REPUBLIC OF SOUTH AFRICA

SOUTH GAUTENG HIGH COURT
JOHANNESBURG

CASE NO: 2011/35273

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

....
DATE SIGNATURE

In the matter between:

MAKGOMARELA, VIRGINIA ITUMELENG Plaintiff

(on behalf of KM)

and

THE PREMIER OF GAUTENG First Defendant
MEC FOR HEALTH Second Defendant

J U D G M E N T

LAMONT, J:

[1] The plaintiff instituted proceedings against the two defendants in her personal and representative capacity as guardian and mother of her minor child. The plaintiff claimed damages arising out of alleged negligent conduct on the part of the defendants which resulted in the baby being born with cerebral palsy.

[2] The parties agreed to separate the questions of merits and quantum and I will make the relevant order.

[3] The plaintiff after a normal pregnancy was admitted to hospital on 8 December 2005 at 17h30. The plaintiff remained overnight. The next morning at approximately 11h00 she was examined. During that examination she disclosed that she was approximately 42 weeks pregnant. Various tests were conducted including a CTG (Cardiotachograph). A decision was made to administer four Prostin tablets to induce labour. The tablets were administered during approximately 11h00. The plaintiff was next monitored at 20h10 at which time she was moved to the labour ward. At 20h45, 21h45 and 22h45 she was again monitored. There was no further monitoring until12h50 by which time the plaintiff was in the second stage labour. The time of the delivery was 01h00 with the end of the third stage at 01h10. After birth the baby was examined by a doctor; the baby was resuscitated, a drip was inserted, oxygen was administered and the baby was transferred to the neonatal intensive care unit. One minute after birth the baby's Apgar was 6; 5 minutes after birth it remained at 6; 10 minutes after birth it became 7. The

baby at birth was floppy, sucked poorly and did not cry. The baby was sent to the radiology unit within the hospital with a note reflecting that the clinical findings were that birth asphyxia had occurred. At 01h28 the baby's pH was 7.178 and acid base status -19.8 mmol/L.

[4] Until the baby was discharged the symptoms persisted; the baby from time to time received oxygen, had fits continued not to suck well and was distressed. On the day of discharge it was recorded that the baby was well recovered, but neurological state and sucking were to be followed up.

[5] During May 2006 the baby fitted. An examination revealed that the baby was suffering from cerebral palsy. This was the first time cerebral palsy was diagnosed.

PROSTIN

[6] The plaintiff was given four 0,5 mg Prostin tablets to induce labour. The guidelines published during March 2002 which reflect the standard treatment read as follows:

> "*Ripening of the cervix*
>
> *This is necessary when the cervix is unfavourable for induction (unaffected or difficult to rupture membranes or by bishop scoring).*
>
> 1. *Establish foetal well-being with a CTG tracing before starting cervical ripening.*
> 2. *Give intravaginal prosteglandin E² 1 mg (e.g. tablets), repeated after six to twelve hours.*
> 3. *If possible, run a CTG tracing 30 to 60 minutes after inserting prostaglandin.*
> 4. *When the cervix is ripe (favourable for induction), induce labour.*"

The aforegoing represents the treatment to be applied by the medical staff.

[7] The reference to prostaglandin is a reference to a hormone contained within the Prostin tablets.

7.1 Prostin may only be administered to patients who are not in labour. The plaintiff was in labour at the time she was given Prostin and she should not have been treated in this way.

7.2 The plaintiff having been given four tablets was given double the dose which should have been administered (2 mg instead of 1 mg). The dose represents four times the dosage recommended by the manufacturer of the drug (0.5mg).

7.3 The danger inherent upon treating patients with Prostin is that there can be hyperstimulation. An excessive dosage can result in the patient having too many contractions which are too strong too close together and which occur too quickly. The consequence of the excessive number of strong contractions occurring too quickly is that the baby is unable to recover between the contractions. The mechanism causing the baby distress is the reduction of blood flow to the baby during each contraction. In consequence of the reduced blood flow the heart of the baby slows down (known as a deceleration). The oxygen to the baby is carried in the blood. If the blood flow reduces the oxygenation of the baby reduces. If the baby is affected and does not recover the baby can become hypoxic. Hypoxia is a

> state of inadequate oxygenation. The excessive administration of Prostin accordingly can result in the baby being under-oxygenated and in consequence suffering harm. This is particularly so if Prostin is administered when labour has already begun.

