IN THE CORONERS COURT

OF VICTORIA

AT MELBOURNE

Court Reference: COR 2014 2294

FINDING INTO DEATH WITHOUT INQUEST

Form 38 Rule 60(2)

Section 67 of the ***Coroners Act 2008***

I, AUDREY JAMIESON, Coroner having investigated the death of BABY XY

without holding an inquest:

find that the identity of the deceased was BABY XY

born 6 May 2014

and the death occurred on 6 May 2014

at Sunshine Hospital, 176 Furlong Road, St Albans Victoria 3021

from:

1 (a) PERINATAL ASPHYXIA

Pursuant to section 67(1) of the **Coroners Act 2008**, I make findings with respect to **the following circumstances**:

1. Baby XY was a 41 week gestation infant who died six hours after she was born. Her mother, YZ was 29 years of age and XY was her first baby. Ms YZ 's pregnancy was uncomplicated, and her expected date for delivery was 26 April

2014. A morphology ultrasound performed on 11 December 2013, when Ms YZ was 20 weeks' pregnant, was normal.[1]

2. On 2 May 2014, Ms YZ attended the Sunshine Hospital outpatient Pregnancy Day Stay Unit. A post term Cardiotocography (CTG)[2] indicated a baseline heart rate within the normal range, with borderline baseline variability.[3] There were no decelerations in the heart rate. On vaginal examination, the cervix ripening process had begun, however the foetal head was high.[4] A stretch and sweep[5] of the cervix was performed. A measurement of the Amniotic Fluid Index (AFI) was nine centimetres, within the normal range.[6] Ms YZ was advised on normal foetal movements and when to present to hospital prior to discharge home.

3. On 4 May 2014, Ms YZ attended the Sunshine Hospital outpatient Pregnancy Day Stay Unit for a repeat CTG and stretch and sweep of the cervix. The 70 minute long CTG recording showed initial reduced variability,[7] which improved in the last 15 minutes to be within the normal range.[8] The baseline foetal heart rate was between 155 to 160 beats per minute (bpm). 'Good foetal movements' were documented. A vaginal examination indicated the foetal head remained high, with no change in the cervix. A stretch and sweep of the cervix was repeated.

4. At approximately 1.50pm on 5 May 2014, Ms YZ went into labour and she was subsequently admitted to Sunshine Hospital at 4.00pm. The membranes spontaneously ruptured at 4.45pm, and the liquor was described as pink in colour. At this time, a vaginal examination of

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[1] A second trimester ultrasound performed between 18-20 weeks gestation to detect foetal structural and growth abnormalities and placental location.

[2] A CTG is electronic foetal monitoring of the heartbeat. CTGs are a widely used technique for assessing foetal wellbeing. During both pregnancy and labour, the foetal heart rate (FHR) may be monitored by either auscultation or electronic monitoring (EFM). Abnormal CTG changes are sensitive but non-specific markers of foetal distress in that a normal CTG is reassuring but an abnormal CTG may not necessarily indicate foetal distress.

[3] Normal baseline variability is indicative of adequate foetal oxygenation. Baseline variability describes fluctuations in the heart rate above and below the baseline. The normal baseline range is 110-160 beats per minute. Normal baseline variability is defined as fluctuations around the baseline in the range of 5-25 beats per minute.

[4] The cervix was 1-2centimetres dilated 0.5centimetres long with the presenting foetal head high in relation to the ischial spines.

[5] A stretch of the cervix aiming to initiate labour by releasing endogenous oxytocin hormone.

[6] Amniotic fluid index is a measurement of the amniotic fluid volume by ultrasound. The AFI volume is one of the important investigations that contribute to the assessment of feto placental condition. The AFI normal range is 5-24cms.

[7] Baseline variability is the single most important feature of the trace in determining foetal wellbeing. Normal variability of between 5-25bpm is indicative of adequate foetal oxygenation.

[8] The CTG recorded for an hour and ten minutes.

the cervix showed full effacement,[9] four centimetres dilation, with the presenting foetal head measured two centimetres above the ischial spines.[10]

5. During labour, two doses of benzyl penicillin were administered to Ms YZ , as a vaginal swab at 35 weeks was positive for Group B Streptococcus (GBS). The first dose was administered intravenously (IV) at 9.00pm, approximately four hours after the membranes ruptured.

6. According to the labour partogram,[11] the foetal heart rate (FHR) was assessed by intermittent auscultation by the midwife from admission at 4.45pm to 11.15pm,[12] with no recorded deceleration in the FHR.[13] The maternal vital sign observations[14] were documented to be within the normal range and the liquor draining was pink in colour.[15] During the first stage of labour Ms YZ was actively labouring in a birthing pool. At 8.45pm Ms YZ was having the occasional involuntary push with contractions. A vaginal examination by the midwife Ms Julie Pring found the cervix to be eight centimetres dilated, -1 in relation to the ischial spines, in a left occiput transverse position.

7. Ms YZ remained in the birthing pool and commenced involuntary spontaneous pushing with contractions at 9.45pm.[16] At 10.30pm with no progress in descent of the foetal head, the attending midwife Ms Pring repeated a vaginal examination of the cervix. There was an anterior lip of the cervix, which was able to be pushed away. A urinary catheter was inserted, draining 250mLs from the bladder.

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[9] In the primiparous woman the cervix must be fully effaced before dilation can progress beyond 4 cm. Therefore effacement is a key determinant in the primiparous woman for the diagnosis of labour.

[10] 2/5 above the pelvic brim.

[11] Chart documentation of labour.

[12] Intermittent auscultation of the foetal heart rate every 15-30 minutes during a contraction using a hand held Doppler machine.

[13] A FHR a baseline of 110-160bpm with no audible decelerations in the FHR.

[14] Temperature, heart rate and blood pressure.

[15] There was no documented meconium stained liquor. Meconium is the first stool of a baby, and is composed of materials ingested when in utero. The presence of meconium in liquor during labour prior to delivery can be suggestive of foetal distress.

