IN THE CORONERS COURT
OF VICTORIA
AT MELBOURNE

Court Reference: COR 2004 4579

FINDING INTO DEATH WITH INQUEST[1]

Form 37 Rule 60(1)

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

Inquest into the Death of: MARY ANNA POWLINA KORZENIEWSKI

Hearing Dates:	15-24 February 2010 & 30-31 August 2010, 1- 3 September 2010, 20 October 2010
Appearances:	• Dr Sharon Keeling of Counsel(15-24 February), Ms Geraldine Gray of Counsel (30 August – 3 September, 20 October) -Slater & Gordon -on behalf of the family - Jolant Flasza (partner), Peter Korzeniewski (brother) • Ms Maryanne Hartley SC - DLA Phillips Fox - on behalf of Ballarat Health Service • Mr John Constable of Counsel- John Ball & Sons -on behalf of Dr Moreno • Ms Dawne Galbally of Counsel- Middletons - on behalf of Midwives Ann Pullin and Cliff Adney
Police Coronial Support Unit:	L/S/C Greigory McFarlane - Assisting the Coroner
Findings of:	AUDREY JAMIESON, CORONER
Delivered on:	17 September 2013
Delivered At:	Coroners Court of Victoria Level 11, 222 Exhibition Street Melbourne 3000

[1] The Finding does not purport to refer to all aspects of the evidence obtained in the course of the Investigation. The material relied upon included statements and documents tendered in evidence together with the Transcript of proceedings and submissions of legal representatives/Counsel. The absence of reference to any particular aspect of the evidence, either obtained through a witness or tendered in evidence does not infer that it has not been considered.

I, AUDREY JAMIESON, Coroner having investigated the death of MARY ANNA POWLINA KORZENIEWSKI

AND having held an Inquest in relation to this death on 15-24 February, 30-31 August , 1-3 September and 20 October 2010

at the County Court of Victoria at Melbourne

find that the identity of the deceased was MARY ANNA POWLINA KORZENIEWSKI

born on 9 April 1967

and the death occurred on 29 December 2004

at Ballarat and District Base Hospital, Ballarat Health Service, Drummond Street, Ballarat 3350

from:

1(a) HYPOXIC BRAIN INJURY COMPLICATING CARDIORESPIRATORY ARREST DURING MANUAL REMOVAL OF PLACENTA (UNDER SPINAL ANAESTHETIC) FOLLOWING POSTPARTUM HAEMORRHAGE IN THE PRESENCE OF A PATENT FORAMEN OVALE

in the following summary of circumstances:

1. On 26 December 2004 at 8:37pm, Ms Mary Anna Korzeniewski[2] gave birth to a healthy boy, Tommy. The third stage of labour was incomplete. At approximately 9:45pm, Mary was transferred to the operating theatre for manual removal of the placenta. At approximately 10:33pm, she suffered a cardiac arrest. Resuscitation was initiated and maintained for approximately 40 minutes when there was a return of cardiac output. On return of her circulation, profound bleeding was seen and a decision was made to proceed with a hysterectomy. Mary's condition stabilised prior to transfer to the Intensive Care Unit (ICU). Subsequent investigations indicated that Mary had suffered irreversible brain injury. Active medical treatment was withdrawn and she died on 29 December 2004.

[2] Ms Korzeniewski's family requested that she be referred to as Mary during the course of the Inquest. For consistency, I have, in most part, avoided formality and also referred to her only as Mary throughout the Finding.

BACKGROUND CIRCUMSTANCES

2. Mary was born on 9 April 1967. She was 37 years old at the time of her death. She lived in Gordon with her partner, Jolant Flasza.

3. Mary had a history of obsessive-compulsive disorder and depression for which she was prescribed Clomipramine.[3] This was her first pregnancy (primigravida). She accessed antenatal care only once when she was in her third trimester. During pregnancy, Mary developed gestational diabetes but it remained under control throughout the remainder of and up to 23 December 2004, and she did not require treatment with insulin.

4. Mary's pregnancy was otherwise perceived as being uncomplicated. Her presumed due date was mid-January 2005.

SURROUNDING CIRCUMSTANCES

5. On 25 December 2004, at approximately 5:00pm, Mary was admitted to Ballarat and District Base Hospital (Ballarat Hospital) following spontaneous rupture of her membranes. She was approximately 37 weeks gestation. She was administered intravenous amoxicillin due to her positive group B Streptococcus carriage. Spontaneous labour did not follow and on 26 December 2004 at approximately 3:00pm, labour was induced using intravenous Syntocinon.[4] At 7:25pm, she was fully dilated and at 8:37pm, that is, just over five hours into her labour, Mary achieved a natural vaginal delivery (NVD) of a healthy boy, Tommy. Shortly thereafter, 10 units of intramuscular Syntocinon were administered to assist with placental separation.

6. The third stage of labour was delayed. At approximately 9:15pm, Mary was sat on a bedpan and asked to blow into a glove to encourage separation of the placenta. She passed approximately 200 mls of blood and blood clots into the pan[5] but the placenta did not separate.

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[3] Clomipramine is a tricyclic antidepressant.

[4] Syntocinon can be used to bring on (induce) labour. It can also be used during and immediately after delivery to help the birth and to prevent or treat excessive bleeding. Syntocinon is a man-made chemical that is identical to a natural hormone called oxytocin. It works by stimulating the muscles of the uterus (womb) to produce rhythmic contractions.

[5] Exhibit 1 – Statement of Jan Mitchell dated 15 December 2009.

7. At approximately 9:30pm, Obstetrics Resident, Dr Huthaifa Alobaidy,[6] telephoned the on-call Obstetrics Registrar, Dr Michael Veal, to advise him of the delay in the third stage of labour despite the implementation of *the usual techniques*, such as getting Mary to empty her bladder and applying continuous cord traction.[7] Dr Veal requested Dr Alobaidy to arrange for Mary to be transferred to theatre for manual removal of placenta (MRP).