MONITORING

[8] Hypoxia is a recognised side effect of the administration of Prostin to a patient. It is for this reason that the procedure set out above requires an initial CTG reading and subsequent readings to be taken 30 to 60 minutes after the Prostin has been inserted. If CTG tracings are taken then it is possible to determine whether or not the baby is in distress and accordingly take steps to deal with such distress. If no CTG reading can be taken the monitoring must be manually done.

[9] The view of the expert Dr Pistorius called on behalf of the plaintiff was that it is common practice for the manual readings to be taken at 30 minute intervals.

[10] The Department of Health March 2002 Guidelines for Maternity Care which is referred to above sets out additional requirements insofar as monitoring is concerned. Routine monitoring of the first stage of labour requires in respect of the foetal condition that the foetal heart rate be measured half-hourly before, during and after each contraction (para 6).

[11] The same document requires that if the pregnancy is post-dates (i.e. the pregnancy exceeds 40 weeks gestation) that additional monitoring take place. That monitoring includes monitoring the foetus during labour with CTG if it is available (it was available and was not used save as set out above). At the time of the admission of the plaintiff to the hospital the height of fundus was measured at 38 weeks. This indicates a two week premature foetus. On the next day however the plaintiff furnished information that she believed she was 42 weeks pregnant. Accordingly the treating doctors from that time forward had reason to suspect that the plaintiff was post-dates and should have been put on guard that they were dealing with a post-dates pregnancy. They should have taken steps to deal with the worst scenario namely that plaintiff was 42 weeks pregnant. The monitoring which was required should have been performed every 30 minutes to an hour by way of each monitoring occasion requiring three readings being taken and considered.

[12] The only labour partogram monitoring took place at 20h45, 21h45 and 22h45. That labour partogram reflects a normal baby. No monitoring took place between 22h45 and birth at 00h50 to 01h00.

HYPOXIA

[13] It is probable that the baby was hypoxic at birth:

13.1 She was diagnosed as such.

13.2 She was administered treatment to resuscitate her. That treatment included administration of oxygen and fluids.

13.3 She was floppy.

13.4 She had poor sucking reflex.

13.5 She was blue in her extremities.

13.6 She did not cry.

13.7 She was sickly and was immediately sent to the intensive care unit (indicating that the treating doctor regarded her condition as requiring that treatment).

BLOOD GAS READINGS

[14] The baby's blood gas measured 28 minutes after birth, measured at a pH of 7.178 and a base deficit of 19.8 mmol/L. These measurements are relevant to demonstrate hypoxia. If a baby is hypoxic it burns glucose to produce energy. This burning of glucose produces acid. This is the reason why the more hypoxic the child the more acidic the blood gas and acid base status.

[15] The readings were taken 28 minutes after birth. The readings are accurate as to the baby's condition at that time. They do not reflect the baby's blood gas at birth as they reflect the improved state of the baby after treatment and the lapse of 28 minutes. The treatment of the baby between the time of birth and the time of taking the test, including giving the baby

oxygen and fluids. The effect of this treatment is to raise the pH level and reduce the negative acid base level. Accordingly although the exact reading cannot be established the pH level at the time of birth prior to treatment was lower than reflected in the blood gas tests.

[16] In the morning after the birth at 09h53 tests were taken and the pH of the baby's blood gas had improved to 7.36 (indicating that the baby had been hypoxic at birth) and the baby's acid base status had reduced to -9,4 mmol/L. An acid base status of between -12mmol/L indicates asphyxia depending on which experts view is followed. An acid base level of -19.8 mmol/L reflects that the baby was hypoxic at birth on any experts approach. It is possible to determine what the levels were at birth i.e. prior to the treatment slightly more accurately by considering the acid base level rate of change as the acid base level changes more slowly than the pH reading rate of change. Hence the later reading of the acid base level will more accurately reflect the extent of the hypoxia at birth. The acid base level was extreme even 28 minutes post birth and after treatment hence the pH level must have been much lower than reflected. It is impossible to scientifically accurately perform any calculation.

[17] On the probabilities the baby suffered hypoxia in consequence of the treatment administered during the course of the birth. On the probabilities the pH level was less than is reflected at 28 minutes after birth as the baby's condition would have improved in between. It was much closer to 7 and could well have been less than 7.