[16] According to the Victorian consensus labour and birth guideline, slow progress in the second stage of labour should be assessed by reviewing the efficiency of the contractions, abdominal palpation to check for full bladder, position, station and rate of descent of the presenting part and vaginal examination to confirm full dilation. After two hours of the active phase, an experienced obstetrician should be involved in the management plan and care. Birth would be expected to take place within three hours of the onset of the active phase. In the nulliparous woman, the combination of the passive and active stage of second stage can be within three hours.

8. At 11.43pm, the midwife Ms Pring documented concern with a rising baseline foetal heart rate and an audible deceleration in the foetal heart rate down to 75 beats per minute (BPM). Ms Pring alerted the obstetric registrar Dr Latika Cilly for assistance, who performed a vaginal examination and applied a foetal scalp electrode attached to a CTG.[17] At this time,[18] continuous recording of the foetal heart commenced via a foetal scalp electrode. The CTG had a high baseline heart rate[19] ranging from 160 to 180 beats per minute (bpm), prolonged decelerations[20] lasting in a range of 30 seconds to two minutes. This section of CTG indicated the presence of four uterine contractions in 10 minutes, and prolonged decelerations.

9. At approximately 11.50pm, Dr Cilly reviewed Ms YZ's progress of labour given there was active maternal pushing with no progress in foetal head descent for over an hour, along with foetal heart decelerations recorded on CTG. The vaginal examination confirmed full dilation and a plan was made to review in an hour. At 12.15am on 6 May 2014, the midwife noted the presence of meconium liquor for the first time. The IV was replaced and a urinary indwelling catheter inserted.

10. In consultation with the obstetric on call consultant Dr Reena Jacobs, a decision was made by Dr Cilly at 12.50am for a trial of instrumental delivery in theatre.[21] It was estimated Ms YZ had been pushing for two hours. The midwife informed Dr Cilly Ms YZ was not progressing and that meconium liquor had been observed. Ms YZ was consented for the trial of instrumental delivery in theatre; plus or minus an emergency lower uterine caesarean section. According to the medical record, Dr Cilly informed theatre, the anaesthetist, and the paediatrician.

11. The delivery of the foetal head required two attempts in theatre using vacuum extraction suction, followed by the application of Neville Barnes forceps. The umbilical cord was found to be tight around the foetal neck and meconium stained liquor noted. Dr Cilly noted foetal head retraction in to the perineum, prompting a call out for assistance to theatre.

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[17] The foetal scalp electrode is a device placed under the skin on the presenting part of the foetus whilst in-utero to assess the foetal heart rate pattern.

[18] On 5 May 2014 at 11.43pm

[19] The normal antenatal and intrapartum CTG has a baseline of 110-160bpm.

[20] A decrease in foetal heart rate below the baseline of more than 15bpm and lasting for longer than 90 seconds, but less than 5 minutes.

[21] An instrumental delivery may be performed in theatre rather than the birth centre room to ensure adequate anaesthesia and additional personnel in attendance to assist.

12. A document in the Western Health medical record with no MR number or heading, noted the foetal head was born at 1.42am. Dr Cilly documented recognition of 'turtle neck', where the foetal head burrowed in to the perineum facing Ms YZ 's right side. The cord was around the neck in the presence of thick meconium. Baby XY's head was stuck on the perineum for between three and five minutes due to shoulder dystocia. Baby XY was born floppy at 1.45am. There was a three minute interval between the delivery of the head and the remainder of her body.

13. Baby XY was immediately placed under the care of the attending paediatric registrar, Dr Wittick. Dr Wittick assessed Baby XY as being in poor condition, covered in meconium,[22] with no heart rate and no breathing effort. Dr Wittick conducted oropharyngeal suction and then intubated[23] and started Baby XY on mechanical ventilation within one minute. Cardiopulmonary resuscitation (CPR) was commenced at one and a half minutes, as per the standard neonatal resuscitation guidelines. Assistance was provided by the anaesthetic fellow, Dr Palakviel. Apgar scores were recorded as being 0 at one minute and 1 at five minutes.[24] A heartbeat was first heard on auscultation at six minutes of life. Adrenaline[25] was given via the endotracheal tube (ETT) at 10 minutes. An unsuccessful attempt at umbilical vein catheterisation at this point led to the insertion of an intraosseous cannula[26] at 16 minutes. A peripheral intravenous cannula was inserted at 18 minutes. Baby XY 's heart rate at this stage was 60 beats per minute, with continuation of CPR. Further adrenaline was given via the ETT at 17 minutes. At 19 minutes, the heart rate was noted to be 100 beats per minute and CPR was ceased. An intravenous bolus of fluid (normal saline) was given at 20 minutes. At this time

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[22] Meconium is normally stored in the neonate's bowel until after birth, but sometimes it is expelled into the amniotic fluid, or liquor, prior to or during labour and delivery. The stained amniotic fluid is a recognised sign of foetal distress, and puts the neonate at risk of meconium aspiration where meconium is inhaled and causes chemical pneumonitis, or inflammation within the neonate's lungs.

[23] Placing of an endotracheal tube (ETT) into the trachea to enable breathing via a machine.

[24] The Apgar score was designed to standardise the way caregivers evaluated a baby's physical wellbeing at birth, helping to provide a general understanding of how well each baby makes the physical transition to independent life from their mother. The Apgar score utilises five physical signs of a baby at birth, giving each a possible score of 0, 1 or 2, reaching a total assessment of up to 10 points. The score is usually given by the caregiver when the baby is 1 minute old and again when they are 5 minutes. A low 1 minute Apgar score is not predictive of adverse outcomes but very low Apgars at 5 minutes and beyond are associated with poorer short and long term outcomes.

[25] 2.5mLs 1:10,000 at 1.55am followed by 3mLs 1:10,000 Adrenaline at 2.03am. Adrenaline is an adrenoreceptor agonist used in the resuscitation setting to stimulate heart contraction.

[26] Cannula into the bone marrow, usually the lower leg.

there was difficulty getting an adequate measure of oxygen saturation via a pulse oximeter.[27] Dr Parbhoo, a consultant paediatrician arrived at 25 minutes of life. The heart rate had stabilised over 120 beats per minute and an oxygen saturation reading was documented as 77% at 27 minutes.

