IN THE CORONERS COURT
OF VICTORIA
AT MELBOURNE

Court Reference: COR 2009 003481

FINDING INTO DEATH WITH INQUEST

Form 37 Rule 60(1)

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

As amended on 18 December 2015 pursuant to *Section 76 of the Coroners Act 2008*

Inquest into the Death of: Baby Kylie HAMILTON

Delivered On:	4 December 2015
Delivered At:	Coroners Court of Victoria 65 Kavanagh Street Southbank Victoria 3006
Hearing Dates:	2 and 3 December 2013
Findings of:	Coroner Paresa Antoniadis SPANOS
Representation:	Mr R. HARPER of Counsel, instructed by Mr T. Ballantyne of Maurice Blackburn, appeared on behalf of Annette Cotter and Chris Hamilton, Baby Kylie's parents. Ms D. FOY of Counsel appeared on behalf of Monash Health (formerly Southern Health).
Police Coronial Support Unit	Senior Constable K. RAMSEY, assisting the Coroner

I, PARESA ANTONIADIS SPANOS, Coroner,

having investigated the death of Baby KYLIE ELYCE SARAH HAMILTON

and having held an inquest in relation to this death at Melbourne

on 2 and 3 December 2013:

find that the identity of the deceased was KYLIE ELYCE SARAH HAMILTON

born on 13 July 2009

and that the death occurred on 16 July 2009

at Monash Medical Centre, 268 Clayton Road, Clayton, Victoria

from:

I (a) PERINATAL ASPHYXIA

in the following circumstances:

BACKGROUND

1. Baby Kylie was the three-day old daughter of Annette Cotter and Chris Hamilton. Ms Cotter was 35 years old when she was pregnant with Baby Kylie, and had previously delivered a daughter by normal vaginal delivery at 41 weeks' gestation in 2001.[1] That child was diagnosed with cystic fibrosis at three years of age.[2]

2. During her second pregnancy, Ms Cotter's antenatal care was provided by Southern Health (now known as Monash Health).[3] She attended her first antenatal appointment in November 2008, when she was about six weeks pregnant.[4] Ms Cotter reported a medical history that included fibromyalgia, rheumatoid arthritis and asthma (managed with seretide), and that her last menstrual period had been in September 2008.[5] An obstetric ultrasound conducted on 12

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[1] Coronial Brief of Evidence [hereafter 'Exhibit O'], Ms Cotter's Monash Health Medical Records dated 6 January 2009.

[2] Ibid.

[3] Southern Health will be referred to hereafter as Monash Health.

[4] 'Exhibit O, Ms Cotter's Monash Health Medical Records dated 13 November 2008.

[5] Exhibit O, Ms Cotter's Monash Health Medical Records dated 6 January 2009. Ms Cotter reported a regular 28-day menstrual cycle.

January 2009 estimated a gestational age of 15 weeks and 3 days, making her estimated due date 3 July 2009.[6]

3. Ms Cotter initially attended the Monday Pregnancy Care Clinic, where she received counselling about cystic fibrosis and aneuploidy screening.[7] An ultrasound at 20 weeks' gestation indicated hypoplastic nasal bones[8] and Ms Cotter was offered a review by the Foetal Diagnostic Unit[9] but declined because the result of any definitive genetic testing would not alter her plans for the pregnancy. At 28 weeks' gestation, Ms Cotter was diagnosed with gestational diabetes mellitus.[10] Her antenatal care was transferred to the Tuesday Pregnancy Care Clinic which catered to pregnant women with diabetes. Ms Cotter's diabetes was well-controlled by diet.[11]

4. On 7 July 2009, during a routine antenatal appointment in the 40th week of her pregnancy, Ms Cotter reported a reduction in foetal movements. A cardiotocograph [CTG][12] was performed by the Maternal Foetal Medicine Unit and the results were reported as 'normal and reassuring'.[13] An induction of labour was scheduled for 13 July 2009, at 41 weeks and three days' gestation, [14] in accordance with Monash Health guidelines. [15]

5. Ms Cotter arrived at the Monash Medical Centre for a scheduled induction of labour at 7.00am on 13 July 2009, accompanied by her partner and her mother. On arrival at the birthing suite at 7.30am, Graduate Midwife [GM] Ebony La Posta noted Ms Cotter's vital

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[6] Exhibit O, Ms Cotter's Monash Health Medical Records – Diagnostic Imagine Report dated 12 January 2009.

[7] Exhibit C, Statement of Professor Euan Wallace. I note that Ms Cotter and Mr Hamilton recall that genetic counselling occurred on 17 November 2008 at the Southern Health Genetic Clinic.

[8] Hypoplastic nasal bones are potentially indicative of chromosomal disorder, Trisomy 21. I note that the results of a second trimester serum screen indicated that there was 'low risk' of Down or Edward Syndrome or neural tube defects [Exhibit O, Ms Cotter's Monash Health Medical Records – Diagnostic Imagine Report dated 19 February 2009].

[9] A specialist foetal anomaly and diagnosis unit at Monash Health.

[10] Exhibit L.

[11] Exhibit C.

[12] A technical means of recording the foetal heartbeat and uterine contractions during pregnancy and/or labour.

[13] Transcript page 156.

[14] This gestational age is contained in Exhibit A. Population studies indicate an increased perinatal and morbidity rate in otherwise uncomplicated pregnancies where gestation continues beyond 42 weeks. See transcript at pages 73-74 and 103 where gestational age is discussed. Estimated gestation at birth was noted as 42.4 weeks on the Maternity Discharge Summary. Note that the Guideline (see footnote 14 below) emphasises the importance of 'accurate dating' to determining whether or not a pregnancy is prolonged.

[15] Southern Health "Prolonged pregnancy management Guideline" version 1.8.

observations, obtained intravenous access, performed an abdominal palpation and auscultated the foetal heart rate [FHR].

6. At 7.45am the night duty Registrar, Dr Sushen Naidoo, performed a vaginal examination of Ms Cotter prior to commencing the induction of labour, noting that her cervix was 'favourable', two-to-three centimetres [cm] dilated and one-to-two cm long.[16]

7. At 7.55am Dr Naidoo performed an apparently successful amniotomy (an artificial rupture of membranes) which produced a small amount of clear fluid. He could not obtain a sample of Baby Kylie's hair to confirm that the forewaters were ruptured and so recommended this occur at the next vaginal examination. Dr Naidoo wrote a medical order for fluids and syntocinon.[17]

8. At 8.25am, GM La Posta applied a CTG to Ms Cotter's abdomen to assess Baby Kylie's wellbeing during the labour and at 8.43am commenced the syntocinon infusion at 12 millilitres per hour [ml/p/h] in accordance with Monash Health's protocol.[18] Shortly after, GM La Posta observed an unusual fluctuation of Baby Kylie's heart rate and reduced variability in the FHR on the CTG trace. [19] The significance of these observations to Baby Kylie's wellbeing, if any, was unclear and so the midwife sought the advice of Nurse Unit Manager, Karen Taylor. RN Taylor advised continuation of the induction, close monitoring of the CTG trace and that midwifery and medical staff be informed of any fresh concerns.

9. The management of Ms Cotter's labour and the delivery of Baby Kylie is the subject of contention and will be discussed in some detail below. Suffice for present purposes to say that at various points during the labour, the CTG trace was non-reassuring, the syntocinon infusion was stopped and re-started and further investigation, in the form of a foetal blood sample [FBS] analysis, was conducted in an effort to ascertain – or confirm – Baby Kylie's wellbeing. The medical staff involved considered that there were reassuring features that supported their

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[16] Exhibit O, Statement of Dr Rebecca Zachariah.

[17] Exhibit A. Syntocinon is a synthetic form of the hormone oxytocin, which is normally produced by a woman's body during labour to cause contraction of the uterus.

[18]" Induction of Labour , precautions, contraindication" and "Induction of Labour (IOL) – Syntocinon" both as @ December 2005

[19] Exhibit A.

plan to continue with the labour until 3.50pm when a Code Green was called for an emergency Caesarean delivery.[20]

BABY KYLIE'S CONDITION AT BIRTH

10. At 4pm on 13 July 2009, Baby Kylie was born via Caesarean delivery in theatre. There was meconium staining of the liquor and meconium was suctioned from the baby's mouth. Cord blood drawn at birth was tested about 25 minutes later[21] when arterial blood yielded a lactate result of 18.1mmol/L, a highly aberrant result.[22]

11. Baby Kylie, weighing 3685 grams,[23] appeared floppy and dusky at delivery[24] and was without a pulse. Resuscitation was commenced immediately and she was intubated, artificially ventilated and transferred to the Neonatal Intensive Care Unit at 4.45pm. Baby Kylie's Apgar scores[25] were zero at one and five minutes, one at 25 minutes, two at 30 minutes and three at 35 minutes after birth,[26] each critically low scores indicative of neurological damage.