8. At approximately 9:45pm, Mary was noted to be hypotensive and tachycardic. Her postpartum blood loss was estimated to be approximately 600mls and there was still no sign of placental separation. She was given rapid intravenous fluid replacement in the form of 1 litre of Hartmann's solution and 500 mls of Haemaccel[8] and transferred to the operating theatre for the MRP. She was administered a spinal anaesthetic by Anaesthetic Registrar, Dr Eleanor Moreno and Dr Veal proceeded to remove the placenta in its complete form. A blood clot measuring approximately 400mls was also removed. Dr Veal found the uterine cavity to be empty and there was little bleeding. He found the fundus to be lax so rubbed it and requested Dr Moreno to administer 10 units of Syntocinon and prepare an infusion of 40 units to run over four hours. At approximately 10:38pm, while a second-degree vaginal tear was being repaired, Mary was noted to look a bit "dusky" and then appeared to have a seizure. ST depression[9] was noted on her ECG followed by ST elevation[10] and then the rapid onset of ventricular fibrillation - Mary suffered a cardio-respiratory arrest. She was initially manually ventilated using a bag-valve facemask, then intubated. Aggressive resuscitation ensued and included the commencement of a blood transfusion. A number of medical specialists attended the operating theatre to assist in the resuscitation including the Anaesthetic Consultant, the ICU Consultant, the Consultant Gynaecologist and a Surgeon. Mary returned to sinus rhythm and developed extreme coagulopathy.[11] She was estimated to

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[6] Dr Alobaidy had changed his surname to "Ishmail" by the time of the Inquest however for consistency, I have referred to him as Dr Alobaidy throughout the Finding as this was his name at the time of Mary's death.

[7] Exhibit 10 – Statement of Dr Michael Veal dated 30 May 2006.

[8] Haemaccel (a registered trademark) is a type of intravenous colloid used in the prevention or treatment of shock associated with reduction in effective circulating blood volume due to hemorrhage, loss of plasma (burns, peritonitis, pancreatitis, crush injuries), or loss of water and electrolytes from persistent vomiting and diarrhoea. (Source: Wikipedia, the free encyclopedia).

[9] ST depression refers to a finding on electrocardiographs. The capital letters, in this case 'S' and 'T', refer to relatively large waves seen on electrocardiographs corresponding to electrical cardiac activity. ST depression can indicate myocardial ischemia (that is, inadequate blood supply to cardiac muscle) due to a myocardial infarction (that is, death of cardiac muscle due to inadequate blood supply).

[10] ST elevation is also evidence of cardiac ischemia.

[11] The extreme coagulopathy was later described as disseminating intravascular coagulopathy (DIC).

have bled approximately 1 litre of blood within a few minutes. After a period of aortocaval compression, the Obstetrics Consultant, Dr Dalton made a decision to perform an abdominal hysterectomy. During this procedure, there was no evidence of uterine rupture and no blood was found in the intraperitoneal cavity. Whilst still in the operating theatre, a Haemocue estimate found Mary's haemoglobin level to be low at 3g/dL. She received 12 units of blood and blood products in the operating theatre.

9. Mary was transferred to the ICU, ventilated and receiving inotropic[12] support. She remained unconscious and was demonstrating signs of significant neurological injury. Over the following 24 hours, Mary's neurological status continued to deteriorate. A CT scan of the brain on 28 December 2004 showed multi-focal cerebral hemispheric cortical and sub-cortical infarction. The appearance was not typical of global ischaemia on its own, and suggested an embolic phenomenon.

10. On 29 December 2004, resuscitative measures were withdrawn after two consultants confirmed brain death. Mary died soon after.

INVESTIGATION

Identity

11. The identity of Mary Anna Powlina Korzeniewski was without dispute and required no additional investigation.

The medical investigation

12. On 30 December 2004, Dr Matthew Lynch, Forensic Pathologist at the Victorian Institute of Forensic Medicine (VIFM), performed an autopsy on the body of Mary. Findings included evidence of hypoxic brain injury, pulmonary oedema and a probe patent foramen ovale[13] along with evidence of the recent hysterectomy. Dr Lynch commented in his report that prior to autopsy, the clinical impression was that Mary had died from complications of postpartum haemorrhage with possible contribution from amniotic fluid embolism (AFE)[14]

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[12] An agent which increases the force of cardiac muscle contraction.

[13] An opening in the septum between the right and left artria (chambers of the heart) that normally closes at birth.

[14] See Medical Deposition completed by Dr Robert Gazzard.

or anaphylaxis from an unidentified agent whilst under anaesthetic.[15] Dr Lynch reported that he considered a number of differential diagnoses. No significant natural disease was identified and examination of multiple sections from the lungs failed to demonstrate any evidence of intravascular foetal squames,[16] which Dr Lynch stated he would expect to see if there was evidence of significant AFE. Similarly, he found no demonstrable evidence of significant air embolism from opening the right ventricle under water.

13. The placenta and uterus had been retained by Ballarat Hospital and provided to Dr Lynch for examination. The uterus had not ruptured and 50mL of blood stained fluid was identified in the peritoneal cavity.

14. Toxicological analysis detected the antidepressant medication Clomipramine and the anaesthetic agent, Propofol. A post mortem trypase level was not elevated and no suitable ante-mortem blood specimens were taken and/or available around the time of the cardiac arrest to explore the differential diagnoses of anaphylaxis.

15. Dr Lynch reported that precise elucidation of the cause of death had proved somewhat problematic hence, why he ultimately adopted a discursive approach to the cause of death, which he ascribed to hypoxic brain injury complicating cardiorespiratory arrest during manual removal of placenta (under spinal anaesthetic) following postpartum haemorrhage.

Clinical Liaison Service

16. The Clinical Liaison Service (CLS)[17] reviewed the medical records on behalf of the Coroner and suggested that statements be obtained from relevant healthcare personnel with a view to clarifying the circumstances surrounding and the management of Mary's postpartum haemorrhage. An independent expert opinion was subsequently sought and obtained from

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[15] See notation in medical records by Anaesthetic Registrar, Dr Moreno.

[16] Flat, keratinised, dead cells shed from the outermost layer of a squamous stratified epithelium.

[17] The role of the CLS was to assist the Coroner's investigation into the nature and extent of deaths, which occurred during the provision of healthcare, and to identify potential system factors in healthcare related deaths. CLS personnel were comprised of practising Physicians and Clinical Research Nurses who drew on their medical, nursing and research experiences, skills and knowledge to independently evaluate clinical evidence for the investigation of reportable and reported healthcare deaths and to assist in identifying remediable factors that may assist in prevention and risk management in health services settings. The CLS was replaced with the Health and Medical Investigation Team (HMIT) in 2010. HMIT sits within the Coroners Prevention Unit, which was established in 2008 to strengthen the prevention role of the coroner. The unit assists the Coroner in relation to the formulation of prevention recommendations, as well as assisting in monitoring and evaluating the effectiveness of the recommendations.

Dr Jacqueline Smith, Obstetrician and Gynaecologist, and after correspondence from Dr Gerard Stainsby, Ananesthetist, was received by the court, an independent expert opinion was sought and obtained from Dr Crowhurst, Senior Consultant in Anaesthesia.

17. Issues identified and not sufficiently clarified in the course of the review included:

- the level of communication between the Obstetric Registrar and the Consultant
- the lack of observations in the Birthing Suite before transfer to theatre
- the amount of actual blood loss and how it was estimated
- an apparent lack of measurement of the haemoglobin level after the blood loss
- whether fluid resuscitation was appropriately tempered to the situation
- whether a spinal anaesthetic was appropriate in the circumstances
- the actual cause of Mary's rapid onset of extremis.