14. Multiple further fluid boluses were given. An umbilical vein catheter was successfully inserted. A venous blood gas at 35 minutes showed severe metabolic and respiratory acidosis. At 40 minutes of life, Baby XY was noted to be making occasional gasps, had no spontaneous movements, her heart rate was 140 beats per minute and her oxygen saturation had improved to 100%. She had evidence of poor perfusion, with mottling and thready pulses on palpation.[28] Her pupils were not reactive.[29] She was given bicarbonate. Baby XY was transferred to the Special Care Nursery (SCN) at 60 minutes. Here she received a further fluid bolus, intravenous antibiotics (benzyl penicillin and cefotaxime)[30] and was started on dobutamine,[31] dextrose[32] and morphine infusions. The Neonatal Emergency Transport Service (NETS) was notified at 2.54am. Baby XY was actively cooled (less than 35 degrees Celsius), as per the standard guidelines concerning possible hypoxic-ischaemic encephalopathy.[33]

15. Investigations were reviewed with blood tests showing a haemoglobin level of 159, white cell count 25.1, platelets 149, C-reactive protein less than 1, potassium 6.7, urea 6.1, creatinine 95, INR 5.9, prothrombin time 68.8, fibrinogen 0.9, activated partial thromboplastin time greater than 100, glucose 23.1 and lactate 23.[34] Baby XY remained significantly acidotic. She was

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[27] A pulse oximeter is a transcutaneous device used to measure oxygen saturations, which are a proxy marker of the arterial haemoglobin saturation of oxygen, or the concentration of oxygen in the blood. Normal ranges for neonates depend on how many minutes old the baby is. Identified target ranges are 2 minutes 65-85%; 3 minutes 70-90%; 4 minutes 75-90%, 5 minutes 80-90%, 10 minutes 85-90%.

[28] Suggests that the cardiovascular system is not functioning normally.

[29] Implies neurological pathology.

[30] In combination, broad spectrum antibiotics.

[31] An inotrope used to help heart function.

[32] Standard neonatal fluid preparation to ensure maintenance of both adequate hydration and adequate blood sugar levels.

[33] Jacobs SE, Morley CJ, Inder TE, et al. Whole-body hypothermia for term and near-term newborns with hypoxic-ischemic encephalopathy: a randomized controlled trial. Arch Paediatric Adolescent Medicine 2011; 165:692.

[34] The haemoglobin is normal. A low haemoglobin in a neonate may suggest foetal/neonatal haemorrhage. The white cell count is normal. The platelet count is marginally low (lower normal range is >150) but this may not be significant. The CRP is a marker of possible infection and is normal. The potassium, urea and creatinine are all raised demonstrating acute kidney injury. This would be due to poor perfusion leading to kidney hypoxia/ischaemia. The coagulation profile (INR, prothrombin time, fibrinogen and APTT) is significantly abnormal implying significant impairment of the baby's ability to clot its blood. The glucose is raised, possibly due to intravenous fluid with

given fresh frozen plasma (FFP) to correct her coagulopathy. A chest X-ray confirmed the ETT was in the correct position, with some non-specific perihilar infiltrates,[35] but no other pathology.

16. The NETS team,[36] led by neonatologist Dr Bhatia, arrived at Sunshine Hospital at 4.17am. The impression was that Baby XY had severe hypoxic-ischaemic encephalopathy.[37] Peripheral perfusion remained poor. The umbilical vein catheter was resited as this was accidentally dislodged when attempting to weigh the baby. Attempts were made at inserting an arterial catheter, but these were unsuccessful.[38]

17. At 6.30am, Baby XY had a prolonged oxygen desaturation to 84% that lasted five minutes and spontaneously resolved. There was concern that she was having seizure activity. She was subsequently given phenobarbitone.[39] Baby XY continued to have increasing oxygen requirements between 7.00am and 7.30am. At 7.38am she became bradycardic with oxygen saturations dropping to 64%. CPR was commenced. A capillary blood gas showed persisting severe acidosis. There was no evidence of equipment failure. Baby XY 's clinical status was reassessed: there was no audible heart rate, her pupils were fixed and dilated, and there was no gag or suck reflex, nor spontaneous movements. A discussion occurred between the medical staff and Baby XY 's parents, and resuscitation was discontinued. She was declared deceased at 7.58am on 6 May 2014.

INVESTIGATIONS

Forensic pathology investigation

18. Dr Yeliena Baber, Forensic Pathologist at the Victorian Institute of Forensic Medicine performed a full post mortem examination upon the body of Baby XY , reviewed a post mortem computed tomography (CT) scan, medical records and an e-Medical Deposition from Sunshine Hospital and referred to the Victoria Police Report of Death, Form 83. At autopsy, Dr Baber observed that Baby XY 's weight was slightly heavier than expected for a 41 week

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dextrose. The lactate is significantly raised, which is likely due to significant hypoxia/ischaemia, and would be contributing to the marked acidosis.

[35] Patchy consolidation in the lungs on chest x-ray.

[36] Now called Paediatric Infant Perinatal Retrieval (PIPER). PIPER is a state-wide service which provides accessible and timely expert advice to health care providers for high risk obstetric care.

[37] Hypoxic-ischaemic encephalopathy is a syndrome characterised by abnormal neurological function, usually manifested as decreased conscious state, seizures, difficulty initiating and maintaining respiration, and depression of tone and reflexes. Its aetiology is wide-ranging, but is usually due to an acute hypoxic and/or ischaemic event which in this case could be either antenatal or perinatal.

[38] Baby XY was weighed later, with a 4490 gram weight recorded on the perinatal death certificate.

[39] Barbiturate medication used commonly for seizure control in neonates.

gestation infant. There was some haemorrhage within the left sternocleidomastoid muscle and in the intercostal muscle below the first rib, posteriorly. No significant natural or congenital disease was identified. Neuropathology examination of Baby XY 's brain showed findings suggestive of previous ischaemic insult in utero. External review of the placenta at the Royal Women's Hospital showed foetal thrombotic vasculopathy (FTV),[40] and chorioamnionitis and funisitis of several days duration. Other features were those of peripartum hypoxia and possibly a foetal anaemia.