EFFECT OF POOR MONITORING

[18] Had the mother and baby been monitored as required the monitoring would have yielded data which would have enabled the medical team to intervene at an early stage so as to prevent hypoxia occurring. A CTG could have been used to effect tracings on a continuous basis as such was available at the hospital.

[19] Had the baby and mother been monitored and managed in accordance with the standard required the hypoxia would not have occurred as the deceleration in the heart rate (reflecting lower blood supply) would have been noticed. The rate and extent of the baby's recovery after each contraction would have been known. This data would have enabled the treating staff to make and timeously act on informed decisions. They disabled themselves from being able to do so due to their poor monitoring.

[20] The experts are *ad idem* that the management of the labour was not in accordance with practice and that the decision to administer Prostin was incorrectly made.

[21] Even after the baby had been treated with oxygen and its oxygen levels were restored the baby suffered from fits and/or convulsions and poor sucking reflexes; it remained floppy; was mildly distressed and on the day before discharge was administered oxygen. The discharge document reflects the baby as being in a stable condition. It is apparent that notwithstanding that

note that the baby was still being treated by use of phototherapy and had recently been treated oxygen supply indicating a poor respiration. The expert called by the plaintiff indicated that he was of the view that the baby had not recovered on discharge. The expert called by the defendants indicated that the baby had recovered fully and was in a stable condition. It is my view that the defendants' expert failed to have regard to the proximity of other treatment and the fact that the baby was not well. In choosing to rely on the recordal being stable and disregarding this evidence he erre.

[22] Subsequent to discharge and during May 2005 the baby again commenced suffering from fits. It appears that the fitting of the baby was controlled at the time of the discharge but that fitting re-appeared during May/June 2005.

[23] Subsequent tests revealed that the baby suffered from cerebral palsy.

[24] It was common cause between the experts that the fact that cerebral palsy was not diagnosed until May/June 2005 is not indicative of the fact that cerebral palsy did not exist at the time of birth or before.

[25] "Cerebral palsy which is characterised by non-progressive abnormal control of movement or posture may not be diagnosed until months or years after birth. Retrospective review of the pregnancy records often cannot show any obvious antenatal cause because foetal brain development and function cannot currently be routinely visualised or monitored. Complications occurring

in the antepartum period common and important causes of cerebral palsy. Epidemiological studies suggest that in about 90% of cases intrapartum hypoxia could not be the cause of cerebral palsy and that in the remaining 10% intrapartum signs is compatible with damaging hypoxia may have had antenatal or intrapartum origins. These studies show that a large proportion of cases are associated with maternal and antenatal factors such as prematurity, intrauterine growth restriction, intrauterine infection, foetal coagulation disorders, multiple pregnancy, antepartum haemorrhage, bridge presentation and chromosomal or genital anomalies". See the article headed *Education and Debate* BMJ 8 August 2006 page 1055. In the article it is recognised that in individual cases it is very difficult to identify retrospectively whether or not there were antenatal causes of cerebral palsy. Damaging hypoxia occurring during labour can be suspected from any clinical signs none of which are specific to the damaging hypoxia and which could therefore reflect other conditions of the foetus. Clinical science and objective investigations need to be considered to attempt to ascertain more reliably what caused the hypoxia.

[26] The timing of the cause of the neuropathology causing the cerebral palsy could take place pre-birth, during the birth or subsequent to the birth. The mother gave evidence and excluded any cause which may have arisen subsequent to the birth of the child. The child was well fed, did not choke and suffered no injury. Whatever the cause was it is not occur subsequent to birth.

[27] Prior to birth the baby suffered no injury, there was no relevant infection (such infection as there appears to have been was a urinary tract infection which does not cross the placenta). The only possible cause which there could have been prior to birth was intrauterine growth restriction. The evidence which it was submitted demonstrated intrauterine growth restriction was the rate of change of weight of the mother during the pregnancy as recorded in the charts of the clinic, as also the rate of change of the fundus height. The fundus height should grow at a rate of 1 cm per week.