As a consequence I determined that an Inquest should be held.

INQUEST

Jurisdiction

18. At the time of Mary's death, the *Coroners Act 1985* (Vic) (the Old Act) applied.

19. The *Coroners Act 2008* (Vic) (the New Act) commenced operation on 1 November 2009. Schedule 1, section 7 of the new Act states "*Subject to clause 10, if the hearing of an inquest has begun under the old Act and the inquest is not completed before the commencement day, the old Act continues to apply on and from the commencement day to the inquest*". Clause 10 does not apply to these circumstances.

20. The question is whether I commenced an Inquest into Mary's death when I held the directions hearing on 14 July 2009. The definition of 'inquest' in the Old Act defines an inquest to include a formal hearing. This definition does not exclude steps taken in preparation for the commencement of an inquest. The New Act defines inquest to be a public inquiry that is held by the Coroners Court of Victoria in respect of a death or a fire. There is little guidance in the New Act as to what constitutes the commencement of an inquest.

21. At the directions hearing I made a determination that an inquest was to be held[18] pursuant to section 17(2)[19] of the Old Act, and touched upon the scope of the inquest, the issues related to the scope and listed the inquest for hearing.

22. I have concluded the directions hearing on 14 July 2009 was as a public hearing, not purely administrative in nature, and it marked the commencement of the inquiry into Mary's death.

23. The interested parties, although invited to do so, did not attempt to impress on me an obligation to prefer one Act over the other. The interested parties appeared to be in agreement that the significant differences between the two Acts related to the publication of this Finding and not the making of any recommendations, but the accountability of an entity to respond to any recommendation. The reliance on one Act over the other would not affect substantive findings. No interested party indicated that they would oppose publication of the Finding and similarly no interested party indicated that they would not respond to recommendations should I determine that the new Act applied.

24. I have determined therefore that the Old Act applies.

Evidence at Inquest

25. *Viva voce* evidence was obtained from the following witnesses at the Inquest:

- Registered Midwife Jan Mitchell
- Registered Midwife Ann Pullin
- Registered Midwife Cliff Adeney
- Dr Huthaifa Ishmail (Alobaidy at the time), Obstetrics Resident
- Dr Michael Veal, Obstetrics Registrar
- Registered Nurse Georgina Scott
- Dr Eleanor Moreno, Anaesthetic Registrar
- Emeritus Professor Norman Beisher, Obstetrician and Gynaecologist

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[18] Transcript (T), page 1, I stated "*I think it's absolutely appropriate that this is a matter that does go to inquest*".

[19] Section 17(2) A coroner who has jurisdiction to investigate a death may hold an inquest if the coroner believes it is desirable.

- Dr Gerard Stainsby, Anaesthetist
- Dr Jacqueline Smith, Obstetrician and Gynaecologist
- Dr Denys Fortune, Consultant Pathologist
- Dr John Crowhurst, Senior Consultant Anaesthetist.

Events prior to Mary's collapse

a) Blood loss and the question of hypovolaemia/unrecognised postpartum haemorrhage

The labour ward

26. Mary was stable post delivery of Tommy but the placenta was undelivered. Medical intervention was deemed necessary. Associate Nurse Manager, Cliff Adeney and Midwife Ann Pullin had just commenced their shift at 9:00pm and had received a general handover about Mary's situation at the labour ward desk.[20] For the following approximately 30 minutes, midwives Adeney and Pullin were the primary carers of Mary. Midwife Adeney stated that seeing to the removal of the placenta became his priority.[21]

27. At approximately 9:35pm, Midwife Adeney spoke to Mary about her delivery. She was sitting up in bed cuddling Tommy. She looked pale but was talking and orientated. For the purposes of transferring Mary to the operating theatre, Midwife Adeney reviewed her blood loss *by looking under the sheet at the pinkies/kylies*[22] *on which she was sitting* and *estimated her blood loss on the pinkies at that time to be around 400ml* – an amount that he did not consider excessive.[23] Midwife Jan Mitchell handed over to Midwife Pullin that she had estimated Mary's blood loss to be approximately 500mls, made up of 200mls at birth, 200mls into the bedpan when attempting to remove the placenta and a further 100mls onto a second kylie.[24] In evidence, there was some uncertainty whether the kylies had been changed or whether a clean one had been placed on top of the one placed under Mary for the delivery, which was a practice said to be common.[25] In her notes dated 1 January 2005,

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[20] Exhibit 5 - Statement of Cliff Adeney dated 14 December 2009, Exhibit 3 – Statement of Ann Pullin dated 15 February 2009.

[21] Exhibit 5 – Statement of Cliff Adeney dated 14 December 2009; T @ p 107.

[22] Absorbent pads with plastic backing clinically used to absorb bodily fluids.

[23] Exhibit 5 – Statement of Cliff Adeney dated 14 December 2009.

[24] Exhibit 1 – Statement of Nurse Mitchell dated 15 September 2009.

[25] T @ p 117.

Midwife Mitchell[26] said that she had changed the kylie pads under Mary after the delivery of Tommy and before she left the ward. After delivery she checked the uterus and noted it to be firm and contracted. In her evidence, Midwife Mitchell remained consistent about her estimate of the blood loss on the two kylies, but she could not be certain if she had changed the kylie or placed another on top.[27] She agreed that the practice of weighing the kylies is more accurate but it was not usual practice at the time. She said that as a midwife, she had to make those estimations every day and the accuracy of her estimates were based on experience.

28. Midwife Pullin said she did not change the kylie under Mary and she did not place either of the two under her that were left on the bed when Mary was transferred from the delivery bed to the theatre bed. Of these two kylies, she said that the bottom one had what appeared to be liquor mixed with approximately 200mls of blood on it, supporting the proposition that it was present under Mary at the time of birth and the top kylie contained approximately 200mls of blood.[28] Of this amount, approximately 100mls was from a "gush"[29] after Dr Alobaidy examined Mary and tried to remove the placenta. She also estimated approximately 50mls of blood was contained on perineal pads (also known as peri pads) she removed from Mary prior to her transfer to theatre that she had not included into her estimate of total blood loss.[30] Midwife Pullin also confirmed that she measured the 200mls in the pan that Midwife Mitchell had left in the pan room.

29. Overall, I found Midwife Pullin a credible witness who was consistent in her evidence about Mary's blood loss. I also accept her evidence that Mary was not actively bleeding at the time she was facilitating Mary's transfer from the delivery bed onto the theatre bed.