19. Dr Baber ascribed the cause of XY 's death to perinatal asphyxia. In coming to this opinion, Dr Baber pointed to a combination of several factors. Chorioamnionitis is a sign of ascending genital tract infection, and funisitis represents foetal response to amniotic infection, but is not necessarily an indication that the infection has spread to involve the foetus. There was no evidence of infection at autopsy. Dr Baber noted that current literature has conflicting reports about the relationship of ascending infection and neonatal death. There were hypoxic features in the placenta and evidence of FTV, both of which would have compromised the foetus prior to delivery, confirmed by histological evidence of foetal hypoxia in XY 's brain and liver. Furthermore, adrenal haemorrhage showed that the foetus was physiologically stressed around the time of delivery. In addition, there was evidence of meconium aspiration on histology.

20. Dr Baber opined that the combination of all these issues would have resulted in the foetus being physiologically compromised prior to birth, and less able to withstand the rigors of a difficult delivery. Dr Baber stated that it was not possible to say whether the outcome would have been the same with a more straight-forward delivery, or with less physiological compromise before birth.

Police investigation

21. On 6 May 2014, Victoria Police attended Sunshine Hospital and obtained a statement from Paediatrician Dr Martin Wright. In his statement, Dr Wright reported that he arrived at Sunshine Hospital at 7.35am on 6 May 2014, and was informed that Baby XY needed CPR for her heart to work and required a ventilator. Dr Wright noted that they expected Baby XY to be a little bit sick after the complications at birth, but not as unwell as she had become. In the hours after Baby XY 's death, Dr Wright spoke with clinicians involved in her care, and was informed that there were no real concerns in the lead up to her birth, during Ms YZ's

[40] Blood clots in the foetal circulation of the placenta which has been associated with neonatal brain injuries.

pregnancy. Dr Wright was unsure as to why Baby XY was so sick, and as to why she had died.

Coroners Prevention Unit review

22. I asked the Coroners Prevention Unit (CPU)[41] to review the circumstances surrounding Baby XY 's death. The CPU reviewed evidence including medical records from Sunshine Hospital and General Practitioner Dr John Guymer at Wyndham House Clinic. Statements were obtained from Obstetric Registrar at Sunshine Hospital Dr Latika Cilly, Clinical Midwife Consultant at Western Health Tina Pettigrew, and Specialist Obstetrician and Gynaecologist and Clinical Services Director of Women's and Children's Services at Western Health Associate Professor (A/Prof) Glyn Teale.

23. The CPU identified that Dr Cilly's documented medical assessment of the CTG at 11.50pm on 5 May 2016 indicated the foetal heart rate was within the normal baseline and variability, with no decelerations noted. The foetal head was determined to be in the pelvis with caput[42] noted and Ms YZ to be fully dilated. On vaginal examination, Dr Cilly assessed the 'pelvis adequate'. A plan was documented to review in an hour, with continuous CTG monitoring of the foetal heart. In her statement, Dr Cilly noted that she planned to review Ms YZ sooner if circumstances changed.

24. In her statement, Dr Cilly was asked to comment on the CTG recordings, and she noted intermittent foetal heart rate decelerations that coincided with contractions, with a quick recovery.[43] These occurred at midnight, 00.05am, 00.30am and 00.45am. However, it was noted that the CTG did not influence Dr Cilly's decision for an instrumental delivery in theatre. Dr Cilly's decision was instead influenced by the delay in the birth progress, with a suspected cephalo pelvic disproportion, and meconium stained liquor.

25. The review noted that from 0.40am, the decelerations in foetal heart rate reduced in number. There was no CTG recording from 01.10am to 1.26am where the CTG resumed briefly, presumably relating to the time Ms YZ was transported to theatre. The CTG was

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[41] The Coroners Prevention Unit (CPU) was established in 2008 to strengthen the prevention role of the coroner. The unit assists the coroner with research in matters related to public health and safety and in relation to the formulation of prevention recommendations, as well as assisting in monitoring and evaluating the effectiveness of the recommendations. The CPU comprises a team with training in medicine, nursing, law, public health and the social sciences.

[42] Soft swelling on the foetal scalp.

[43] Variable decelerations are repetitive decreasing of FHR with rapid onset and recovery.

discontinued during the insertion of spinal catheter for the administration of a spinal anaesthetic at 01.20am. In her statement, Dr Cilly noted that when she examined Ms YZ in the lithotomy position in theatre, the foetal head was +1 in relation to the pelvis, with a small amount of caput.

26. It was identified that the sequence of events and timing of the delivery were documented on a specific form designed to document the steps performed to manage shoulder dystocia.[44] This provides evidence of timing and standard steps followed to assist the delivery of Baby XY 's shoulders, which did follow the standard shoulder dystocia procedure.[45] In her statement Dr Cilly noted that almost immediately after Baby XY's head was delivered, she used the McRoberts manoeuvre on Ms YZ , and asked a midwife to apply suprapubic pressure. This failed to deliver Baby XY 's body. Dr Cilly then attempted to deliver Baby 's posterior arm, which failed, and at this stage she called for a paediatric code blue and asked for the consultant Dr Jacobs to be contacted. A midwife then attempted to deliver Baby XY 's posterior arm, but this again failed. Dr Cilly then used the Woodscrew manoeuvre in another unsuccessful attempt to deliver Baby XY 's posterior arm. A midwife was then able to deliver her posterior arm. Dr Cilly noted that this process meant there was a three minute gap between the delivery of Baby XY's head and her body.

Antenatal and Perinatal Insults

27. The review noted that forensic pathologist Dr Baber was of the opinion that Baby XY 's death was due to perinatal asphyxia, which occurred because of a combination of several factors. It was noted that Dr Baber had opined that alone, each factor: in-utero ischaemic brain insult; meconium and amniotic fluid aspiration; FTV and chorioamnionitis, was not solely responsible for the death of Baby XY . However, it was the combination of all the factors that resulted in Baby XY being physiologically compromised prior to birth. Therefore, she was less able to withstand a difficult delivery with the cord wrapped tightly around her neck and shoulder dystocia.