12. In light of Baby Kylie's grave prognosis following neurological investigations confirming no brain stem activity, and after consultation with specialists and her family, a decision was made to withdraw life support. Baby Kylie died at 2.58pm on 16 July 2009.

CORONIAL INVESTIGATION – SOURCES OF EVIDENCE

13. This finding is based on the totality of the material the product of the coronial investigation of Baby Kylie's death. That is the brief of evidence compiled by S/C Kelly Ramsey of the Police Coronial Support Unit, the statements, reports and testimony of those witnesses who

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[20] As will be seen in paragraphs 29 and following below, this is a generalisation and oversimplification of the situation.

[21] GM Whitehouse noted a 20 minute delay between blood sampling and testing [Exhibit O, Ms Cotter's Monash Health Medical Records: 'Birth Summary'].

[22] Lactate levels in FBS of less than or equal to 4.1 mmol/L are 'normal', the range 4.2-4.8 mmol/L is 'pre-acidotic' and values greater than 4.8 mmol/L are indicative of acidosis, requiring immediate delivery of the foetus. See Exhibit O, extract from Royal Australian and New Zealand College of Obstetricians and Gynaecologists' 'Fetal Surveillance – A Practical Guide' (page 59, on foetal blood sampling).

[23] Exhibit O, Ms Cotter's Monash Health Medical Records – Maternity Discharge Summary

[24] Exhibit L.

[25] The Apgar score allows clinicians to quickly evaluate a newborn's physical condition. Five factors are each scored on a scale of 0 to 2 (with 2 being the best score) are used to evaluate the baby's condition: Appearance (skin colour), Pulse (heart rate), Grimace response (reflexes), Activity (muscle tone), Respiration (breathing rate and effort). The Apgar test is usually administered at one- and five- minutes after birth. Ten is the highest score possible, but is rarely obtained.

[26] Exhibit O, Ms Cotter's Monash Health Medical Records – Maternity Discharge Summary

testified at inquest and any documents tendered through them, and the final submissions of Counsel. All of this material, together with the inquest transcript, will remain on the coronial file.[27] In writing this finding, I do not purport to summarise all the material and evidence, but will refer to it only in such detail as is warranted by its forensic significance and in the interests of narrative clarity.

PURPOSE OF A CORONIAL INVESTIGATION

14. The purpose of a coronial investigation of a *reportable death* is to ascertain, if possible, the identity of the deceased person, the cause of death and the circumstances in which death occurred.[28] The *cause* of death refers to the *medical* cause of death, incorporating where possible the *mode* or *mechanism* of death. For coronial purposes, the *circumstances* in which death occurred refers to the context or background and surrounding circumstances, but is confined to those circumstances sufficiently proximate and causally relevant to the death, and not merely all circumstances which might form part of a narrative culminating in death.[29]

15. The broader purpose of any coronial investigations is to contribute to the reduction of the number of preventable deaths through the findings of the investigation and the making of recommendations by coroners, generally referred to as the *prevention* role.[30] Coroners are also empowered to report to the Attorney-General in relation to a death; to comment on any matter connected with the death they have investigated, including matters of public health or safety and the administration of justice; and to make recommendations to any Minister or public statutory authority on any matter connected with the death, including public health or safety or the administration of justice.[31] These are effectively the vehicles by which the prevention role may be advanced.[32]

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[27] From the commencement of the *Coroners Act 2008* (the Act), that is 1 November 2009, access to documents held by the Coroners Court of Victoria is governed by section 115 of the Act.

[28] Section 67(1) of the *Coroners Act 2008*. All references which follow are to the provisions of this Act, unless otherwise stipulated.

[29] This is the effect of the authorities – see for example *Harmsworth v The State Coroner* [1989] VR 989; *Clancy* v *West* (Unreported 17/08/1994, Supreme Court of Victoria, Harper J.)

[30] The 'prevention' role is now explicitly articulated in the Preamble and purposes of the Act, cf: the *Coroners Act 1985* where this role was generally accepted as 'implicit'.

[31] See sections 72(1), 67(3) and 72(2) regarding reports, comments and recommendations respectively.

[32] See also sections 73(1) and 72(5) which requires publication of coronial findings, comments and recommendations and responses respectively; section 72(3) and (4) which oblige the recipient of a coronial recommendation to respond within three months, specifying a statement of action which has or will be taken in relation to the recommendation.

16. It is important to stress that coroners are not empowered to determine the civil or criminal liability arising from the investigation of a reportable death, and are specifically prohibited from including in a finding or comment any statement that a person is, or maybe, guilty of an offence.[33]

FINDINGS AS TO UNCONTENTIOUS MATTERS

17. In relation to Baby Kylie's death, most of the matters I am required to ascertain, if possible, were uncontentious from the outset. Her identity and the date and place of death were not at issue. I find, as a matter of formality, that Kylie Elyce Sarah Hamilton, born on 13 July 2009, aged three days, died at Monash Medical Centre in Clayton on 16 July 2009.

18. Nor was the cause of Baby Kylie's death contentious. Forensic Pathology Registrar, Dr Marian Wang, of the Victorian Institute of Forensic Medicine [VIFM], performed a post-mortem examination of Baby Kylie's body, under the supervision of Forensic Pathologist Dr Melissa Baker, also from VIFM. Dr Wang reviewed the circumstances of the death as reported by the police to the coroner, medical records from Monash Health, postmortem CT scanning of the whole body [PMCT] and the results of ancillary investigations when preparing a written report of her findings.[34]

19. At autopsy, Dr Wang observed that the lungs contained numerous squames (skin cells) within the alveolar spaces, diffusely throughout all lung fields.[35] This finding indicated 'gasping' by a foetus in distress prior to delivery due to hypoxia.[36] Dr Wang also noted that the brain was oedematous (swollen) and histology showed features consistent with an hypoxic-ischaemic insult.[37] Dr Wang commented that these findings, together with the clinical history of foetal bradycardia, meconium-stained liquor, suction of liquor from the mouth and the Apgar score at birth, were consistent with meconium aspiration syndrome.[38]

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[33] Section 69(1).

[34] Exhibit K.

[35] Exhibit K.

[36] Transcript page 142.

[37] Exhibit K.

[38] Exhibit K. Dr Wang makes further comments about the indicia, causes and complications of meconium aspiration syndrome, however, in light of Dr Baber's qualification of the significance (and timing) of meconium in Baby Kylie's death, I have omitted them.

20. Dr Wang adopted the findings reported by Dr Virginia Billson following her histological examination of the placenta.[39] Dr Billson observed that the mature third trimester placenta she examined showed evidence of meconium staining, commonly associated with an acute process of severe foetal hypoxia.[40] There were also a number of chronic ischaemic hypoxic changes within the placental tissues and evidence of foetal thrombotic vasculopathy which raised the possibility of a maternal/foetal thrombophilic process together with longstanding uteroplacental circulatory compromise.[41]

21. Dr Wang identified no other natural or congenital disease, nor any injuries that may have caused or contributed to Baby Kylie's death.[42] Toxicology results were consistent with recent medical intervention (morphine was detected) and other ancillary investigations, namely microbiology, metabolic, cytogenetic and radiology, did not elucidate any other factors that may have contributed to death.[43]

22. Dr Wang was not available to give evidence at inquest and so her autopsy findings were admitted through Dr Yeliena Baber, a Forensic Pathologist who, due to her special interest and expertise, performs many of the paediatric autopsies at VIFM. Dr Baber stated that she agreed with Dr Wang's cause of death and with many of her concluding comments, save for the 'disproportionate'[44] emphasis given to meconium aspiration syndrome which Dr Baber regarded as a clinical diagnosis rather than a finding appropriate to the post-mortem setting.[45] She also noted that there was no evidence of meconium in the lungs, only squamous cells, suggesting that meconium was passed very late in labour.[46]

23. Dr Baber concurred with the cause of death formulated by Dr Wang, namely, that Baby Kylie's death was due to perinatal asphyxia.[47] She noted that "perinatal" was a generic term used by pathologists to encompass the antenatal and intrapartum period particularly when, as in Baby Kylie's case, interconnected processes – chronic and acute hypoxic events –

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[39] Exhibit K.

[40] Exhibit J.

[41] Exhibits J and K. I will return to Dr Billson's histological analysis of the placenta later.

[42] Exhibit K.

[43] Exhibit K.

[44] Transcript page 141

[45] Transcript page 144.

[46] Transcript pages 143-144.

[47] Exhibit K and Transcript page 141.

contributed to the death.[48] Later I will return to Dr Baber's evidence about the relative contribution of these processes to Baby Kylie's death.