30. Dr Alobaidy stated that the gush of blood described by Midwife Pullin was from the umbilical cord when it snapped as he had attempted to deliver the placenta by controlled

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[26] Exhibit 2 – Notes of Jan Mitchell dated 1 January 2005 and Exhibit 1 – Statement of Jan Mitchell with amendments dated 15 September 2009.

[27] T @ pp 7, 9 and 29.

[28] Exhibit 3 – Statement of Ann Pullin dated 15 December 2009, T @ pp 76-77.

[29] Exhibit 3.

[30] Exhibit 3.

cord traction. This amount was therefore foetal blood, not maternal blood.[31] He estimated this loss at about 50mls. He said that Mary was not actively bleeding at the time.[32]

31. After midwives Adeney and Pullin had transferred Mary to theatre, they rechecked the blood loss on their return to the labour ward and concluded that the estimated blood loss was 600mls.[33]

32. I find that the total amount of estimated maternal blood loss prior to theatre remained consistent at about 550-600mls, a third of which was measured in a jug in the pan room by Midwife Pullin. Dr Jacqueline Smith, who gave evidence as an independent expert, said that an estimated blood loss of 550mls in this setting is "nothing unusual".[34]

(ii) Mary's vital signs and clinical appearance pre-operatively

33. At approximately 9:45pm, Mary's clinical state appeared to change. In preparation of her transfer to theatre, Midwife Pullin recorded Mary's vital signs on a pre-anaesthetic sheet – blood pressure (BP) was 120/80mmHg, heart rate was 100 beats per minute (bpm), but she later said that the pulse was "weak and thready". According to Midwife Pullin, Mary also looked pale and had cold, clammy skin. She reported her observations to Midwife Adeney who in turn took Mary's BP and pulse. He obtained a BP of 60mmHg systolic and a pulse of 140 bpm.[35] He was concerned about this apparent change in Mary's condition, although she remained orientated and was not complaining of feeling unwell.[36] Midwife Adeney did not record these vital signs but commenced setting up for the introduction of a second intravenous line for fluid resuscitation and requested that someone summon Dr Alobaidy. Midwife Adeney did not initiate any other action in particular, he did not call a Code Blue/MET call and both Midwife Pullin and Dr Alobaidy said in evidence that they were not told of these vital signs by Midwife Adeney.

34. Around the time that Midwife Adeney obtained the BP of 60mmHg systolic, there was no other evidence of vascular shutdown. Dr Alobaidy inserted a second intravenous line

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[31] T @ p 176.

[32] T @ p 175.

[33] T @ pp148-149.

[34] T @ 1104.

[35] Exhibit 5 – Statement of Cliff Adeney dated 14 December 2009.

[36] T @ p 109.

without difficulty. He recorded a BP of 90/60mmHg and ordered the commencement of colloid fluid replacement in the form of Haemaccel 500mls[37] and Hartmann's solution1000mls. Dr Alobaidy said that Mary was pale but had no signs of hypovolaemic shock.[38] Mary was lucid at this time and signed the consent form for MRP.

35. Before leaving the labour ward for the operating theatre, Midwife Pullin recorded Mary's observations on the Anaesthetic Chart. Blood pressure is recorded to be 120/80mmHg[39] and her pulse was 100 bpm. Midwife Pullin did not record the time she took these observations but the time was estimated to be approximately 9:45pm. On the way to theatre, Midwife Pullin "pumped"[40] intravenous fluids into Mary at the direction of Midwife Adeney. On arrival at theatre at approximately 10:00pm, the BP recording of 60mmHg systolic was not given in handover to Anaesthetic Nurse Georgina Scott who received Mary in theatre. In the handover to Nurse Scott, Midwife Pullin advised her that Mary's BP in the ward had been 90mmHg systolic but Nurse Scott could not recall if this blood pressure reading preceded Midwife Pullin's own recording of 120/80mmHg on the pre anaesthetic sheet. The pumping of fluids was not reflected on any charts received in theatre but Nurse Scott recalled being told by Midwife Pullin that Mary had received approximately 1500mls[41] of fluid and she recalled seeing Midwife Pullin pumping the Hartmann's drip.[42] There was no handover or recording of Mary's pulse having been thready and despite Midwife Adeney being involved in the transfer of Mary to the operating theatre, he did not provide a handover to Nurse Scott about his detection of a BP of 60mmHg systolic.

36. The evidence of Midwife Adeney that Mary's BP was 60mmHg systolic is difficult to reconcile with the preponderance of evidence including his own that Mary showed no other signs of shock at that time.[43] However, I do not need to reject his evidence on this issue outright because I am satisfied that if he did hear a systolic BP of 60mmHg systolic, it was an aberration from which Mary appeared to be recovering from even before fluid resuscitation was implemented. Midwife Adeney's response was to facilitate the insertion of

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[37] T @ p 271.

[38] T @ p 194.

[39] T @ p 93, 95.

[40] T @ 69, 99-100.

[41] T @ p 524.

[42] T @ p 530.

[43] T @ p 122, 131.

an additional intravenous line for rapid fluid replacement. Rapid fluid replacement was an appropriate treatment response to an episode of postpartum hypotension. Professor Beischer, who also provided expert evidence, said he would not criticise this treatment.[44] Furthermore, I do not accept that an *isolated* reading of a blood pressure of 60mmHg systolic, absent other clinical indices of heamodynamic compromise, which was showing spontaneous signs of improving before treatment commenced and from which Mary had recovered before being received at theatre, is indicative of hypovolaemia or hidden bleeding.

37. Having considered all the evidence on the presentment and the significance of an isolated blood pressure of 60mmHg systolic, I accept the opinion of Dr Smith who said that transient hypotension in the labour ward after acute blood loss was not unusual and the rapid response of the rise in blood pressure after intravenous fluids were administered indicated that Mary had not been substantially hypovolaemic. Dr Smith said if not all, then most of Mary's blood loss had been replaced preoperatively such that she should have been stable under anaesthetic.[45] Dr Smith also said that it was not particularly significant if Mary's blood pressure had indeed been 60mmHg systolic in the labour ward because according to Dr Smith, that particular episode of hypotension in the ward bore no correlation to what subsequently occurred in theatre.[46] I accept Dr Smith's opinion in this regard and find that the discovery of a blood pressure of 60mmHg systolic was *one of the background circumstances, that is to say (sic) non-causal.*[47]

(iii) In theatre

38. On arrival at theatre, Mary had three intravenous infusion lines set up to be delivered via two intravenous cannulae. One line had 500mls of Haemaccel still connected, but which had run through. The Hartmann's solution line and the Syntocinon infusion line were piggybacked to be administered through the same cannula. A one litre flask of Hartmann's solution was running and approximately 200mls were remaining. The Syntocinon infusion (10 units in 1 litre) had approximately 100mls remaining.[48] The evidence of whether the Syntocinon infusion was still running when Mary arrived at theatre was equivocal. In her

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[44] T @ p 824.