Neonatal Resuscitation

28. Review of the medical documentation for the resuscitation of Baby XY demonstrated that the medical staff largely followed the standard Neonatal Resuscitation Guideline, as established

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[44] Shoulder dystocia is a vaginal cephalic delivery that requires additional obstetric manoeuvres to deliver the baby after the head has delivered and gentle traction has failed.

[45] Royal College of Obstetricians and Gynaecologists guideline Number 42, 2nd edition, March 2012.

by the Australia Resuscitation Council.[46] Particularly, the review found that the standard resuscitation sequence was followed, progression through the sequence was appropriate and problem solving happened in a timely manner, particularly in requesting further senior assistance, intubation and gaining line access.

29. It was identified that areas of difficulty in the resuscitation included problems obtaining oxygen saturations using a pulse oximeter. It was noted that this is often challenging in a newborn and requires experience. The review noted it was unlikely that having the relevant information would have changed the outcome in this case. Another area of contention was the use of bicarbonate. While this is not recommended for routine use in neonatal resuscitation, it was noted that it can still be considered in cases where metabolic acidosis is a significant feature of the clinical picture, which was the situation in Baby XY 's case.

30. In his statement, A/Prof Teal noted an internal review identified there was a difference of opinion between the paediatric and anaesthetic staff regarding the correct dose of adrenaline to be administered at Baby XY's neonatal resuscitation, when a code blue was called at approximately 1.42am on 6 May 2014. A/Prof Teale stated that while practice improvement recommendations were developed in relation to this issue, within Western Health's review deliberations it was not believed this issue altered the outcome.

CTG recordings during labour

31. A/Prof Teale reported there was the ability to perform a foetal scalp lactate[47] at Sunshine Hospital. However, he noted that a foetal scalp lactate is performed when a mother is in labour and there is a problem with the CTG recording, or the CTG is abnormal and/or registered foetal distress. In this case, A/Prof Teale said there was no issue with the CTG recording and no signs of foetal distress – therefore the foetal scalp lactate was not performed.

32. Dr Cilly reviewed a copy of the CTG trace and opined that it had reassuring features because there was variability and the baseline was maintained. Dr Cilly did note that the trace was classified as abnormal because of a variable deceleration at approximately 12.15am on 6 May 2014, which occurred after a contraction. Due to this variable deceleration, the CTG trace as a whole could not be described as normal. Dr Cilly noted there were other variable decelerations

[46] Australian Resuscitation Council (AU). The ARC Guidelines: Section 13 – Neonatal Guidelines [Internet]. East Melbourne, Australia; 2010 [cited 11 Mar 2015]. Available at: http://resus.org.au/guidelines.

[47] Foetal blood sampling, is an adjunct to an abnormal CTG, which is used to better assess the foetal acid base status. It may be considered when the FHR does not improve with conservative management.

on the CTG trace, such as at 12.00 midnight, 12.05am, 12.30am and 12.45am. However, these variable decelerations occurred during a contraction and the baseline recovered quickly. In Dr Cilly's opinion, these decelerations were likely to be associated with head compression during the second stage of labour, which was to be expected.

33. The review noted there were no immediate arterial and venous blood gases performed on Baby XY and although the foetal haemoglobin was low, a maternal Kleihauer[48] test looking for evidence of foeto-maternal haemorrhage was not performed.[49]

Gestational Diabetes

34. The review noted that Ms YZ had an oral glucose tolerance test (GTT) on 20 November 2013 at approximately 18 weeks gestation, and then again on 29 January 2014 at approximately 28 weeks gestation. The GTT results at 18 weeks were normal. At zero minutes, Ms YZ 's venous plasma glucose level was 4.4 mmol/Litre, then 11.1 mmol/Litre at 60 minutes and 5.8 mmol/Litre at 120 minutes.[50] The GTT results at 28 weeks gestation were 4.2mmol/L at zero minutes, 9.7mmol/L at 60 minutes and 8.6mmol/L at 120 minutes.[51] In his statement, A/Prof Teale acknowledged that the 120 minutes result of the 28 week GTT was outside the normal range, and meant that Ms ZY should have been diagnosed as a gestational diabetic.

35. A/Prof Teale acknowledged there was a failure to diagnose Ms YZ with gestational diabetes during her pregnancy. A hard copy of the GTT result was in the photocopied medical record. However, an internal review at Western Health identified that the abnormal 28 week GTT results were not entered into Western Health's pathology computer system, BossNet, until 18 July 2014. The medical records explained that this was due to a 'computer transfer error'.

36. Western Health's review found that the pathology provider, Melbourne Pathology, did not generate a report for Ms YZ's 28 week GTT. However, A/Prof Teale noted that best practice required the treating clinician at Western Health to make proactive enquiries at the time to ascertain the results of the 28 week test. This follow up was not done.

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[48] Detection and quantification of trans-placental haemorrhage.

[49] A sample of blood for a Kleihauer test is taken when there is evidence of a low haemoglobin in a baby soon after birth.

[50] Royal Australian and New Zealand College of Obstetricians and Gynaecologists' (RANZCOG) diagnostic criteria for diabetes. Accessed 1 September 2015 at www.ranzcog.com.au. According to the RANZCOG guideline these were within the normal range, with the 60 minute result borderline.

[51] The normal range is 3.3-7.7 mmol/Litre.

37. A/Prof Teale stated that Western Health has been advised that Melbourne Pathology has introduced a new system for providing the results of GTTs. This new system was being implemented around the time of Ms YZ 's 28 week GTT in January 2014.

Subsequent action by Western Health

38. In his statement, A/Prof Teale noted that Baby XY 's case was the subject of an in-depth review by the Women's and Children's Division at Western Health, and also reviewed by the Serious Adverse Events Committee. Following these reviews, a number of recommendations were implemented.

39. Since Baby XY 's death, Western Health has implemented a dedicated improvement project to ensure all recommended antenatal tests are offered, completed and recorded in clinical records. The purpose of this project is to put the onus on the clinician who ordered the test to make proactive enquiries as to any apparently incomplete test results. A/Prof Teale noted that the project applies to any outstanding GTT results.