24. On the basis of the evidence and advice provided by Drs Wang, Billson and Baber, I find that the cause of Baby Kylie's death is perinatal asphyxia.

FOCUS OF THE CORONIAL INVESTIGATION AND INQUEST

25. In common with many other coronial investigations, the primary focus of the coronial investigation and inquest into Baby Kylie's death was on the circumstances in which she died.
26. Ms Cotter's antenatal care was provided by Monash Health's Pregnancy Care Clinics, including one specifically for diabetic women. She was seen by a number of different doctors and midwives at the Clinics. As no issues about the adequacy of her antenatal care were raised by Ms Cotter or on her behalf during the investigation and inquest, the antenatal period was not the focus of the coronial investigation.
27. Similarly, there were no issues raised about the adequacy of the clinical management and care provided to Baby Kylie at Monash Medical Centre between her delivery by Caesarean section on 13 July 2009 and her death on 16 July 2009, and this period was not the focus of the coronial investigation.
28. The primary focus of the coronial investigation and inquest into Baby Kylie's death was on the obstetric management of Ms Cotter during labour by the medical and nursing staff at Monash Medical Centre. In this finding, it is convenient to examine Ms Cotter's intrapartum course into six periods, broadly delineated by key clinical decision points.

INDUCTION OF LABOUR AND SYNTOCINON INFUSION #1: 7.55am-10.25am

29. Throughout the morning shift, GM La Posta was the primary midwife caring for Ms Cotter. Ms Cotter's vital observations were satisfactory[49] and Baby Kylie's heart rate was initially noted to be 156 beats per minute [bpm], within the normal range.[50]

30. At 7.55am, Dr Naidoo performed an apparently successful amniotomy which produced a small amount of clear fluid.[51] He ordered that syntocinon be commenced and that a further vaginal examination occur in about four hours, once 'good contractions' were established.[52]

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[48] Transcript pages 150-151.

[49] Exhibit O, Ms Cotter's Monash Health Medical Records – Progress Notes 13 July 2009 at 8am.

[50] Exhibit O, Ms Cotter's Monash Health Medical Records – Progress Notes 13 July 2009 untimed (prior to 8.43am).

31. At 8.25am, the CTG was applied to Ms Cotter's abdomen.[53] A CTG uses ultrasound to record the foetal heart rate and uterine contractions via two transducers placed on the pregnant woman's abdomen. The transducers are connected to a monitor that displays the FHR numerically but the FHR is also audible to those in the immediate delivery area. The CTG produces a printed record – or trace – that plots each captured measurement.[54]

32. The CTG trace enables clinicians to determine the baseline foetal heart rate[55] and to 'see' variations,[56] accelerations[57] and decelerations[58] of the FHR and the frequency,[59] duration and apparent intensity of uterine contractions. Interpretation of the features documented by the CTG allow early detection of foetal distress and inform clinical management. The CTG trace is assessed by midwifery and obstetric medical staff. Indeed the ability to correctly interpret a CTG trace is a core competency for both professions.[60]

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[51] Exhibit A.

[52] Exhibit O, Ms Cotter's Monash Health Medical Records – Progress Notes 13 July 2009 untimed (prior to 8.43am).

[53] Exhibit A.

[54] Not all foetal heart beats or contractions will be recorded if there is suboptimal contact between the transducers and woman's abdomen. CTG transducers may be removed for short periods, such as to allow the labouring woman to take a toilet break, resulting in a gap in the CTG trace.

[55] The baseline foetal heart rate is the average heart rate of the foetus in a ten-minute period. A normal foetal heart rate is between 110 and 150 bpm.

[56] A degree of variability (variation of the FHR form one beat to the next) is regarded as a good indicator of foetal wellbeing. The amount of variability from the baseline FHR and its duration may be interpreted as 'reassuring', 'non-reassuring' or 'abnormal'.

[57] Accelerations are an abrupt increase in baseline heart rate of more than 15 bpm for greater than 15 seconds. Accelerations are considered reassuring and when they occur alongside uterine contractions are seen as a sign of a healthy foetus. The absence of acceleration (in an otherwise normal CTG trace) is of uncertain clinical significance.
[58] Decelerations are an abrupt decrease in baseline heart rate of more than 15 bpm for greater than 15 seconds. There are a number of types of decelerations, each with varying significance. An 'early deceleration' starts when a uterine contraction begins and resolves when the contraction ends. This type of deceleration is considered physiological rather than pathological. 'Variable deceleration' is seen as a rapid fall in baseline heart rate with a variable recovery phase; they may be variable in duration and may be unrelated to uterine contractions. Variable decelerations are often seen during labour, in patients with reduced amniotic fluid volume and are often caused by umbilical cord compression. Accelerations before and after a variable deceleration are known as the 'shoulders of deceleration' indicating that a foetus is not yet hypoxic and is adapting to reduced blood flow. Variable decelerations can sometimes resolve if the mother changes her position. The presence of persistent variable decelerations should be closely monitored and variable decelerations without 'shoulders' is suggestive of foetal hypoxia. A 'late deceleration' begins at the peak of uterine contraction and should resolve after the contraction ends. This type of deceleration indicates there is insufficient blood flow through the uterus and placenta and may produce foetal hypoxia and acidosis. The presence of late decelerations may indicate further investigation (such as foetal blood sampling) to determine whether the foetus is hypoxic and/or there is a need for emergency caesarean delivery. 'Prolonged deceleration' is a deceleration of more than two minutes' duration. Decelerations of two-to-three minutes are considered 'non-reassuring' while decelerations lasting more than three minutes are considered 'abnormal' and may require clinical intervention.

[60] Clinicians assess all aspects of the CTG to develop an overall impression (or clinical judgement) of its features as 'reassuring', 'non-reassuring' or 'abnormal'.

33. At 8.43am, syntocinon was commenced at infusion rate of 12 ml/p/h in line with the hospital's protocol. Shortly after, GM La Posta observed reduced variability in the FHR[61] and, about ten minutes later, 'variable decelerations'[62] and '? absent variability'[63] on the CTG trace. The midwife was unsure whether these were signs of foetal distress, given that the CTG and syntocinon had been used for about 30 minutes or less, or normal physiological responses such as a change in the foetal sleep phase or a reaction to the introduction of syntocinon.[64] Nonetheless, GM La Posta consulted her supervisor, RN Taylor, who advised her to continue the induction, closely monitor the CTG trace and escalate any further concerns to midwifery and medical staff.[65]

34. There was a short interruption to the CTG trace at 9.23am when the device was removed so that Ms Cotter could go to the bathroom. When it was reattached, Baby Kylie's heart rate dropped below the baseline (150-155 bpm)[66] and then returned. Ms Cotter was seated in a beanbag as she was starting to feel discomfort due to experiencing three mild-to-moderate contractions every ten minutes.[67] At 9.40am, the FHR decelerated from its baseline again, while day shift doctors Larmour and Teh were present, but they gave no direction for any change of management.[68]

35. Between 9.45 and 9.55am, following an increase of the syntocinon infusion rate from 24 ml/p/h to 36 ml/p/h[69] in accordance with the protocol, the CTG trace became 'increasingly abnormal'[70] with absent baseline variability and prolonged decelerations lasting up to three minutes in duration.[71] GM La Posta advised Baby Kylie's parents that she was concerned by

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[61] Exhibit O, Ms Cotter's Monash Health Medical Records – Progress Notes 13 July 2009 at 8.50am.

[62] Exhibit O, Ms Cotter's Monash Health Medical Records – Progress Notes 13 July 2009 at 9.15am.

[63] Exhibit O, Ms Cotter's Monash Health Medical Records – Progress Notes 13 July 2009 at 9.15am.

[64] Exhibit A and Transcript page 11.

[65] Exhibit A and Transcript page 9.

[66] Exhibit O, Statement of Dr Rebecca Zachariah.

[67] Exhibit O, Statement of Dr Rebecca Zachariah.

[68] Exhibit A.

[69] Exhibit O, Statement of Dr Rebecca Zachariah, and Transcript page 18.

[70] Exhibit A.