[45] T @ p 1091.

[46] T @ pp 1112-1113.

[47] *Keown v Kahn* [1999] 1 VR 69, @ 76.

[48] Exhibit 13 – Statement of Georgina Scott dated 15 December 2009.

statement[49] Nurse Scott said it was running at the time but in her *viva voce* evidence said it was turned off.[50] Midwife Adeney said it was "running" in his retrospective Nursing Progress notes, but in his *viva voce* evidence thought this unlikely, as Mary was receiving rapid fluid replacement on the way to theatre with Haemocell and Hartmann's solution and Syntocinon infusions are never "pumped" in.[51]

39. It seems unlikely that the Syntocinon infusion was still running when Mary arrived at theatre. The submissions made on behalf of the family that the Syntocinon infusion was inappropriately ceased are based on the premise that Mary had suffered a postpartum haemorrhage following her labour. I address the issue of postpartum haemorrhage later in my Findings however, I have not identified any evidence that the subsequent events or outcome is causally connected to the running of the Syntocinon infusion at this time.

40. Mary was transferred into the anaesthetic room and Nurse Scott performed routine observations. Mary was sitting upright, was conversing normally, answering questions[52] and complained of back pain. She was not pale, cold or clammy according to Nurse Scott.[53] In the pre-operative anaesthetic area, Nurse Scott recorded Mary's BP as 135mmHg systolic and that her radial pulse was strong.

41. Dr Veal was surprised to hear that fluids had been pumped into Mary. Nevertheless, he said that even if he had known this, he would not have changed the course of management that he took because he believed Mary to be haemodynamically stable by the time she was in the operating theatre. He said that an episode of hypotension would not have altered the treatment that he subsequently implemented.[54]

42. I accept the evidence of Nurse Scott, Dr Moreno and Dr Veal, that Mary was haemodynamically stable when she was received into the operating theatre.

43. In respect of the blood loss in theatre, Dr Veal said that he generally tended to overestimate blood loss, but his estimate of the 400mls blood clot was also done on visualising it within a

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[49] *Op cit.*

[50] T @ p 522.

[51] T @ p 180.

[52] T @ p 523.

[53] T @ p 523.

[54] T @ p 508.

550mls capacity kidney dish.[55] He was confident that this clot was a retro-placental clot – it was easy to identify and it was easy to remove the placenta. Once removed, he rechecked the uterus as being empty,[56] that is, there was no concealed blood loss within the uterus.[57] As such, I have no reason to doubt this *estimated* amount of 400mls because it was an estimated within a receptacle with an objectively determined volume.

(iv) Overall blood loss before Mary's collapse

44. Midwife Mitchell attended on Mary during her labour and delivery. She estimated the amount of blood loss in labour to be 200mls. She said this was a usual volume of blood loss during labour [58] and she was confident about her estimation. The total estimated blood loss in the labour ward before transfer to theatre was 600mls. This was checked by Midwife Adeney when he returned to the ward after Mary's transfer to theatre,[59] and he had a discussion with Dr Veal confirming this estimate after Mary's collapse. Professor Beischer said that a postpartum blood loss of 1000-1200ml can be withstood by a healthy young woman without there being significant hypovolaemia.[60] Dr Smith said for a major collapse and for a woman to be in serious risk of dying, she would expect to see a blood loss in the vicinity of 3 litres.[61] Dr Dalton said that when he came into the theatre, he saw what he estimated to be approximately 600ml of blood that in his opinion was within the normal range expected with a MRP.[62]

45. Professor Beischer's conclusion and use of the word "exsanguination" to explain Mary's cause of death was regrettable. He did not have the entire medical record at the time he completed his report. He based his opinions on the belief that only blood loss could have caused the events that occurred and *apropos* of that belief, there must have been unobserved and/or unrecorded blood loss. When Professor Beischer heard all the relevant facts, he ultimately accepted that it was unlikely that there was any significant unobserved,

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[55] T @ p 398.

[56] T @ p 399.

[57] T @ p 486.

[58] T @ p64.

[59] T @ pp 126-127.

[60] T @ p 820, 862.

[61] T @ p 1092.

[62] T @ p 294.

unrecorded blood loss[63] and that he had exaggerated when using the word "exsanguination"[64]in his report.[65]

46. Dr Fortune[66] and Dr Crowhurst[67] also reported that blood loss had played a role in Mary's death but similarly had not been in possession of the whole clinical record. Once aware of all the relevant facts, they were similarly less firm in their respective opinions.

47. It was the family's submission that:

> *....the cause of Mary's death was cardiac arrest, which arose by reason of hypovolemia and hypotension (sic), consequent to the combination of unrecognised and untreated significant postpartum haemorrhage and spinal anaesthesia. The reason for that was because Mary suffered from significant unrecognised postpartum haemorrhage because of the likely volume of blood loss being higher than estimated by the midwives. That was because the visual estimation of the blood loss was unreliable, and it was known at the time to be unreliable.*[68]

48. In other words, the family submits that the combination of factors that contributed to Mary's collapse arose from the under-observation and underestimation of blood loss by the midwives. This in turn led to a general inadequate clinical assessment and volume resuscitation before the administration of a spinal anaesthetic. The family rejected the hypotheses of AFE and/or air embolism. The cause of Mary's death was, according to the family, – *hypotension (sic) consequent to spinal anaesthesia, complicating under treated significant postpartum haemorrhage.*[69]

49. An estimation of blood loss is no more than that – a rough calculation of the quantity or extent of something. In Mary's case, the estimation of blood loss and the uncertainty about

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[63] T @ pp 913-914.

[64] T @ p 934.

[65] Exhibit 19 – Correspondence/reports of Dr Norman Beischer dated 25 November 2008, 6 February 2009 and 28 January 2010.

[66] Exhibit 34 – Statement of Dr Denys Fortune dated 10 July 2010.

[67] Exhibit 39 – Statements of Dr John Crowhurst dated 31 March 2009, 26 April 2009 and 11 January 2010.

[68] T @ p 1384.

[69] T @ p 1385.

the antecedent cause(s) of her collapse have been assumed to equate to an underestimation of blood loss. Arguably the calculation of the amount of blood loss lacked rigor because some of the estimations were not based on any comparable weighing of wet kylies/ pinkies and the estimation by one nurse was not checked at the time by a second nurse. Furthermore, the evidence of the number of changes of kylies/pinkies could not be substantiated by examination of the clinical record because the documentation of blood loss also lacked rigor. However, I am not prepared to dismiss the experience[70] and thus capacity of the midwives to estimate blood loss during *normal* deliveries and when the evidence of the midwives was scrutinised, their estimation of blood loss remained consistent. I accept that at all times in the labour ward and then subsequently in theatre, Mary's blood loss was being observed, accounted for, treated and then considered within the differential diagnoses, as a possible cause of Mary's arrest.

