IN THE CORONERS COURT
OF VICTORIA
AT MELBOURNE

Court Reference: 2010 1279

FINDING INTO DEATH WITH INQUEST

Form 37 Rule 60(1)

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

Inquest into the Death of:

Deceased:	SUMMER JAMSEK
Findings of:	HIS HONOUR, PHILLIP BYRNE
Delivered On:	Tuesday, 21 June 2016
Delivered At:	Coroners Court of Victoria 65 Kavanagh St, Southbank
Hearing Dates:	23 November – 2 December 2015
Representation:	Mr A. Mukherjee, for Ms Jamsek Ms F. Ellis, for Dr Manikkam Ms S. Keeling, for Dr O'Sullivan Mr P. Halley, for Frances Perry House Mr R. Muir, for Ms K Russell
Police Coronial Support Unit, Assisting the Coroner:	LSC Tracey Ramsey SC Kelly Ramsey

I, PHILLIP BYRNE, Coroner, having investigated the death of SUMMER EMERALD ROSE JAMSEK

AND having held an inquest in relation to this death on 23 November – 2 December 2015
at MELBOURNE
find that the identity of the deceased was SUMMER EMERALD ROSE JAMSEK
born on 4 April 2010 at Frances Perry House, Parkville
and the death occurred on 5 April 2010
at Royal Children's Hospital, Parkville, Victoria
from:

1 (a) PERSISTENT PULMONARY HYPERTENSION IN THE NEWBORN (PPHN)

in the following circumstances:

BROAD CIRCUMSTANCES

1. Baby Summer Jamsek was born at Frances Perry House just after 4pm on 4th April 2010. She was delivered by obstetrician Dr Huon O'Sullivan. Ms Jamsek and Baby Summer were cared for after delivery by midwife Stephanie Tsesmetzis.

2. Dr O'Sullivan transferred care of Baby Summer to paediatrician, Dr Noel Manikkam, who assumed the care of Baby Summer shortly prior to 5pm on 4th April 2010. Baby Summer was monitored, initially, and subsequently admitted to the Special Care Nursery (SCN) at Frances Perry, where she was cared for by the midwife in charge, Ms Robyn Henshall.

3. At about 7:20pm, Dr Manikkam undertook a full neonatal examination of Baby Summer. When concerns about the baby's condition were conveyed to him, Dr Manikkam attended Frances Perry and undertook a further examination/assessment of Baby Summer. He was concerned that Baby Summer's condition may have been due to cardiac issues and requested NETS to attend, with a view to transferring Baby Summer to the Royal Children's Hospital.

4. NETS attended shortly prior to 11pm on 4th April and took over care of Baby Summer. They proposed to stabilise her and convey her to the Royal Children's Hospital. At about 11:45pm, Baby Summer experienced a sudden collapse in respect of which the NETS team experienced great difficulty in resuscitating her, despite aggressive therapy.

5. Subsequently, Baby Summer was conveyed to Royal Children's Hospital where her condition was grave; as it turned out irretrievable. Baby Summer died shortly after 8am on 5th April 2010, after life support was withdrawn.

CORE FINDINGS REQUIRED TO BE MADE

6. Section 67 of the Coroners Act 2008 provides the core findings I am required to make. In this case, the controversial findings I am required to make are:

   - The cause of death
   - The circumstances in which death occurred

7. In his written submission, Mr Mukherjee, counsel for Ms Sonja Jamsek, Baby Summer's mother, stated the following issues required resolution:

   - What was the cause of Baby Summer's death?
   - When did the cause of Baby Summer's death begin and/or manifest itself?
   - Was the cause of Baby Summer's death capable of detection contemporaneously?
   - Whether the cause of Baby Summer's death ought to have been detected and treated earlier?
   - Would earlier detection have led to Baby Summer surviving?

   I find that analysis helpful and have sought to answer those questions.

RELEVANT LAW

8. I believe it is important at the outset to include in my finding aspects of the law which impact upon my responsibilities under the *Coroners Act 2008*. Very often parties leave the Court with an unfulfilled expectation. There is often an expectation that the coroner will lay or apportion blame, or level strident criticism of the party they see as 'at fault'. Often such parties allege negligence. It is not my role to determine those matters.

9. "*Keown v Khan*",[1] a decision of the Victorian Court of Appeal, represents a landmark judgment which, in my opinion, provided much needed guidance to Victorian (and other)

[1] (1999) VR 69

coroners. His Honour Mr Justice Callaway, adopting a statement contained in the Brodrick Committee (UK) Report,[2] said:

> *"In future the function of an inquest should be simply to seek out and record as many of the facts concerning the death as public interest required, without deducing from those facts any determination or blame."*[3]

Again quoting the Brodrick Committee (UK) Report, His Honour noted:

> *"In many cases, perhaps the majority, the facts themselves will demonstrate quite clearly whether anyone bears any responsibility for the death; there is a difference between a form of proceeding which affords to others the opportunity to judge an issue and one which appears to judge the issue itself."*[4]

So, while not laying or appropriating blame, a Coroner should endeavour to establish the CAUSE, or CAUSES, of a death; the distinction is fine, but real. As Callaway J.A. described it in *Keown v Khan*:

> *"In determining whether an act or omission is a cause or merely one of the background circumstances, that is to say a non-causal condition, it will sometimes be necessary to consider whether the act departed from a norm or standard or the omission was in breach of a recognised duty, but that is the only sense in which para. (e) mandates an inquiry into culpability. Adopting the principal recommendation of the Norris Report, Parliament expressly prohibited any statement that a person is or may be guilty of an offence. The reasons for that prohibition apply, with even greater force, to a finding of moral responsibility or some other form of blame".*[5]