DATE	WEIGHT	HEIGHT	WEEKS
5 June 2005	50 kilograms		
8 July 2005	50 kilograms		4 weeks
5 August 2005	52 kilograms		28 weeks
26 August 2005	55 kilograms		
2 September 2005	55 kilograms		33 weeks
23 September 2005	55 kilograms		33 weeks
7 October 2005	54 kilograms		34 weeks
28 October 2005	56 kilograms		35 weeks
4 November 2005	55 kilograms		36 weeks
2 December 2005	60 kilograms but weeks queried (referred for gestational age)		38 weeks
28 October 2005			35 weeks

[28] The submission was that the weight change reflected a poor rate of growth of the baby. The poor growth could be occasioned so the submission went by intrauterine restriction. The submission was that the rate the baby grew as represented by the height of fundus was not constant, did not increase at the correct rate and was an indication that there could have been intrauterine restriction.

[29] The evidence against the submission and facts which founded it consists of the following:

29.1 There was never any attention drawn to an abnormal rate of change of the baby as to height or weight when the plaintiff attended the clinic which she regularly did as and when required.

29.2 The records themselves appear to be inaccurate in a number of respects particularly for example with regard to the transposition of the numbers from a number to a graph. This is indicative in my view of potential inaccuracies relating to the taking of the data and is generally a warning that the document is to be approached cautiously.

29.3 An examination of the brain of the baby at approximately the time of birth indicates no abnormalities. If any abnormalities had been occasioned during the pregnancy such should have evidenced in the test.

29.4 At the time the cerebral palsy was discovered a scan of the brain reflected a nature and extent of deterioration of the brain consonant with the injury being occasioned approximately at the time of birth.

[30] The suggestion that there was pre-existing injury to the baby at the time of birth is in my view highly speculative and appears on analysis of the facts on their own relating to likelihood of the event occurring to be unlikely.

[31] The likelihood of damages prior to birth cannot be excluded if one has regard to the postulates. The facts relating thereto when considered alone are unlikely to have caused the cerebral palsy.

[32] It remains to consider the probability of the cerebral palsy having been sustained during the hypoxic incident at birth. The scientific opinion is that cerebral palsy caused by an intrapartum hypoxic event is established if three essential criteria are present. The essential criteria are:

1. Evidence of a metabolic acidosis in intrapartum foetal umbilical arterial cord or very early neonatal blood.

2. Early onset of severe or moderate neonatal encephalopathy in infants of greater than 34 weeks' gestation.

3. Cerebral palsy of the spastic quadriplegic or dyskinetic type.

[33] "All three of the essential criteria are necessary before an intrapartum hypoxic cause of cerebral palsy can begin to be considered. If any one of the essential criteria is not met it strongly suggests that intrapartum hypoxia was not the cause of the cerebral palsy. If blood gas data are not available it cannot be assumed from other signs that hypoxia was present at birth since these signs lack specificity either individually or as a group. When all three essential criteria are met it is then necessary to determine whether the hypoxia was acute or chronic. If evidence for some of criteria 4 to 8 is missing

or contradictory the timing of the onset of the neuropathology becomes increasingly in doubt. Individually, these latter criteria are only weekly associated with an acute intrapartum damaging hypoxic event because … they may be caused by other factors such as infection. Logically most of the final five criteria would have to be present for the balance of probabilities to suggest an acute timing to the hypoxic event. Contrary evidence rather than missing evidence – for example a normal Apgar score at 5 minutes – would weigh against a serious acute event". See Article labelled *Education and Debate British Medical Journal* 1999 319: 1054-9.

[34] The criteria which together suggest an intrapartum timing but by themselves are non-specific include:

> "*4. A signal hypoxic event occurring immediately before or during labour.*
>
> *5. A sudden rapid and sustained deterioration of the foetal heart rate pattern usually after the hypoxic signal event where the pattern was previously normal.*
>
> *6. Apgar scores are 0 to 6 for longer than five minutes.*
>
> *7. Early evidence of multi-system involvement.*
>
> *8. Early imaging evidence of acute cerebral abnormality.*"

[35] Of the criteria which are essential to define acute event items 1, 2 and 3 are all present save in respect of the pH. The pH level was found to be as set out above at 7.178, 28 minutes after birth. The acid base deficit far exceeded requirement and was at -19.8 mmol/L. The question which arises is

whether or not one of the criteria is missing in that the pH level was above the level of 7.00 as required. The pH level was only above the level required some 28 minutes after birth and after oxygen and other resuscitation had been administered to the baby. The base deficit indicates a severe hypoxia even after treatment. The base deficit changes more slowly than the pH. Not only is the pH reading inaccurate in that it measures the state of the baby's blood after treatment but it is ineffective in that it is taken at the wrong time. It is not possible to extrapolate backwards with any degree or certainty. The expert evidence was limited to a suggestion that the pH was less than what was found.