40. In addition, on each resuscitaire there is now a folder attached which clearly outlines the correct dose of adrenaline to be administered to a newborn baby. The purpose of this is to avoid any confusion and make sure there is no need for discussion about the dose of adrenaline to be administered during a neonatal resuscitation.

41. All anaesthetic staff who begin working at Western Health now receive specific training in relation to neonatal resuscitation. A/Prof Teale noted that while this is not directly linked to Baby XY 's case, it is an important improvement to the neonatal and paediatric service at Western Health.

42. A/Prof Teale added that all members of the Western Health emergency department paediatric team now undertake specific scenario training in the operating theatre. This is to ensure that all members of this team are familiar with the layout of the theatre and the procedures in place if called to assist in a neonatal resuscitation. Once again, this improvement was not directly linked with Baby XY 's case.

Further investigation by the Court

43. Further investigation by the Court ascertained that while A/Prof Teale had referred to 'Melbourne Pathology' in his statement, it was in fact Melbourne Health Pathology Service at the Royal Melbourne Hospital that had provided the GTT to Ms YZ .

44. In a statement dated 2 December 2016, Dr Malcolm Mohr, Chemical Pathologist at Melbourne Health Pathology Service, noted that a GTT (including fasting, one hour and two hour tests), Vitamin D and Full Blood Examination were taken from Ms YZ on 29 January 2014 at the Melbourne Health collection centre at Melbourne Private Hospital.

45. Ms YZ 's details and test request were entered into the laboratory computer system – 'KESTRAL', but Ms YZ was not registered as being pregnant. This resulted in the report listing the reference intervals for adult patients, as opposed to the reference intervals for pregnant patients.

46. Dr Mohr advised that Melbourne Health Pathology Service was introducing a new computer system in January 2014. The test order code (OGTT1X) request only allowed one result to enter into the patient report file if the fasting result was entered first. The results from the one hour and two hour glucose samples had no area to be inserted, as they were set as 'Optional' in the KESTRAL laboratory computer. Dr Mohr noted that this intermittent failure was not appreciated at the time.

47. The fact that the one hour and two hour glucose samples had not been reported upon was not picked up on any worklists because the test order code used did not specify that three glucose samples must be reported. The fasting glucose, Vitamin D and Full Blood Examination results were sent on completion to Dr Pham in Obstetrics and Gynaecology at Sunshine Hospital and Dr McCarthy at Sunshine City Medical Centre.

48. Dr Mohr stated that an enquiry by Clinical Midwife Consultant at Western Health, Tina Pettigrew triggered the finding of the two unreported glucose test results and the manual entry of the one hour and two hour glucose samples on to the report on 18 July 2014.

49. Dr Mohr advised that the system error was amended on 29 September 2014, when all samples for fasting, one hour and two hour glucose test results were changed from being 'optional' to 'required' results, so as to prevent the error occurring again.

Expert opinion from Dr David Simon

50. The CPU recommended an opinion be sought from an expert obstetrician, who could evaluate the pregnancy care, labour and birth to identify if there was an opportunity for earlier intervention which would have prevented the death of Baby XY .

51. An expert opinion was sought from and provided by Dr David Simon, a specialist obstetrician and gynaecologist, Clinical Lead of Obstetrics and Gynaecology at the West Gippsland

Hospital, and Lecturer and Discipline Head of Women's Health at Monash University Gippsland Rural Medical School.

52. Dr Simon noted that Ms YZ had a family history of diabetes and it was appropriate that an early GTT was undertaken to exclude diabetes. He reported that gestational diabetes, especially if poorly controlled, increases the chance of macrosomia, shoulder dystocia and late stillbirth. If this information had been known, then the conduct of the antenatal care would have been different. Dr Simon stated that Ms YZ would have been referred to a diabetes educator and dietician for advice, and would have commenced daily self-monitoring of her blood glucose levels. She would likely have been referred to a collaborative medical model of care. Ms YZ 's care may have included an extra ultrasound in the latter weeks of her pregnancy to determine whether the baby was big; and may have included an induction of labour prior to term. In addition, care would have included continuous intrapartum foetal monitoring with CTG for the duration of the labour if macrosomia, poor diabetic control or the need for medication for diabetes had been present. Dr Simon added that the knowledge that a woman with a suspected big baby has gestational diabetes may influence a doctor's decision whether or not to conduct a trial of instrumental delivery, rather than proceed to caesarean section.

53. Dr Simon noted that meconium stained liquor is an intrapartum risk factor for foetal hypoxia, and prolonged second stage and instrumental birth are risk factors for shoulder dystocia. In addition, macrosomia above 4.5kg is a risk factor for shoulder dystocia. Dr Simon noted that the fact Baby XY was just under this weight was not recognised antenatally by the case-load midwife. He observed that Baby XY 's weight at autopsy was 4.42kg, and reported that 4.41kg is the 97th centile for 41 week female babies. However, Dr Simon also noted that symphysiofundal height measurements in the latter weeks of pregnancy and on Ms YZ 's admission to the labour ward were in the expected range for a normally grown baby. Dr Simon stated that it is recognised that clinical estimation of foetal weight is unreliable, and third trimester ultrasound estimation of foetal weight also has a significant margin for error. In the absence of diabetes or of a fundal height greater than expected, there appeared to be no reason for the case-load midwife to suspect that Baby XY would be macrosomic.

54. Dr Simon noted that the recognised normal baseline foetal heart rate is 110-160. The foetal heart rate was above 160 at 11.32pm and 11.34pm, and this was the time that CTG monitoring was certainly warranted. However, Dr Simon noted this was just 13 minutes prior to when CTG monitoring did in fact commence.

55. Dr Simon noted that no adequate interpretation of a CTG can begin without determination of the baseline foetal heart rate, and for much of Ms YZ's CTG this was very difficult. The baseline foetal heart rate and variability should be determined at points in time where there is absence of an acceleration, deceleration or contraction. The difficulty arose in interpreting this particular CTG because there are 4-5 contractions each 10 minutes, with most lasting 90 seconds to two minutes, and there are few times, at least until soon before transport to theatre, when decelerations and uterine activity were absent.