[71] Exhibit A.

changes in the FHR and that she would summon medical staff. At 9.58am Ms Cotter returned to her hospital bed in anticipation of an examination by a doctor.[72]

36. At 10am, Obstetrics and Gynaecology Registrar Dr Wan Teh attended in response to GM La Posta's concern about the CTG trace. Dr Teh examined Ms Cotter's abdomen for foetal position and then performed a vaginal examination which indicated that the amniotomy had been successful and that her cervix was effaced and dilated to 4cm.[73] Dr Teh reviewed the CTG trace, observing that there were 'a few non-reassuring features'[74] such as reduced baseline variability and atypical prolonged shallow variable decelerations.[75] Her management plan was for continued close monitoring of the CTG trace and a further vaginal examination in four hours.[76]

37. Following Dr Teh's examination, there were two further prolonged decelerations of Baby Kylie's heart rate.[77] At 10.11, GM La Posta remained concerned about the persistent decelerations of Baby Kylie's heart rate and so sought the advice of RN Taylor.[78] Following discussions with senior medical staff, the induction of labour continued with no change to the management plan. The FHR appeared to recover and remain stable for some minutes.[79]

38. At 10.19am, Baby Kylie's heart rate again decelerated, to a rate of 100 bpm. GM La Posta took Ms Cotter's pulse to ensure that the CTG was not recording the maternal heart rate by mistake. She ascertained that the CTG was in fact recording Baby Kylie's heart rate which had failed to return to its baseline for a period of three and a half minutes. GM La Posta suspected that the prolonged drop in the FHR[80] had been caused by uterine hyper-stimulation as Ms Cotter had experienced six or seven mild-to-moderate contractions in the previous ten

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[72] Exhibit A.

[73] Exhibit O, Ms Cotter's Monash Health Medical Records – Progress Notes 13 July 2009 at 10am [Dr Teh] and Exhibit L.

[74] Exhibit L.

[75] Exhibit L.

[76] Exhibit O, Ms Cotter's Monash Health Medical Records – Progress Notes 13 July 2009 at 10am [Dr Teh].

[77] Exhibit A.

[78] Exhibit A and Transcript page 20.

[79] Exhibit A.

[80] The normal range for FHR is between 110 and 160 bpm.

minutes.[81] The midwife ceased syntocinon at 10.25am, advised Obstetrics and Gynaecology Registrar Dr Larmour and requested a further review by Dr Teh.[82]

39. By 10.35am, Dr Teh had reviewed the CTG trace and noted a 'prolonged deceleration'[83] of the FHR and that overall the CTG was 'not improving'.[84] She also examined Ms Cotter's cervix which was dilated to 5 cm.[85] Dr Teh noted that no further decelerations of Baby Kylie's heart rate had occurred since the syntocinon infusion had been stopped.[86]

40. Dr Teh advised GM La Posta to recommence the syntocinon infusion at 11am at a rate of 12 ml/p/h. In the event that the FHR decelerated following recommencement of syntocinon, the infusion should be ceased immediately and Dr Teh called to perform a foetal blood sample [FBS] test.[87]

41. At about 10.52, GM La Posta observed that Baby Kylie's heart rate decelerated once again in the absence of syntocinon infusion. She summoned Dr Teh and RN Taylor in light of the 'abnormal' CTG trace.[88]

42. Dr Teh reviewed the CTG trace at 10.53am and observed that the baseline FHR was 140-145 bpm with reduced variability and prolonged decelerations lasting up to eight minutes.[89] She interpreted the ongoing decelerations of Baby Kylie's heart rate as 'non-reassuring'[90] and determined that a FBS test was indicated.[91]

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[81] Exhibit A.

[82] Exhibit A and Transcript page 20.

[83] Exhibit L.

[84] Exhibit O, Ms Cotter's Monash Health Medical Records – Progress Notes (written retrospectively in relation to) 13 July 2009 at 10.35am [Dr Teh].

[85] Exhibit L.

[86] Exhibit L.

[87] Exhibits L and O, Ms Cotter's Monash Health Medical Records – Progress Notes (written retrospectively in relation to) 13 July 2009 at 10.35am [Dr Teh].

[88] Transcript page 22.

[89] Exhibit O, Statement of Dr Rebecca Zachariah.

[90] Exhibits L and O, Ms Cotter's Monash Health Medical Records – Progress Notes (written retrospectively in relation to) 13 July 2009 at 10.53am [Dr Teh].

[91] Transcript page 161 and 162 and Exhibit L.

FOETAL BLOOD SAMPLE: 11am-11.17am

43. Although CTG is the most common method of foetal monitoring during labour, it is ill-suited to identifying foetal hypoxia. Thus, in the presence of a non-reassuring CTG trace, clinicians may seek to clarify the diagnosis by a conducting a FBS test. The test, using blood taken from the foetus' scalp capillaries, allows measurement of the lactate level in blood which is used as an indicator of foetal hypoxia. A foetal lactate level of 4.1 mmol/L or lower is considered 'normal'.[92]

44. Shortly after 11am, two unsuccessful attempts were made to obtain a FBS, while Ms Cotter was lying in the left- and right- lateral positions, before Staff Obstetrician, Dr Rebecca Zachariah, was called to assist.[93]

45. Dr Zachariah reviewed the CTG trace and agreed that a third attempt to obtain a FBS should be made.[94] Ms Cotter and her partner were told that if a FBS could not be obtained, delivery of Baby Kylie by Caesarean section would be advisable.[95] Dr Zachariah noted, however, that the CTG trace showed improvement in variability secondary to scalp stimulation during the previous FBS attempts and that this was 'usually associated with foetal wellbeing'.[96]

46. At 11.17am, a FBS was obtained by Dr Zachariah and the lactate level recorded as 3.1 mmol/L,[97] which is within the normal range. Dr Teh was reassured that there was no evidence of foetal hypoxia and that the labour could be continued[98] with the syntocinon infusion. When asked at inquest whether the treating team discussed repeating the FBS test sometime later, Dr

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[92] Lactate levels in FBS of less than or equal to 4.1 mmol/L are 'normal', the range 4.2-4.8 mmol/L is 'pre-acidotic' and values greater than 4.8 mmol/L are indicative of acidosis, requiring immediate delivery of the foetus. See Exhibit O, extract from Royal Australian and New Zealand College of Obstetricians and Gynaecologists' 'Fetal Surveillance – A Practical Guide' (page 59, on foetal blood sampling).

[93] Exhibit L.

[94] Exhibit O, Statement of Dr Rebecca Zachariah.

[95] Exhibit O, Statement of Dr Rebecca Zachariah.

[96] Exhibit O, Statement of Dr Rebecca Zachariah.

[97] Exhibits A, L and O, Ms Cotter's Monash Health Medical Records – Progress Notes (written retrospectively in relation to) 13 July 2009 at 11.17am [Dr Teh].

[98] Transcript page 159 and 164.

Teh testified that, pursuant to the hospital's protocols, if the lactate level is normal 'and there are no other clinical indications',[99] another lactate test would not be performed.

LABOUR AUGMENTED WITH SYNTOCINON [INFUSION #2]: 11.19am-11.25am

47. Pursuant to Dr Zachariah's orders, the syntocinon infusion was recommenced at 11.19am at a relatively low rate of 12 ml/p/h. At 11.20am GM La Posta observed that Baby Kylie had an episode of bradycardia lasting seven or eight minutes.[100] The midwife ceased syntocinon again at 11.25am and again informed RN Taylor and medical staff.[101]

48. At inquest, Dr Teh characterised the post-readministration of syntocinon change to Baby Kylie's heart rate as a 'prolonged deceleration'.[102] She confirmed that the deceleration was sufficient to justify turning off the syntocinon infusion again because Baby Kylie was not 'coping well' with it.[103]

49. Accordingly, at 11.25am, Dr Teh and her colleagues decided that Ms Cotter's labour should continue without augmentation. The clinical team considered that the labour would progress without syntocinon because Ms Cotter was a multigravida, and she was experiencing some contractions following the artificial rupture of membranes.[104] The midwife would continue to monitor the CTG trace and a further vaginal examination would occur around 2pm.[105]

FIRST PERIOD OF UNAUGMENTED LABOUR: 11.25am-2.15pm

50. After the syntocinon infusion was ceased on this occasion, Ms Cotter's contractions continued and the CTG trace was interpreted as 'normal'.[106]

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[99] Transcript page 159.

[100] Transcript page 24-25 and Exhibit A. I note that Ms Cotter's medical records, in a summary of 'CTG Report 10.20-11.35am' record a sequence of decelerations. The last of these is described as '2 consecutive prolonged decels down 30 beats lasting 3-4 minutes. In her statement, GM La Posta describes a 'further bradycardic episode lasting 7-8 minutes' following the FBS and in her evidence at inquest, she qualified her interpretations by describing the same deceleration as 'a borderline prolonged bradycardic episode of four-and-a-half to five minutes'.

[101] Exhibit A.

[102] Transcript page 162.

[103] Transcript page 162 and 164.

[104] Transcript page 163.

[105] Exhibit O, Statement of Dr Rebecca Zachariah.

[106] O, Ms Cotter's Monash Health Medical Records – Progress Notes 13 July 2009 at 1.15pm - 'CTG report 1135-1310'.