50. Scrutinising the exact measurement of blood loss in isolation to Mary's holistic clinical presentation was fraught. Similarly, an attempt to retrospectively conclude that Mary was in moderate shock by reference to the Royal Women's Hospital guideline, *Anaesthetic Management of Postpartum Haemorrhage*[71] fails to take into account that Mary was being observed in a controlled environment where her blood loss was being observed.

b) Haemoglobin (Hb) level and the Haemacue result

51. I find that the history of rectal blood loss in or around 2001 is of no consequence in relation to the monitoring of Mary's haemoglobin level during her pregnancy and post delivery. She underwent two colonoscopies with no abnormalities detected. There was no evidence of any subsequent complaints of rectal blood loss.

52. Mary's haemoglobin level[72] was checked on 28 October 2004 during her antenatal check-up. She was not anaemic at this time. According to Dr Smith, this is in accordance with current practice, and that an Hb would normally be performed the day after delivery. Up until the time of the onset of profound coagulopathy after the return of cardiac output following Mary's arrest and resuscitation, there was no apparent clinical indication to re-

[70] T @ p 26 (Nurse Mitchell) & T @ p 70 (Midwife Pullin).

[71] Exhibit 9 - The Royal Women's Hospital - 'Anaesthetic Management of Post-Partum Haemorrhage' dated 23 November 2009.

[72] The normal haemoglobin level for adult females is 120-150 g/dL.

check Mary's haemoglobin level earlier or perform coagulation studies, as her situation was not out of the ordinary.[73] Dr Stainsby agreed that performing an Hb level before Mary left the labour ward would provide no information relevant to acute blood loss. Fluid resuscitation is determined from the clinical setting and an Hb level is more helpful after fluid resuscitation has been commenced so as to assist in determining whether an appropriate balance between blood and non-blood products has been given.[74] Dr Beicher was not critical that that no blood had been cross-matched before Mary went to the operating theatre as O negative blood was available if a transfusion had been indicated.[75]

53. In relation to Haemacue result obtained in the operating theatre, the preponderance of evidence was that the result of 3g/dL was inaccurate, could not be relied upon and was not relied upon. According to Dr Dalton:

> *It really doesn't give any useful information and it's less reliable in situations where there's significant bleeding or there's some other cardiovascular compromise.*[76]

54. I am satisfied that there was no therapeutic utility to be gained by performing an Hb, or cross matching blood for Mary before transporting her to the theatre for the MRP. The lack of the information of her Hb level played no role in the decisions that were made consequent to her collapse. I am also satisfied that there was no delay in transfusing Mary with blood products once she developed coagulopathy.

c) The use of spinal anaesthesia in the circumstances

55. I accept that Dr Moreno spoke to her consultant, Dr Neil Shorney after she received the call that Mary was coming to the theatre. I have no reason to doubt her evidence that she spoke to Dr Shorney about the type and dosage of anaesthetic agent to administer to Mary.

56. Dr Moreno was aware of the estimated blood loss of 600mls from the labour ward. Dr Moreno spoke to Mary. She was alert. She was sitting up. There were no signs of respiratory distress. Her blood pressure was 130mmHg systolic and her pulse was 100 bpm. Dr Moreno spoke to Mary about the administration of a spinal anaesthetic and the lack of cognitive

[73] T @ p 1104.

[74] T @ pp 1063-1064.

[75] T @ p 877.

[76] T @ p 301.

impediment following a spinal anaesthetic compared to a general anaesthetic. There were no contraindications to the administration of a spinal anaesthetic at the time and no specific indications that a general anaesthetic would have been more appropriate as espoused by Dr Crowhurst.

57. The hypothesis made on behalf of the family was that Dr Moreno used a high spinal block which affected Mary's circulation and in association with hypovolaemia, contributed to her collapse. Dr Crowhurst criticised the use of a high block and the dosage of the anaesthetic agent used by Dr Moreno. He also made reference to the potential for the spinal block to advance with positioning of the patient, and in particular that this could have occurred if Mary had been placed in the Trendelenburg position.[77] The evidence of this was equivocal. The anaesthetic record indicated that Mary was placed in the lithotomy position.[78] The evidence of Mary moving her arms and subjectively denying any upper limb neurological symptoms does not support the proposition that she had been compromised by a "high block". In his evidence, Dr Crowhurst accepted that the dose of the spinal anaesthetic was a common dose used in Victoria. He also accepted that many anaesthetists would aim for a block of T6 rather than the T9-T10 block that he would use.

58. Dr Stainsby gave evidence that he had no concerns in relation to the spinal anaesthetic used and that it was appropriate in the circumstances. Furthermore, Mary's gestational diabetes was of no consequence in relation to the decision regarding the type of anaesthetic to administer or of her anaesthetic management *per se*.

d) The likelihood of Mary suffering an embolic event

59. The evidence of the possibility of an AFE was equivocal. From a pathologist's perspective, Dr Lynch said that such a diagnosis was dependent on the finding of foetal squames. On the other hand, from the clinician's perspective, Professor Cade said that squames are neither a sensitive nor specific test for AFE and that the diagnosis is based on clinical findings/observations.[79] Dr Dalton agreed that AFE was a clinical diagnosis and that the presence of foetal squames in the pulmonary vasculature was a variable phenomenon.

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[77] In the Trendelenburg position the body is laid flat on the back (supine position) with the feet higher than the head by 15-30 degrees.

[78] The lithotomy position involves the positioning of an individual's feet above or at the same level as the hips (often in stirrups), with the perineum positioned at the edge of an examination table.

[79] T @ p 1194

60. According to Dr Lynch, he found no evidence of AFE. He stated that he had performed five maternal death post mortem examinations in 18 years and that it was his expectation that he would find foetal squames in post mortem tissue samples if there had been an AFE.