56. Dr Simon stated that one or two hours of maternal pushing would not normally indicate the need for a caesarean section if the CTG was seen to have reassuring features and there was no diabetes, even when a baby is felt to be big. He opined that a trial of instrumental delivery conducted in theatre was a reasonable option. Dr Simon added that the management of the shoulder dystocia by Ms Pring and Dr Cilly appeared appropriate. In particular, an appropriate sequence of manoeuvres was used and documented, and the decision to alternate between the person attempting the manoeuvres was appropriate.

57. Dr Simon noted that from 11.43pm, Baby XY 's foetal heart rate dropped from 100 to 60, where it remained for 90 seconds. It then climbed to a probable baseline of 165 with uncertain baseline variability. When asked if there were clinical indications for an earlier delivery, Dr Simon proposed, but on balance rejected, the option of immediate caesarean at 11.43pm. Given that the bradycardia began to recover within two minutes of its nadir, and with the CTG apparently demonstrating reassuring features within 30 minutes, continuation of labour at 12.17am seemed a reasonable decision.

58. Dr Simon believed that only with the wisdom of hindsight would he have interpreted the clinical situation and CTG as requiring urgent delivery before about 12.35am. Paradoxically, less obvious shallow late decelerations that were present between 12.40am and 1.00am, when combined with the whole clinical scenario including new meconium liquor, were a more sinister pattern than the more obvious decelerations seemed earlier in the trace. Dr Simon stated that an urgent caesarean section could reasonably have been organised at 1.00am after 20 minutes of persistent late decelerations, which may have had Baby XY delivered 20-25 minutes earlier than in fact occurred. Dr Simon stated that whilst he is not a neonatal physiologist, such an earlier delivery may not have been early enough to save Baby XY from serious harm.

59. Dr Simon referred to literature which depicts the danger of persistent prolonged decelerations – *'we need to be particularly cautious in the care of the foetus with limited reserves who may be*

less able to tolerate the hypoxia causing the prolonged deceleration. The... foetus of a diabetic mother... (is) much more likely to become compromised following prolonged decelerations than an otherwise healthy foetus' (Baker et al. Fetal surveillance – a practical guide, FSEP).

Supplementary correspondence from Dr David Simon

60. In supplementary correspondence with the Court dated 9 August 2016, Dr Simon emphasised that knowledge of gestation diabetes would have increased the *a priori* risk of macrosomia and foetal distress, so inevitably have increased vigilance or routine screening for these issues. While Dr Simon was not aware of Western Health's policies, knowledge of the gestational diabetes would have at least prompted blood sugar level monitoring, perhaps a third trimester ultrasound, perhaps early induction if thought to be a normally grown baby, and definitely early induction if thought to be a large baby, with perhaps elective caesarean if thought to be very large. There would have perhaps been continuous CTG, and great caution for midcavity trial of instrumental delivery where the baby was thought to be big.

61. Dr Simon noted that if gestational diabetes had been known, then a CTG would probably have already been in operation prior to 11.43pm, so more of a 'baseline picture' would be known to judge the significance of features seen from 11.43pm to midnight. While the CTG was recognised to be abnormal from the outset at 11.43pm, an 'abnormal' CTG does not equate to the need to deliver the baby immediately. Dr Simon reported that the whole clinical picture is taken into account, and a guiding element can be whether there are reassuring features. Looked at in isolation, the 12.09am to 12.19am section of the CTG could be interpreted as normal. However, Dr Simon wondered if this was actually a very unlucky and understandable misreading of the CTG, where the next contraction was coming on and the foetal heart rate falling, before it returned to a baseline of 160. Dr Simon opined that one could never come to this interpretation without seeing the later evolution of the trace, and knowing the poor outcome. A delay of three minutes with shoulder dystocia in a well baby, even after a difficult forceps delivery, should not result in a baby that remained extremely unwell and died.

62. Dr Simon reported that once the frequent contractions spaced out a bit and the deep decelerations settled on the CTG, it was clearer to see that the CTG was abnormal. However, this was not an obvious abnormality, and Dr Simon acknowledged his opinion may well be influenced by the outcome.

63. Dr Simon also acknowledged that a 'safety first' attitude may have prompted other obstetricians to deliver Baby XY at midnight, but he believed many others would not have done so, just

on the facts available at the time. Dr Simon believed Dr Cilly could have been rung earlier to return once meconium was evident and the decelerations returned.

Supplementary Forensic Pathology Report

64. On 1 September 2016, Dr Baber completed a supplementary report after reading Dr Simon's expert opinion. Dr Baber noted that she did not have information regarding Ms YZ 's gestational diabetes at the time of completing her autopsy report. The presence of maternal gestational diabetes could explain the heavier-than-expected weight of Baby XY at the time of delivery at 41 weeks gestation. However, Dr Baber reported that the cause of Baby XY 's death remained unchanged as perinatal asphyxia.

Mention Hearing on 6 December 2016

65. A Mention Hearing was held on 6 December 2016, in order to progress my investigation; enable parties to raise any further issues that might warrant the holding of an Inquest, or alternatively an in-chambers Finding; and to advise parties of my intention to make comments in this matter which could be perceived as adverse. At the Mention Hearing, Baby XY's family were represented by Slater and Gordon Lawyers, Western Health by K&L Gates, and Melbourne Health by MinterEllison Lawyers.

66. Paula Pulitano of Slater and Gordon advised the Court that while the mention hearing had focused upon systemic errors which related to Baby XY 's death, her family had concerns about the prolonged labour and its management, and the contribution that this had, if any, to Baby XY 's death. Ms Pulitano stated that a report had been obtained from Dr Robert Lyneham, dated 4 December 2015, relating to these issues. She requested and was granted time to seek further instructions as to whether to tender the report.

67. At the Mention Hearing, Peter McGrath of K&L Gates stated that Western Health had conceded it did not follow up on obtaining the full GTT results, and acknowledged this was a departure from best practice. Mr McGrath added that the hospital would like to take the opportunity to apologise[52] to Baby XY's family for not reaching a higher standard of care.