51. At about 12.20pm, Dr Zachariah reviewed the CTG trace. She observed that Baby Kylie's baseline heart rate was 135-140 bpm, with 'periods of adequate variability and no further deceleration'.[107] Dr Zachariah noted that a change in baseline is not uncommon in labour and, given Baby Kylie's normal lactate level and the absence of further persistent decelerations, the management plan agreed at 11.25am need not be changed.[108] Before leaving the hospital, Dr Zachariah provided an oral handover of Ms Cotter's course to the afternoon shift Senior Obstetric Registrar Dr Nicholas Bedford.[109]

52. At about 1.30pm, following a verbal hand over from GM La Posta, GM Andrea Whitehouse became Ms Cotter's primary midwife. She checked Ms Cotter's vital signs and performed an abdominal palpation noting a cephalic presentation,[110] longitudinal lie,[111] right occipital anterior position,[112] two-fifths above[113] the brim. Pink liquor was visible per vagina.[114] Ms Cotter was experiencing three mild-to-moderate contractions, lasting 45-50 seconds, in ten minutes. GM Whitehouse observed that Baby Kylie's baseline heart rate was 120 bpm.[115]

53. At 2.15pm, Dr Teh examined Ms Cotter. The Obstetric Registrar's observations were consistent with those of the midwife[116] and she also noted that Baby Kylie's head was at station -2[117] and that Ms Cotter's cervix was 5cm dilated as it had been nearly four hours

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[107] Exhibit O, Statement of Dr Rebecca Zachariah.

[108] Exhibit O, Statement of Dr Rebecca Zachariah.

[109] Exhibit N. Dr Zachariah left due to ill-health.

[110] Head down

[111] Foetus' spine is parallel to that of the mother

[112] Foetus' back is aligned with the right rather than left side of the mother's abdomen

[113] 'Two-fifths above the brim' means only 2/5th of the foetus' head can be felt above the pelvic cavity upon abdominal examination (that is, 3/5ths of the head has descended and that the head is 'moderately engaged').

[114] The midwives (and Ms Cotter's medical records) speak to the absence of amniotic fluid until 11.45am after a small amount of clear fluid was noted at the time of the amniotomy. Prof Wallace testified at inquest about the significance of the volume and colour of amniotic fluid at amniotomy, and that it was not uncommon in an induced labour for there to be little fluid released as the baby acts as a 'cork in a bottle' preventing them from passing through the cervix [100]. His comments were generally directed to the apparent time at which meconium was passed (in his view, late in the pregnancy, possibly around 3.45pm during sustained bradycardia) [Transcript page 102].

[115] Exhibit O, Ms Cotter's Monash Health Medical Records – Progress Notes 13 July 2009 at 1.30pm. In her statement [Exhibit B], GM Whitehouse recorded the FHR baseline as 125-130 bpm.

[116] On abdominal examination, Dr Teh assessed Baby Kylie's head as being 2-3 fifths above the brim (indicating that the head was *slightly* to moderately engaged).

[117] The 'foetal station' refers to the position of presenting part of the foetus in relation to the mother's pelvis. 0 station refers to the foetus' head being even with the ischial spines (the narrowest part of the pelvis). The foetus is said to be 'engaged' when the largest part of the head has entered the pelvis. If the presenting part lies above the ischial spines, the station is reported as a negative number from -1 to -5.

earlier. The CTG trace was interpreted as 'reassuring',[118] showing a baseline heart rate of 130 bpm, with normal variability and no further decelerations.[119]

54. At inquest, Dr Teh testified that the progress of Ms Cotter's labour was 'inadequate'[120] because the treatment team had been reliant on syntocinon to induce labour[121] but had been unable use it for lengthy periods and at a high enough rate to produce effective contractions due to Baby Kylie's difficulty coping with the infusion.[122] Nonetheless, she remained optimistic that the treatment team would be able to assist Ms Cotter to achieve a vaginal delivery,[123] particularly as she had given birth before.[124]

55. Thus, at 2.15pm, given Ms Cotter's 'slow progress' and the reassuring CTG trace, Dr Teh ordered that syntocinon be recommenced to augment labour but at an infusion rate of 6 ml/p/h, below the starting dose specified in the hospital's protocol.[125] Careful monitoring of the CTG trace was to continue and Ms Cotter was to be re-examined in about four hours.[126]

56. At inquest, Dr Teh rejected the suggestion that a Caesarean delivery should have been recommended[127] or at least canvassed with Ms Cotter[128] at 2.15pm for a failure to progress. Dr Teh enumerated the risks of Caesarean delivery for mother[129] and child,[130] and concluded that proceeding with vaginal delivery was better for the mother in circumstances were they

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[118] Exhibit L.

[119] Exhibit L.

[120] Transcript page 167.

[121] All of the obstetricians giving evidence at inquest acknowledged a difference between spontaneous and induced labours and the progress that could/should be expected.

[122] Transcript pages 164, 167-169: a view with which Dr Bedford concurred [at 187].

[123] Transcript 168, 170 and 171.

[124] Transcript page 164.

[125] Transcript page 167 and Exhibits L and O, Ms Cotter's Monash Health Medical Records – Progress Notes 13 July 2009 at 2.15pm [Dr Teh].

[126] Exhibit O, Ms Cotter's Monash Health Medical Records – Progress Notes 13 July 2009 at 2.15pm [Dr Teh].

[127] Transcript page 168.

[128] Transcript page 169.

[129] Transcript page 172.

[130] Transcript page 169.

were reassured by Baby Kylie's FBS lactate level and subsequent improvement of the CTG trace.[131]

LABOUR AUGMENTED WITH SYNTOCINON [INFUSION #3]: 2.15pm-3pm

57. At about 2.15pm, the syntocinon infusion was recommenced and GM La Posta covered Ms Cotter's care while GM Whitehouse took a meal break. Baby Kylie's baseline was 'within normal limits', at 125 bpm, when the infusion was restarted.[132] However, over a thirty-minute period between 2.30pm and 3pm, GM La Posta noted a gradual decrease in the baseline FHR from 125 bpm to 115 bpm, and then to 110 bpm,[133] before settling at a baseline bradycardia of 100bpm.[134]

58. Around 2.45pm, as GM Whitehouse returned from her break, GM La Posta was helping Ms Cotter change her position in an effort to correct Baby Kylie's baseline bradycardia.[135] Ms Cotter's intravenous fluids were also increased with the same intent. Drs Teh and Bedford were present, having been asked to review the 'unusual' CTG trace.[136]

59. Ms Cotter's partogram documents that syntocinon was ceased sometime between 2.45pm and 3pm.[137] It is not clear from the available evidence who determined that the infusion should be stopped or his/her reasons for doing so.[138] However, in the absence of uterine hyperstimulation,[139] it is reasonable to infer that the syntocinon infusion was ceased for a third time due to concern about the CTG trace.

60. GM La Posta described the change of Baby Kylie's baseline heart rate as 'not necessarily non-reassuring'.[140] She confirmed that the FHR had settled to a 'baseline bradycardia of 100 bpm'

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[131] Transcript page 170-171.

[132] Exhibit A.

[133] Exhibit A.

[134] Exhibit A.

[135] Exhibit B.

[136] Exhibit M.

[137] Exhibit O, Ms Cotter's Monash Health Medical Records – Partogram, 13 July 2009.

[138] Transcript page 27 [GM La Posta] and 189 [Dr Bedford]: both denied knowing the origin/rationale for the decision to cease syntocinon, indeed Dr Bedford [at 190] appears to indicate he would have continued the syntocinon.

[139] Transcript page 190. Uterine hyperstimulation (and its apparent effect on Baby Kylie) had been GM La Posta's justification for stopping syntocinon at 10.25am.

[140] Transcript page 27.

and that 110 bpm was 'the lower range of [a] normal' foetal heart rate. [141] However, she denied that Baby Kylie's heart rate at that time was a significant finding, and denied that the FHR was abnormal or non-reassuring.[142] Nonetheless, she testified that she had called in RN Leslie Baker of the Foetal Monitoring Unit 'because [she] was interested in her interpretation of the changes to the CTG' and to 'reconfirm we had taken the appropriate management' by ceasing syntocinon again.[143]

61. GM La Posta's evidence was that it is not uncommon for the FHR in post-maturity pregnancies, like Ms Cotter's, to settle at a lower rate of between 100-110 bpm. She added, instructively, that such lower baseline FHRs 'with accelerations present and no decelerations' and/or a 'heart rate between 110 and 160 [bpm] with normal baseline variability [are] reassuring feature[s] of a trace'.[144]

SECOND PERIOD OF UNAUGMENTED LABOUR: 3pm-3.50pm

62. Drs Teh and Bedford reviewed Ms Cotter at 3pm. They considered the gradual reduction in Baby Kylie's baseline heart rate as 'unusual' but 'not non-reassuring'.[145] Dr Teh noted that the baseline FHR was 110 bpm, with normal variability and no decelerations.[146] Ms Cotter was experiencing three mild-to-moderate contractions every ten minutes.[147] Dr Teh planned to leave the syntocinon infusion off, maintain careful monitoring of the CTG trace and obtain a second opinion from the Foetal Monitoring Unit.[148]

63. Although no note of RM Baker's advice about the CTG trace appears in Ms Cotter's medical records, it is evident that she attended at about 3.05pm.[149] Ms Cotter's treating team recall

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[141] Transcript page 27.