61. In relation to the possibility of Mary having suffered from an air embolism, Dr Lynch said that if Mary had an air embolism, he would not expect to find evidence of air embolus at the time he performed a post mortem[80] examination on Mary, that is, two days after her death and approximately 63 hours after the catastrophic event in theatre. A post mortem CT scan may have demonstrated air embolism effect on the brain however, VIFM did not have a CT scanner at the time of Mary's death. Dr Lynch said that there is a theoretical risk of air embolism occurring during some kind of procedure performed in the peripartum period such as manipulation of the cervix and/or MRP.[81] If Mary's collapse and clinical signs were in keeping with an air embolism process according to the Anaesthetist or the Intensivist, he would defer to their opinion and acknowledge that they might be right.[82]

62. Despite a number of propositions/hypotheses put to Dr Lynch, he said that his opinion on the cause of death as ascribed in his report[83] had not altered. He did however concede that because the sampling process undertaken during the post mortem is not exhaustive, it was possible that foetal squames may have been present in other areas of the lungs that he had not sampled.[84] But the identification of a probe patent foramen ovale at autopsy added weight to the possibility of Mary suffering from some form of embolic event. Dr Lynch said that amniotic fluid embolism could enter the venous system in the pelvis and end up in the arterial system because of a functionally patent foramen ovale[85] in circumstances:

> *..if her physiology became sufficiently deranged, and appropriately deranged, then it's conceivable that this hole would then have allowed egress of contents of right atrium into the left atrium.*[86]

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[80] T @ p 1002.

[81] T @ p 1003.

[82] T @ p 1003.

[83] Exhibit 28 – Report of Dr Matthew Lynch dated 30 August 2010.

[84] T @ pp 1022-1023.

[85] T @ p 1005.

[86] T @ p 1029.

63. Dr Dalton said AFE is "an extremely rare and enigmatic condition"[87] but the presence of a patent foramen ovale supports an embolic explanation for Mary's collapse.[88]

64. Professor Cade stated that a diagnosis of AFE was not one of exclusion - that is, excluding other possible causes to reach the diagnosis; but one of inclusion and the development of DIC was one of the presenting clinical indices. Mary did develop DIC. Professor Cade was of the opinion that Mary's death was caused by AFE, and that reference to AFE and patent foramen ovale should be included into the descriptive cause of death as ascribed by Dr Lynch.[89]

65. Dr Smith accepted Professor Cade's opinion that AFE was the most likely precipitating cause of Mary's collapse and subsequent severe coagulopathy.[90] Dr Smith also accepted that the timing of the collapse relevant to the MRP was consistent with there being an opportunity for a foreign substance, either air or amniotic fluid, to enter the maternal circulation.[91]

66. Dr Stainsby argued that the most likely cause of Mary's sudden and catastrophic collapse was due to an air embolism, but that given the time between her collapse and her death one would not expect any specific findings relating to air embolism at autopsy. The clinical indices consistent with air emboli included the earlier episode of hypotension, which Mary recovered from with only a small amount of fluid replacement, and looking mildly pale and unwell on the labour ward.[92] In theatre there was the sudden emergence of ST elevation seen on the electrocardiogram and the "dusky" skin colour noted around the same time. Dr Stainsby stated that these observations were not consistent with hypovolaemia. Similarly, the post resuscitation CT scan of the brain was interpreted as being indicative of multiple emboli to the brain and did not have the appearance of global ischaemic insult - that is, what would be expected in systemic hypovolaemic insult where there is deprivation of the blood supply to the brain.

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[87] T @ p 319.

[88] T @ p 367.

[89] T @ p 1195.

[90] Exhibit 31 – Statement of Dr Jacqueline Smith dated 19 November 2009.

[91] T @ pp 1116-1117.

[92] T @ p 1049.

67. According to Professor Cade, the seizure that preceded Mary's arrest was also inconsistent with hypovolaemia.

e) The likelihood of Mary suffering an anaphylactic reaction

68. The proposition that Mary suffered an anaphylactic reaction to some unidentified agent is no more than a mere possibility within a range of possibilities. The passage of time between her collapse and her demise significantly impeded any meaningful exploration of this proposition. I heard no evidence which advanced this proposition.

f) The resuscitation in theatre

69. I make no criticism of the management of Mary's collapse and resuscitation in the operating theatre. The response was prompt and well supported by senior medical personnel from within the hospital. Professor Beischer agreed that the resuscitation was of heroic proportions and although he differed from other experts in respect of his preference for the use of Ergometrine instead of hysterectomy, he did ultimately agree that it was a judgement call for the clinicians who were actually present at the time. He conceded that at the time the clinicians proceeded to hysterectomy, they were in a difficult position in making any decisions given the number of differential diagnoses. Similarly, he was critical of the treating clinicians for not administrating prostaglandins and specifically critical of Dr Dalton for inserting a vaginal pack. I do however prefer the explanations of Dr Dalton and Dr Veal in response to this criticism regarding prostaglandins and of Dr Dalton for his use of a vaginal pack.

FINDINGS

70. Mary had an uneventful pregnancy, which by all account was considered to be a 'normal' pregnancy. She had minimal antenatal contact with medical and allied health professionals and apart for the development of gestational diabetes, her pregnancy was without complication. She experienced spontaneous rupture of her membranes and thereafter labour was augmented with Syntocinin to achieve normal vaginal delivery of a health baby boy. There is nothing abnormal about this scenario. By all accounts there is nothing in this scenario that should have or could have alerted all those involved with Mary, of what was to come.

71. There is no evidence of unexplained blood loss following labour, merely an unsupported hypothesis of unexplained blood loss. The scrutiny of the legitimacy of this hypothesis has

occurred in part because of an absence of a definitive cause of Mary's postpartum collapse, coupled with an absence of a thorough unambiguous contemporaneous record of Mary's clinical course. For example, there is no contemporaneous record or indeed reporting of a BP of 60mmHg systolic, recording of a thready pulse or effective communication to all involved in Mary's care, of the "pumping" of fluids. The possibility of unexplained blood loss and hypovolaemia morphed from one of a limited number of explanations within the differential diagnoses, to *the* cause of Mary's collapse and subsequent death. I do not however accept this explanation. The application of Occam's Razor[93] is appropriate[94] in the circumstances. Unexplained blood loss and/or hypovolaemia have not been proved to a *reasonable satisfaction.*[95]

72. **I find** that the preponderance of evidence excludes blood loss and/or hypovolaemia as the antecedent cause of Mary's collapse, cardiac arrest and subsequent death – all blood loss was accounted for and even when an allowance is made for underestimation, the amount of blood loss could not have itself been the cause of death. In addition, I accept the submissions that Mary's clinical presentation was not one that the doctors said they would expect to and/or had seen in cases of hypovolaemia. I also accept that when assessing the haemodynamic impact of blood loss, it needs to be looked at in the context of what was replaced up to the time of the MRP. I am satisfied that Mary had received sufficient fluid replacement to correct any possible volume imbalance that occurred during and because of childbirth.

73. Similarly, I do not accept that Mary's catastrophic collapse was related to the effects of the spinal anaesthetic administered for the purposes of MRP.

74. **I find** that the administration of a spinal anaesthetic was appropriate in the circumstances. I make no adverse finding against Dr Moreno in her management of Mary.

75. I am persuaded that the probable antecedent cause of Mary's profound collapse and subsequent death was an embolic phenomenon of a nature I am not able to determine.