[36] As to the criteria which would suggest an intrapartum timing for the hypoxia item four was present, item five was not measured, item six was present, item seven was present and item eight was established at the time the brain was examined in 2006. (During June 2006 the radiology of the brain reflected bilateral hypodensities involving mainly grey matter in parietal globes indicating atrophy).

[37] The approach of a court to evidence of opinions expressed by experts is set out in *Michael and Another v Linksfield Park Clinic (Pty) Ltd and Another* 2001 (3) SA 1188 (SCA) at 1200.

> *"[36] That being so, what is required in the evaluation of such evidence is to determine whether and to what extent their opinions advanced are founded on logical reasoning. That is the thrust of the decision of the House of Lords in the medical negligence case of Bolitho v City and Hackney Health Authority [1998] AC 232 (HL (E)). With the relevant dicta in the speech of Lord Browne-Wilkinson we respectfully agree. Summarised, they are to the following effect.*

> *[37] The Court is not bound to absolve a defendant from liability for allegedly negligent medical treatment or diagnosis just because evidence of expert opinion, albeit genuinely held, is that the treatment or diagnosis in issue accorded with sound medical practice. The Court must be satisfied that such opinion has a logical basis, in other words that the expert has considered comparative risks and benefits and has reached a defensible conclusion (at 241G - 242B).*
> *[38] If a body of professional opinion overlooks an obvious risk which could have been guarded against it will not be reasonable, even if almost universally held (at 242H).*
>
> *[40] . . . This 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 200 SC (HL) 77 and the warning given at 89D – E that*
> *"(o)ne 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 been proved or disproved - instead of assessing, as a Judge must do, where the balance of probabilities lies on a review of the whole of the evidence.'*
> *It is well established that what is expected of a medical practitioner is the general level of skill and diligence possessed and exercised at the time by members of the branch of the profession to which he belongs. Van Wyk v Lewis 1924 AD 438 at 444".*

[38] A court is required to assess all the evidence which it has before it with a view to establishing whether it is possible to assess what probably caused the event.

[39] There is no probable evidence founding any of the suggestions made by the defendant as to the cause. I have dealt with this issue *supra*. In considering the evidence of whether or not the hypoxic event was sufficient to cause the cerebral palsy it appears to me that in all probability notwithstanding scientific evidence that the pH was less than 7 that it was either in fact less than 7, or else in this particular case the pH was sufficiently low for the criteria to have been sufficiently present.

[40] The only event which occurred which probably could have resulted in the cerebral palsy was the hypoxic event at birth.

[41] The treating personnel did not maintain the general level of skill and diligence possessed and exercised at the time by the members of the branch of the professions to which they belonged (see *Van Wyk v Lewis* 1924 AD 438 at 444).

[42] The monitoring was substandard. Had the monitoring taken place as is required the distress of the baby would have become apparent and other procedures could have been adopted. The insertion of Prostin was ill-advised and not in accordance with general practice in that the administration was not called for at all and inasmuch as the dose exceeded the recommended dosage. The treating staff ware faced with a patient and baby which required special attention. There was reason to believe that the baby was post-dates. Having administered Prostin, special management and observation were required during the course of the delivery monitoring which could and should have taken place did not take place. The treatment was so inadequate that the relevant data required to assess the acidity at the time of birth was not taken.

[43] In my view negligence has been established. In consequence of that negligence the baby suffered a hypoxic event at birth causing the cerebral palsy.

[44] I accordingly find that the plaintiff has established liability.

[45] I make the following orders.

1. The issue of liability and quantum are separated.
2. The defendants are liable to pay damages to the plaintiff in such amount as the plaintiff is able to establish at the resumed hearing.
3. The balance of the trial is postponed sine die.
4. The defendants are to pay the costs of the action including the qualifying fees of Dr. Pistorius.

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C.G. LAMONT
JUDGE OF THE SOUTH GAUTENG
HIGH COURT, JOHANNESBURG

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DEFENDNANTS ATTORNEYS:	**THE STATE ATTORNEY**
DATE/S OF HEARING:	**12 October 2012**
DATE OF JUDGMENT:	**1 November 2012**