[142] Transcript page 27-28. However, I note that when asked why the syntocinon was turned off between 2.45pm and 3pm, GM La Posta explained that it was due to 'changes in the CTG that were non-reassuring' [28].

[143] Transcript page 28. Dr Bedford commented that the 'slow drop' [Transcript page 183] of Baby Kylie's heart rate was something he had not seen before (or since) and as he was unsure of its significance, he sought the advice of the FMU.

[144] Transcript pages 43-44.

[145] Exhibits L and M.

[146] Exhibit O, Ms Cotter's Monash Health Medical Records – Progress Notes 13 July 2009 at 3pm [Dr Teh].

[147] Exhibit O, Ms Cotter's Monash Health Medical Records – Partogram, 13 July 2009.

[148] Transcript page 167 and Exhibits L and O, Ms Cotter's Monash Health Medical Records – Progress Notes 13 July 2009 at 3pm [Dr Teh].

[149] Exhibit N.

being advised that though the trace was unusual, there were no worrying features to indicate any action beyond continued close observation.[150]

64. At about 3.10pm, GM Whitehouse resumed her role as primary midwife, observing that Ms Cotter's contractions were occurring four times in ten minutes and were of mild to moderate intensity without augmentation with syntocinon.[151] She also noted that the CTG trace indicated that Baby Kylie had a 'baseline bradycardia of 100 bpm'.[152]

65. At 3.15pm, Ms Cotter requested and received nitrous oxide as pain relief, due to increasing discomfort during contractions. This was considered a 'pretty promising sign'[153] that the contractions were increasing in intensity, that labour was no longer wholly dependent on the administration of syntocinon[154] and would proceed more quickly.[155]

66. At 3.20pm, GM Whitehouse informed Dr Larmour that Baby Kylie's baseline heart rate had decreased to 90 bpm and was fluctuating between 80 and 100 bpm.[156] Dr Lamour attended and reviewed the CTG trace but did not make any change to Ms Cotter's management. Ms Cotter asked for nitrous oxide to be decreased. A little after 3.30pm, Dr Lamour returned to review the CTG trace, which showed a baseline bradycardia of 100 bpm.[157]

67. At 3.36pm, Baby Kylie's heart rate was 45 bpm, with attempts to return to the baseline of 90 bpm. After about three minutes, the FHR returned to 90 bpm.[158] By 3.40pm, Ms Cotter's contractions were continuing at the same intensity and frequency, but she asked that pain relief be increased. She was in discomfort and as a result, the CTG transducer lost contact and was unable to record the FHR consistently.[159] GM Whitehouse summoned supervisory midwifery staff to assist her to locate the FHR and re-position Ms Cotter.[160]

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[150] Exhibit M.

[151] Exhibit B.

[152] Exhibit B.

[153] Transcript page 187.

[154] Transcript page 187.

[155] Transcript page 191.

[156] Exhibit B.

[157] Exhibit B.

[158] Transcript page 50.

[159] Transcript page 50.

[160] Transcript page 51.

68. At 3.42pm, medical staff were paged to Ms Cotter's room and, following a second page, Dr Teh arrived at 3.46pm.[161] GM Whitehouse auscultated Baby Kylie's heart rate, recording a rate of 70 bpm.[162] Dr Teh reviewed the CTG trace which showed bradycardia down to 80 bpm for up to eight minutes prior to her arrival.[163]

69. At 3.47pm, Dr Teh applied a foetal scalp electrode[164] which indicated that Baby Kylie's heart rate was 80 bpm.[165] Dr Teh called a 'Code Green' for an emergency Caesarean section delivery due to the evident foetal bradycardia.[166]

EXPERT OBSTETRIC EVIDENCE

70. At inquest, I had the benefit of the evidence provided by independent expert, Dr Michael Sedgley, Obstetrician & Gynaecologist, and Professor Euan Wallace, the Director of Obstetric Services at Monash Health,[167] in addition to the statements each[168] had provided during the investigation.

71. In his statements, Dr Sedgley raised two issues[169] relating to Monash Health's antenatal surveillance of Ms Cotter and Baby Kylie and, while he remained concerned that an amniotic fluid test had not been performed,[170] there was generally little disagreement between him and Prof Wallace at inquest[171] about this aspect of clinical management and care.

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[161] Exhibit B.

[162] Exhibit B.

[163] Exhibit L.

[164] A foetal scalp monitor is an electrode placed directly on the foetal scalp through the cervix which detects actual beat-to-beat electrical signals of the foetal heart to enable monitoring of the heart rate.

[165] Exhibit B.

[166] Exhibit L.

[167] Dr Sedgley and Prof Wallace testified concurrently; their evidence appears in the Transcript pages 63-120.

[168] Exhibits D, E and F [Dr Sedgley] and Exhibit C [Prof Wallace]. Exhibits G, H and I were also tendered through Prof Wallace.

[169] The two issues were: (1) that accurate management of Ms Cotter's post-maturity pregnancy was made more difficult by the relative inaccuracy of gestational age estimation from a 15-week ultrasound and (2) whether post-maturity monitoring should have been greater between 7 July 2009 (40th week check-up) and induction on 13 July2009, particularly in the context of reported reduced foetal movement on the earlier date [Exhibits D and F].

[170] Transcript pages 78-79.

[171] Transcript pages 75-78 and 79-81 [Prof Wallace's response, the latter being the remaining point of contention – no indications for amniotic fluid index in Ms Cotter's case in light of the hospital's guidelines].

72. Both Dr Sedgley and Prof Wallace agreed that spontaneous and induced labours progress, and are managed, differently.[172] Prof Wallace added that in an induced labour, like Ms Cotter's, it was not uncommon for clinicians to adjust, stop and re-start syntocinon in an effort to obtain the correct rate of dilation, without causing too many contractions or unduly distressing the baby.[173]

73. There was also agreement between Dr Sedgley and Prof Wallace about FBS testing during labour and the clinical benefits and limitations of CTG monitoring.[174] In relation to the FBS test, Prof Wallace testified that its results were likely to be very accurate,[175] but while the test was a useful measure of *acute* respiratory acidosis, it was a much less useful measure of *chronic* acidosis, that is, where there has been compensation for acidosis over time.[176]

74. Both Dr Sedgley and Prof Wallace considered that a CTG trace was a useful 'filter[177] but, on its own, was a 'fallible' test.[178] That is, while a "normal" CTG trace is extremely reassuring to clinicians, an abnormal CTG trace cannot tell clinicians how likely it is that a baby will actually be compromised due to poor oxygenation, as opposed to potentially so.[179] Nonetheless, Prof Wallace acknowledged that a multiplicity of certain kinds of abnormal features on a CTG made hypoxia increasingly more likely.[180]

75. There was also broad agreement between Dr Sedgley and Prof Wallace about the features of the Baby Kylie's CTG trace that made it 'not normal' from the outset.[181] They noted numerous non-reassuring features; namely, multiple, prolonged and persistent decelerations of the foetal heart rate, many of which were late in timing and associated with the administration

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[172] Exhibit E and transcript page 82 [Prof Wallace].

[173] Transcript pages 82-83.

[174] Transcript page 116. Prof Wallace testified that midwifery and medical staff at Monash Health are taught about the physiological bases of CTGs and FBS tests and their limitations [Transcript page 88].

[175] Transcript page 9.5

[176] Exhibit C [emphasis added].

[177] Transcript page 87.

[178] Transcript page 86.

[179] Transcript page 87.

[180] Transcript page 87.