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[52] I refer to Section 70 of the *Coroners Act 2008* (Vic), which provides that an 'apology means an expression of sorrow, regret or sympathy but does not include a clear acknowledgement of fault'. In addition, Section 70 provides that an apology 'does not constitute an admission as to any matter for the purposes of findings that are made under section 67 or 68'.

68. By way of letter dated 19 December 2016, Ms Anne Shortall of Slater and Gordon Lawyers advised the Court that Baby XY 's family did not wish to prolong the coronial investigation, and had hence chosen not to file Dr Lyneham's report.

COMMENTS

Pursuant to section 67(3) of the **Coroners Act 2008**, I make the following comments connected with the death:

1. The course and emphasis of the coronial investigation into the death of Baby XY has been significantly influenced by the emergence of evidence that Ms YZ was suffering from undiagnosed gestational diabetes. Without question, if this information had not become retrospectively available, the interpretation of the CTG by clinicians on the evening of 5 and 6 May 2014, would have warranted a greater focus.

2. Indeed, the investigation has identified that significant systemic errors resulted in a failure to diagnose Ms YZ with gestational diabetes during her pregnancy. Ms YZ 's glucose tolerance test on 29 January 2014, at approximately 28 weeks' gestation, provided a two hour result of 8.6mmol/L, whereas the normal range is between 3.3 and 7.7 mmol/Litre.

3. It is concerning that the Melbourne Health Pathology Service's system operated so that only one of Ms YZ 's three 28 weeks' gestation GTT results would be downloaded onto her file and thus reported to clinicians. It further defies comprehension that this system error was only amended on 29 September 2014, some eight months after the initial test, and two months after Ms Pettigrew's enquiry on 18 July 2014. When two of Ms YZ 's GTT results remained outstanding, the responsibility rested upon her Western Health clinicians to follow up with the Melbourne Health Pathology Service. The fact this did not occur was an oversight with devastating consequences.

4. The evidence provided by Dr David Simon suggests that had Ms YZ – a woman with a family history of diabetes – been appropriately diagnosed with gestational diabetes, she would have been the recipient of very different antenatal care. In particular, I note that gestational diabetes increases the chance of macrosomia, shoulder dystocia and late stillbirth. As a result, clinicians involved in Ms YZ 's care would have been more vigilant and alert to the particular risk of these issues arising. Ms YZ would have in all likelihood been referred to a diabetes educator and dietician; been subject to daily self-monitoring of blood glucose levels; had an additional ultrasound in the late weeks of her pregnancy; continuous intrapartum foetal monitoring with CTG for the duration of labour; and possibly had an induction of labour prior

to term. In addition, knowledge of a woman with a suspected big baby may have influenced a doctor to proceed to caesarean section ahead of trialling instrumental delivery.

5. In circumstances where clinicians may have managed Ms YZ 's labour very differently had they had been informed she had tested positive for gestational diabetes, I make no adverse comment against individuals involved in her care on 5 and 6 May 2014. Dr Simon identified that the management of Baby XY 's shoulder dystocia, and the initial trial instrumental delivery in theatre were appropriate. While I note that Baby XY 's weight of 4.42kg was above the 97th centile for 41 week female babies, I also note Dr Simon's evidence that clinical estimation of foetal weight is unreliable and in the absence of diabetes or a fundal height greater than expected, there was no reason for the midwife to suspect Baby XY would be macrosomic.

6. Moreover, I note Dr Simon's view that if gestational diabetes had been known, a CTG would most likely have been in operation prior to 11.43pm, and a more 'baseline picture' would have been known to judge the significance of CTG features from this time. In addition, Dr Simon noted that only with the benefit of hindsight would urgent delivery have been required prior to 12.35am. Also, while an urgent caesarean section could have reasonably been ordered 25 minutes earlier than it actually occurred, this may not have been enough to save Baby XY from serious harm.

7. I note both Western Health and Melbourne Health Pathology Service have taken restorative action in the wake of this tragedy. In particular, I commend Western Health for introducing an improvement project with the purpose of placing the onus on clinicians who order tests to make proactive enquiries regarding any apparently incomplete results. It is to be hoped that in correcting the specific systemic error relating to this matter, Melbourne Health Pathology Service also put in place more general, widely-applicable risk minimisation procedures to prevent other technological errors from arising.

FINDINGS

The weight of the evidence leads me to find, on the balance of probabilities, that the main contributing factor to Baby XY 's death from perinatal asphyxia, was the failure to diagnose her mother, YZ , with gestational diabetes during pregnancy. Had this diagnosis been made, I find that Ms YZ 's antenatal care and the management of her labour would have been fundamentally different and her clinical team would have been significantly more informed about risks.

AND I find that if Ms YZ's clinicians had known she had gestational diabetes, and thus had a higher risk of a macrosomic baby, shoulder dystocia and late stillbirth, the changes to her care, including daily self-monitoring of blood glucose levels; clinicians' alertness to the risk of macrosomia and foetal distress; possible continuous CTG monitoring during labour; and possible induction of labour prior to term, Baby XY 's death could have been prevented.

AND I further find that the failure to diagnose Ms YZ with gestational diabetes was due to both a system error on the part of Melbourne Health Pathology Service, and the failure of Western Health to follow up outstanding Glucose Tolerance Test results contemporaneously.

I accept and adopt the medical cause of death as ascribed by Dr Yeliena Baber, and find that Baby XY tragically died from perinatal asphyxia.

Having regard to the potential educative value of this Finding for the wider community, pursuant to section 73(1A) of the *Coroners Act 2008,* I order that this Finding be published on the internet.

I direct that a copy of this finding be provided to the following:

Mr Vincent Argayoso

Mr Jayr Teng, Western Health Service

Mr Malcolm Mohr, Melbourne Health

Dr David Simon

Ms Anne Shortall, Slater and Gordon Lawyers

Mr Peter McGrath, K&L Gates

Ms Lisa Ridd, MinterEllison Lawyers

Professor Jeremy Oats, Consultative Council on Obstetric and Paediatric Mortality and Morbidity

Signature:

AUDREY JAMIESON
CORONER

Coroners Court of Victoria

Date: **24 January 2017**