version is false."

[74] Taking the above-mentioned principles into account, the Court is inclined to accept the version of the Plaintiff and reject the Defendant's version as not favoured by probabilities. The Court finds that the Plaintiff's opinion is consistent with all the established facts which have duly been proven. In the result the Plaintiff's claim is granted with costs.

Order

⁵ 1984 (4) SA 437 (E) at 440D-G.

[75] In the result the Plaintiff's claim against the duty of care is granted with costs

A E MPOFU
Acting Judge of the High Court
Gauteng Division
Johannesburg]

Date of Hearing: 28 August 2023 - 31 August 2023, 01 September 2023 -
09 September 2023, 26 September 2023 - 29 September 2023 & 03 November 2023

Date of Judgment: 30 January 2024

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