[181] Transcript pages 109 [Prof Wallace] and 90 [Dr Sedgley].

of syntocinon;[182] decelerations that were not associated with uterine hyperstimulation;[183] and, reduced and/or absent variability of the FHR[184] even following the cessation of syntocinon.[185]

76. Both Dr Sedgley and Prof Wallace opined that these features were suggestive of foetal compromise,[186] the independent expert characterising the level of compromise as 'severe' and indicative of a lack of foetal reserve[187] while Prof Wallace testified that these were indicative of a chronically compromised foetus.[188] Although Prof Wallace observed that the care provided to Ms Cotter reflected that the midwives, registrars and consultant recognised that the CTG trace was not normal,[189] it was not evident from the medical record that they appreciated that the trace indicated prolonged foetal compromise.[190]

77. Dr Sedgley and Prof Wallace testified that it was reasonable for Ms Cotter's treatment team to conduct a FBS test after 11am in light of the non-reassuring CTG trace.[191] Both agreed that the lactate reading of 3.1mmol/L obtained at 11.17am was reassuring.[192] Prof Wallace stated that in light of it and the 'hint of [FHR] accelerations' associated with the test itself, he would have managed the labour as his staff did.[193] Dr Sedgley agreed that it was reasonable to continue with the induction of labour given the normal lactate result.[194] He commented that he would have proceeded as did Ms Cotter's treatment team 'for a while' in response to the FBS test result.[195]

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[182] Exhibits C and D.

[183] Exhibit F.

[184] Exhibit C and Transcript page 90.

[185] Exhibits C and D. I note Dr Sedgley's comment that there may be a delay between cessation of the syntocinon infusion and when it has cleared the mother's system, estimated to be around 20 minutes [Transcript page 105].

[186] Exhibits D and C.

[187] Exhibit E and Transcript pages 90 and 105.

[188] Exhibit C.

[189] Transcript page 89.

[190] Exhibit C.

[191] Transcript page 115-116 [Dr Sedgley] and Exhibit C.

[192] Transcript pages 114 [Dr Sedgley] and 88 [Prof Wallace].

[193] Transcript page 88.

[194] Transcript page 116. I note that Dr Sedgley's evidence in this regard represents a significant moderation of the opinion he provided in his statements in which he advocated Caesarean delivery of Baby Kylie at 11am. However, his original view – in light of his evidence at inquest – appears, at least in part, premised on the unavailability of FBS tests to him, in the hospital at which he worked in 2009 [see in particular, Transcript page 90 and 114 and 115].

[195] Transcript page 91.

78. Both experts observed that after about midday, the CTG trace normalised, was more reassuring, and the abrupt decelerations of the FHR ceased.[196]

79. The contention between the views of Dr Sedgley and Prof Wallace pertains to Ms Cotter's intrapartum course as at 2.15pm. At inquest, Dr Sedgley steadfastly maintained his opinion that management of Ms Cotter's labour ought to have been different at this time. Although acknowledging that it was difficult to be 'totally dogmatic'[197] about the management of another clinician's patient, he observed that there was evidence that a Caesarean delivery ought to have been recommended[198] at 2.15pm. By that time – more than five hours after the amniotomy, and after four hours of regular, mild-to-moderate contractions and two rounds of syntocinon – Ms Cotter's cervix dilation was 5cm and Baby Kylie's head remained at station -2, indicating that the labour had failed to progress.[199] In his opinion, one could not say with any degree of confidence that a vaginal delivery would be achieved 'any time soon'[200] and so his management would not have been to restart syntocinon[201] as Dr Teh had done.

80. Dr Sedgley agreed with Prof Wallace[202] that at 2.15pm there were not grounds to perform a Caesarean delivery due to foetal distress, given the more reassuring CTG trace after midday (and the cessation of syntocinon).[203] However, Dr Sedgley explicitly noted that there are 'all sorts' of other features of labour that are important to clinical management, not just a CTG trace.[204] He noted that the foetal compromise apparent on the CTG trace until 11am[205] and, particularly, the concern that Baby Kylie had poor reserves, were continuing worries throughout the labour.[206]

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[196] Exhibits C and F.

[197] Transcript page 106.

[198] Transcript page 109.

[199] Transcript pages 106-109.

[200] Transcript page 108.

[201] Exhibits D, E and F.

[202] Transcript page 110.

[203] Transcript page 106.

[204] Transcript page 117

[205] Transcript page 106

[206] Transcript page 109.

81. Prof Wallace testified that he would not have expected Ms Cotter's treating team to have performed a Caesarean delivery at 2.15pm.[207] Although his primary justification for that opinion was the more reassuring CTG trace after the FBS test, he acknowledged that though labour was an unpredictable process, Ms Cotter's labour had not progressed as quickly as one would have expected, particularly in a multiparous woman.[208]

82. Prof Wallace stated that he would not be 'hypercritical' of a clinician who had performed a Caesarean delivery at 2.15pm 'given anything that's gone before'.[209] He observed that at 2.15pm, it would have been reasonable to raise the possibility of a Caesarean delivery 'down the track' but though 'things looked a bit bleak' in terms of progress, he would have discussed the reassuring findings (the recent CTG trace and FBS result) and encourage Ms Cotter to continue a 'bit longer'.[210] With that end in mind, Prof Wallace opined that recommencing syntocinon at 2.15pm was a 'very, very reasonable' thing to do.[211]

83. Overall, there was substantial agreement between the two obstetric experts concerning the progress and management of Ms Cotter's labour until 2.15pm. Thereafter, the opinions of Dr Sedgley and Prof Wallace diverge, however, I am mindful that 2.15pm is a somewhat arbitrary point (perhaps an artefact of the inquisitorial process) on the continuum of Ms Cotter's labour and its clinical management. This is an observation acknowledged implicitly by Prof Wallace and explicitly by Dr Sedgley. Unfortunately, neither expert was asked to comment on the significance (or otherwise) of the gradual decrease of Baby Kylie's baseline heart rate to a 'baseline bradycardia' after 2.45pm, when syntocinon was used for the third time.[212]

84. Prof Wallace also gave evidence about the timing of Baby Kylie's acute hypoxia. He testified that the arterial umbilical cord blood sample analysed within 30 minutes, and which yielded a

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[207] Transcript page 109.

[208] Transcript page 110 and Exhibit C.

[209] Transcript page 110.

[210] Transcript page 111.

[211] Transcript page 110.

[212] Though I note Prof Wallace commented on the 'single prolonged deceleration at about 2.50pm' which he states occurred in association with 'sustained uterine contractions' and prompted cessation of the syntocinon infusion and review of the CTG trace by the FMU [Exhibit C].

lactate level of 18.1mmol/L indicating acute hypoxia,[213] was likely to be accurate.[214] Taking the FBS test result and that of the cord blood analysis together, Prof Wallace suggested that the acute hypoxic event occurred after 11.17am and before Baby Kylie's birth at 4pm.[215] While Prof Wallace could not eliminate the possibility that other events had contributed to the metabolic acidosis indicated by the cord blood analysis, he opined that the sustained foetal bradycardia at 3.50pm which precipitated Baby Kylie's delivery by emergency Caesarean was sufficient, in and of itself, to cause a lactate level of 18.1mmol/L.[216]

85. Prof Wallace conceded that the acute hypoxic event contributed to Baby Kylie's death[217] and that acute hypoxia could have been avoided by a Caesarean delivery at or before 3pm.[218] He noted that it was difficult to delineate the relative contributions of chronic and acute hypoxia to Baby Kylie's death.[219]

86. Both Dr Sedgley and Professor Wallace agreed that it was evident, probably only with hindsight, that Baby Kylie was significantly compromised prior to labour.[220] Prof Wallace observed that there was no clinical evidence that she was not developing normally[221] and, in particular, that Baby Kylie's weight at birth was not indicative of any form of growth restriction. Although acknowledging that maternal diabetes can confound assessment by making a compromised or 'sick baby seem bigger' or better,[222] Prof Wallace testified that, given that Ms Cotter's gestational diabetes was 'very well controlled with diet', he did not believe this had occurred in Baby Kylie's case.[223]

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[213] Transcript page 97.

[214] Transcript page 96. There had been concern that the delay of 25 minutes between the time of the blood sample and its analysis had compromised the results of the lactate test.

[215] Transcript page 97.

[216] Transcript pages 97-98.

[217] Transcript page 98.

[218] Transcript page 99.

[219] Transcript page 99.

[220] Exhibit F (dated 5 August 2013) and Exhibit C. I note that Prof Wallace suggested that the lack of 'accelerations' in Ms Cotter's pre-induction CTG trace [which he characterised as an antenatal CTG] was an abnormal finding. He noted that the absence of acceleration in a labour CTG would not necessarily be abnormal provided there were other reassuring features, like a normal baseline heart rate and variability [Transcript page 89].

[221] Transcript page 94.

[222] Transcript page 94.

[223] Transcript page 94.