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[93] Occam's Razor - The English philosopher, William of Occam (1300-1349) propounded Occam's Razor:

"Entities should not be multiplied more than necessary" That is, the fewer assumptions an explanation of a phenomenon depends on, the better it is.

[94] Submission of Ms Hartley – T @ p 1427.

[95] *Briginshaw v Briginshaw* (1938) 60 CLR 336.

Amniotic fluid embolus is a more persuasive explanation than air embolism given the profound coagulopathy seen on the return of a cardiac output, although I acknowledge that Dr Stainsby did say that the arrest and resuscitation of themselves could account for this occurrence. Although it would be preferable to have some clarity as to the exact mechanism of Mary's collapse, neither embolic phenomena can be definitively substantiated or indeed excluded by the clinical or forensic evidence. Nevertheless, having rejected blood loss/hypovolaemia as the precursor and having accepted embolic phenomenon as the precursor, it is not necessary for me to make a specific finding on whether it was AFE or air embolus. **I find** that the precursor to Mary's collapse was an embolic event unrelated to the management of her labour, peri-natal period and decision regarding the need to proceed to manual removal of placenta in the operating theatre under a spinal anaesthetic. The embolic event was not foreseeable and hence not preventable despite the catastrophic outcome. The response to Mary's collapse in theatre was prompt, sustained and involved appropriately qualified medical practitioners who collaboratively made management decisions that were at all times aimed at rectifying Mary's parlous state. There was a lack of clinical indices that might have alerted her healthcare providers to what was to occur or indeed, to what caused Mary's profound collapse.

76. In light of the persuasive evidence of an embolic event likely facilitated by the presence of a patent foramen ovale, I intend to direct the Registrar of Births Deaths and Marriages to amend the cause of death to be: hypoxic brain injury complicating cardiorespiratory arrest during manual removal of placenta (under spinal anaesthetic) following postpartum haemorrhage in the presence of a patent foramen ovale.

COMMENTS

77. The submissions made on behalf of the family include but are not limited to the consequences of Dr Alobaidy's concession that Mary fell within the definition of the Ballarat Hospital *nursing* protocol for postpartum haemorrhage,[96] the significance of Dr Alobaidy's operation report entry made following Mary's theatre attendance of "postpartum haemorrhage"[97] and the significance of a patent foramen ovale in the context of a lack of foetal squames identified on autopsy. These submissions are not, in my opinion, made in

[96] T @ 215-216.

[97] Exhibit 40.

context of the totality of the evidence. Similarly, the issues raised on behalf of the family regarding the alleged failure to provide supplementary oxygen during the spinal anaesthetic and the alleged failure to insert an indwelling urinary catheter prior to Mary's arrival in theatre are matters that I do not believe are causally connected to Mary's collapse and subsequent clinical progression. Accordingly, I have not elaborated on these matters.

IN CONCLUSION

78. In coronial matters where the circumstances surrounding the death are associated with medical intervention and management, the standard of communication and documentation inevitably comes under scrutiny. In many of these matters, the absences of recordings of significant vital signs/observations, the absence of times when vital signs are taken and inaccurate recordings of medications or fluids administered are the catalysts for the need for an Inquest to be held so that the deceased patient's clinical course can be factually understood. To make a recommendation that health care professionals pay better attention to their documentation is trite but I dare to make the comment that they be reminded that their documentation is a means of communication between treating healthcare professionals, is a legal document and should act as an *aid memoir*. Retrospective entries are often necessary as the urgency of events does not allow for contemporaneous note making and I accept this to be the situation for Midwife Adeney and Midwife Pullin, who were focused on attending to Mary and transferring her to theatre. Retrospective notes should however clearly reflect that they are retrospective by indicating the date and time they are made, otherwise they give the impression that the information has been conveyed sequentially, which was not the situation in Mary's case.

79. Save for these concluding comments, I make no adverse finding against individual healthcare providers involved in the care of Mary during or following the delivery of Tommy and make no adverse findings in relation to the management of her collapse and resuscitation. The appropriateness of the decisions of the clinicians made before her collapse, at the time of her collapse and during sustained resuscitation attempts must be assessed in the context of the clinical presentation at the time and I am satisfied on the evidence that they were appropriate. Retrospective scrutiny has not diminished the appropriateness of the medical management of Mary. There is no one aberrant indicium of Mary's catastrophic clinical course that I have identified in the evidence that could have or should have been acted upon in some different way that, on the balance of probabilities, I

can find that the outcome could have been altered or would have been different. It was neither foreseen nor foreseeable. It thus follows, that I cannot find that Mary's death was preventable.

80. Mary's death was and remains a real tragedy.

Improvements To The Delivery Of Health Services

81. I am satisfied that Ballarat Health Services undertook a systems review after Mary's death and in response to identified shortcomings have instituted a number of changes including but not limited to:

- the institution of postpartum haemorrhage flow-charts which are placed in each birthing room
- changes to the clinical practice guidelines influenced by an audit of postpartum haemorrhage
- implementation of blood loss estimation training
- the development and implementation of a postpartum haemorrhage trolley in the maternity ward.

82. With the implementation of blood loss estimation training, I am satisfied that this measure obviates the need for a recommendation that the weighing of kylies/pinkies be mandated.

RECOMMENDATIONS

83. I acknowledge the divergence of views between the pathologists and the clinicians about the evidence required to diagnose AFE, nevertheless, it was clear on the evidence that it is considered one of the leading causes of maternal death[98] of which not enough is understood. In an attempt to capture the incidents of AFE, including both where death and survival has occurred, with the aim of improving the knowledge base and potentially the management of women diagnosed with AFE, I accept Professor Cade's comments[99] and **I recommend** that The Royal Australian and New Zealand College of Obstetricians and Gynaecologists

[98] See Exhibit 32 and Exhibit 33.

[99] T @ 1189-1190.

explore the utility of sharing this knowledge through the development, administration and maintenance of an Amniotic Fluid Embolism register.

84. **I direct** the principal registrar to notify the Registrar of Births Deaths and Marriages of the amendment to the cause of death as is reflected above.

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

- Mr Peter Korzeniewski on behalf of the family
- Mr Paul Henderson, Slater & Gordon
- Ms Elaine Fabris, DLA Piper
- Mr Rob Muir, John W Ball & Sons
- Ms Hannah Jankiewicz, Middletons
- Director of Medical Services, Ballarat Health Services
- Consultative Council on Obstetric and Paediatric Mortality and Morbidity (CCOPMM)
- The Royal Australian and New Zealand College of Obstetricians and Gynaecologists

86. I intend to direct that the Finding be published in the absence of any objection to the same.

Signature:

AUDREY JAMIESON
CORONER
Date: **17 September 2013**