EXPERT PATHOLOGISTS' EVIDENCE

87. Dr Virginia Billson, a Consultant Histopathologist with a specialisation in perinatal pathology, who examined histological slides of Baby Kylie's placenta, provided a written report and gave evidence at inquest.[224] Her evidence was that macroscopic examination of the samples demonstrated that the umbilical cord was slightly long and light in weight,[225] given Baby Kylie's gestational age; and that the former characteristic may be associated with entanglement of the cord and low oxygenation.[226] Dr Billson also testified that the membranes were meconium-stained implying the passage of meconium *in utero* due to hypoxia.[227]

88. The placental villi showed a number of chronic abnormal features: chorangiosis, an adaptation whereby the placenta produces more blood vessels than is normal to compensate for poor oxygenation and as such is associated with longstanding hypoxia;[228] foetal thorombotic vasculopathy (blood clots in vessels that can block blood supply to villi) a possible marker of a metabolic or genetic disorder;[229] and haemorrhagic endovasculitis (bleeding around vessels within the placenta) which is associated with foetal thrombotic vasculopathy.[230] Together, these features raised the possibility of a maternal-foetal thrombophilic process in addition to longstanding uteroplacental hypoxic change.[231]

89. Forensic Pathologist Dr Baber concurred with Dr Billson's evidence that the changes in the placenta indicated that uteroplacental circulation had been compromised and that this would have led to foetal hypoxia.[232] The pathologists agreed that chronic hypoxia had been present weeks or months prior to Baby Kylie's birth.[233]

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[224] Transcript pages 122-139.

[225] Transcript page 130 [based on an examination performed by others].

[226] Exhibit J and Transcript page 123.

[227] Transcript page 123.

[228] Exhibit J and Transcript pages 123 and 126.

[229] Exhibit J and Transcript pages 124 and 127.

[230] Exhibit J and Transcript pages 124-125.

[231] Exhibit J.

[232] Transcript page 149.

[233] Transcript pages 133 and 151.

90. Drs Billson and Baber were also in agreement that Baby Kylie was likely a 'macrosomic' but growth restricted baby.[234] There was reference to the clinical literature that states very clearly that a high proportion of babies born to well-controlled diabetic mothers are still significantly larger than those of non-diabetic mothers.[235] Dr Baber observed that the fact that Baby Kylie's birth weight was within the normal range of weights (and the weight of the placenta low for gestational age)[236] demonstrated that she was probably actually growth restricted given that her mother had gestational diabetes.[237]

91. Dr Billson identified acute hypoxic changes within the placental villi. The villi showed the presence of immature red blood cells within the foetal circulation (erythroblastosis) which is indicative of acute, severe foetal hypoxia with or without foetal anaemia.[238] Meconium staining was similarly characterised as an indication of acute hypoxia.[239]

92. Dr Billson commented that while in some cases a chronically hypoxic foetus may have greater respiratory drive,[240] pre-existing hypoxia is likely to increase the risk of acute hypoxia during labour.[241] She observed that is was very difficult to pinpoint the timing of Baby Kylie's acute hypoxia.[242] When asked, she estimated that the acute hypoxia-related changes to Baby Kylie's placenta were most likely to have occurred between one and 12 hours prior to birth.[243]

93. Dr Baber declined to comment on the timing of an acute hypoxic event beyond stating that it was unlikely to have occurred prior to the amniotomy given the absence of meconium staining from the liquor.[244] She concurred with Prof Wallace that Baby Kylie suffered an acute hypoxic event in the course of labour, emphasising that the lactate result of 18.1 mmol/L from

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[234] Transcript pages 133 [Dr Billson] and 142 [Dr Baber].

[235] Transcript page 154.

[236] Transcript page 153.

[237] Transcript pages 141-142.

[238] Exhibit J and Transcript page 124.

[239] Exhibit J.

[240] Transcript page 135 (such foetuses may have a greater ability to withstand hypoxic pressures).

[241] Transcript page 136.

[242] Transcript page 128.

[243] Transcript pages 128-129 and 137. Dr Billson testified that it would take at least an hour for the features she observed (erythroblastosis) to become established.

[244] Transcript page 146.

cord blood was indicative of metabolic acidosis precipitated by an acute event.[245] Dr Baber opined that it was not possible on the available evidence to exclude the possibility of more than one acute hypoxic event.[246]

94. The forensic pathologist testified that Baby Kylie's death was due to perinatal asphyxia, that is, the result of intrapartum hypoxic event(s) on a background of chronic hypoxia.[247] She observed that it was difficult to delineate the relative contributions of the two interconnected processes to Baby Kylie's death.[248] Indeed, on the basis of the autopsy findings, Dr Baber was unable to determine whether Baby Kylie would have survived absent an acute hypoxic event during labour given her chronic hypoxia due to compromised uteroplacental circulation.[249]

CONCLUSIONS

95. The standard of proof for coronial findings of fact is the civil standard of proof, on the balance of probabilities, with the *Briginshaw* gloss or explication.[250] The effect of the authorities is that Coroners should not make adverse findings against or comments about individuals, unless the evidence provides a comfortable level of satisfaction that they departed materially from the standards of their profession and in so doing, caused or contributed to the death.
96. It is axiomatic that the assessment of clinical management and care must be undertaken strictly without the benefit of hindsight. The trajectory of a patient's clinical deterioration may well be obvious after the event. Patterns or causal connections that can be traced from the privileged position of knowing the tragic outcome, may not have been obvious or even appreciable before that outcome.
97. In terms of the circumstances surrounding Baby Kylie's birth and death, the clinical management and care provided to her mother needs to be assessed against what was known, or

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[245] Transcript page 146.

[246] Transcript page 146.

[247] Transcript page 150.

[248] Transcript page 151.

[249] Transcript page 151.

[250] *Briginshaw v Briginshaw (1938) 60 C.L.R. 336 esp at 362-363.* "The seriousness of an allegation made, the inherent unlikelihood of an occurrence of a given description, or the gravity of the consequences flowing from a particular finding, are considerations which must affect the answer to the question whether the issues had been proved to the reasonable satisfaction of the tribunal. In such matters "reasonable satisfaction" should not be produced by inexact proofs, indefinite testimony, or indirect inferences..."

should reasonably have been known at the material time, that is when the nursing/midwifery and medical staff were caring for Ms Cotter during her labour and delivery.

98. Having applied the applicable standard to the available evidence, I find that:

a. Ms Cotter's antenatal care was reasonable and appropriate, including the decision to induce labour at 41 weeks and 3 days gestation in accordance with the relevant Monash Health protocol.

b. The clinical management and care provided to Ms Cotter between the induction of labour with an amniotomy at 7.55am and the commencement of the syntocinon infusion at 8.43am, and 2.15pm, immediately before the decision to recommence the syntocinon infusion for the third time, was reasonably and appropriate. That is, there was a sound clinical basis for each decision taken by nursing/midwifery staff and the doctors involved in Ms Cotter's management, including cessation of syntocinon, medical review of the CTG traces as requested, testing foetal scalp lactate and the involvement of the Foetal Monitoring Unit.

c. That said, the evidence before me supports a finding that by 2.15pm, there was a reasonable clinical basis to deliver Baby Kylie by Caeserean section, due to a failure to progress without augmentation with syntocinon, and a clear correlation between abnormalities in the CTG trace/FHR and the use of syntocinon.

d. Similarly, the evidence before me also supports a finding that the clinical decision to re-commence the syntocinon infusion at 2.15pm was also a reasonable clinical response, that is the approach taken at this time was within a range of reasonable responses to the circumstances as they pertained.

e. Unbeknownst to the clinicians involved in managing Ms Cotter's labour, Baby Kylie had been subjected to chronic hypoxia *in utero*, for weeks and possibly months before her birth, and was therefore likely to suffer more than usual with the normal rigours of a vaginal birth.

f. During the labour, Baby Kylie was also subject to several periods of acute hypoxia, against this background of chronic hypoxia, and had become increasingly compromised.

g. There was an opportunity at 2.45pm with all the clinicians present for reflection on Ms Cotter's overall clinical course up to that point, and a change in management to delivery by caesarean section, that *may* have led to a different outcome, with Baby Kylie born about one hour earlier and *potentially* less compromised.

h. However, it is not possible in my view to find with the requisite degree of certainty that, if born one hour earlier, Baby Kylie would probably have been born in a better condition, and/or would probably have survived, or to assess and to differentiate between the probable impact of chronic hypoxia *in utero* and the probable impact of episodes of acute hypoxia intrapartum.

i. The evidence does not support a finding, to the requisite standard of proof, that there was only one causally relevant episode of hypoxia, immediately before a Code Green emergency Caesarean was called.

j. Once the Code Green was called, the preparation of Ms Cotter for Caesarean section and Baby Kylie's delivery by Caesarean section were undertaken in a timely and appropriate way.

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

Baby Kylie's parents c/o Maurice Blackburn

Monash Health

Dr Michael Sedgely

Dr Virginia Billson

Consultative Council on Perinatal Morbidity and Mortality

Signature:

PARESA ANTONIADIS SPANOS
Coroner
Date: 30 November 2015