10. I have found articulating the dichotomy between finding the cause of death on one hand and finding or apportioning fault, blame or culpability on the other, difficult to articulate. Quite recently, in a judgement of New Zealand Court of Appeal, I saw as good an explanation of the conundrum as I have seen. In the *Coroners Court v Susan Newton & Fairfax New Zealand Ltd* reference is made to *Laws NZ*, Coroners. At paragraph 28 under the heading of "blame", the following statement appears:

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[2] Report of the Committee on Death Certification And Coroners (1971) (UK) ("The Brodrick Report" Cmnd. 4810)

[3] (1999) 1 VR 69, 75

[4] (1999) 1VR 69, 75

[5] Ibid 69, 76

> *"It is no part of the coroner's function to apportion blame for the death. The coroner must however be able to go beyond the mere cause of death if the coroner is to serve a useful social function, and must establish so far as is possible, the circumstances of the death. The implicit attribution of blame may be unavoidable in order for the coroner to ascertain or explain how the death occurred in the wider events that were the real cause." (my emphasis)*[6]

If an adverse finding is made it should be couched in relatively subtle terms, even though the implied attribution of blame/fault is lost on the lay person (who expected direct denouncement of the party they see as responsible for the death under investigation). Once the facts are elucidated, the parties (and others) can make of them what they will.

11. CAUSATION generally goes to the heart of a matter. It has, over the years, been the subject of considerable judicial attention. In *E and MH March v Stramare*,[7] Chief Justice Mason observed:

> *"What was the cause of a particular occurrence is a question of fact which must be determined by applying common sense to the facts of each particular case."*[8]

In *Chief Commissioner of Police v Hallenstein*,[9] Justice Hedigan said that the fundamentals of causation in the context of negligence were applicable to the concept of causation or contribution in the context of coronial matters.

12. STANDARD OF PROOF

In *Briginshaw v Briginshaw*,[10] Dixon J, as he then was, provided a classic statement as to the appropriate standard of proof to be used in civil cases:

> *"...reasonable satisfaction is not a state of mind that is attained or established independently of the nature and consequence of the fact or facts to be proved. 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*

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[6] [2006] NZAR 312

[7] (1991) 171 CLR 506

[8] (1991) 171 CLR 506 para 17

[9] (1996) 2VR1

[10] (1938) 60 CLR 336 @ 362-3

issue has 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".

The issue of the standard of proof to be brought to bear in coronial matters has been the subject of discussion in a number of authorities.[11]

13. In my view, for an act or omission to be a causal factor in a death, the connection must be logical, proximate and readily understandable; not illogical, strained or artificial.

14. When the concept is transported to coronial findings, where the questions for determination relate to entities in their professional capacity, I have to reach a comfortable degree of satisfaction to make an adverse finding.

LEXAPRO

15. I digress to address this issue, which I do not propose to pursue at length, but as the issue has a public interest imperative, and as the matter was referred to in an earlier media report of these proceedings, I think it warrants some attention.

16. During the pregnancy Ms Jamsek was using Lexapro, a selective serotonin reuptake inhibitor (SSRI) antidepressant. Some studies have concluded that there is a possible link between a mother taking SSRI during pregnancy and the risk that her baby may suffer from Persistent Pulmonary Hypertension in the Newborn (PPHN). I find it interesting that even highly qualified medical practitioners; obstetricians, paediatricians and neonatologists were, prior to being involved in this case, either unclear or even unaware of the possible link between PPHN and SSRI's.

17. Initially, several doctors, including Dr Manikkam, considered that Lexapro may have been a causal factor in Baby Summer's death.

18. The issue was a matter of very considerable concern to Ms Jamsek. One can understand her dismay, as apparently it led her to the view that in continuing to take the prescribed medication during her pregnancy she was somehow responsible for a condition that may have resulted in the death of her child.

[11] Anderson v Blashki (1993) 2VR89 Health and Community Services v Gunvich (1995) 2 VR69

19. Although the issue of the possible link was canvassed at some length during the hearing, as a result of evidence led by Ms Keeling later in the hearing in respect of a relatively recent communique by the United States Food and Drug Administration (FDA), the issue became less contentious.

20. As a result of this evidence, I expressed a tentative view that it was highly unlikely I would make a finding that Ms Jamsek's use of Lexapro during pregnancy was a contributing factor in Baby Summer's death.

21. The conclusions reached by FDA in this regard are encapsulated in the following statement from the communique:

> *"At present FDA does not find sufficient evidence to conclude that SSRI use in pregnancy causes PPHN, and therefore recommends that health care providers treat depression during pregnancy as clinically appropriate."*[12]

22. While Drs Costello, Manikkam and Erickson accepted the position as stated by FDA, Dr Butt maintained that whilst there was "*no clear evidence*" of the connection between SSRI use and PPHN at the present time, there had been previous instances where FDA have subsequently withdrawn drugs that had previously been approved. Dr Butt stated:

> *"So what this document says to me is there's conflicting studies, not enough to withdraw the drug. That's all it means. It doesn't mean there's no association."* [13]

I understand Dr Butt's contention.

23. In his submission, Mr Mukherjee accepted that there is insufficient evidence before me to warrant a finding that Ms Jamsek's use of Lexapro during her pregnancy was a contributing causal factor in Baby Summer's death.

24. While I cannot absolutely discount the possibility Ms Jamsek's use of Lexapro during pregnancy impacted upon Baby Summer, that prospect is, in my view, highly unlikely. On balance I am comfortably satisfied it did not.

"CLICKING NOISES"

25. I am unable to determine what was the cause of the sound described as a "clicking noise", made by Baby Summer shortly after birth. I accept that she made some sound, but it was not heard by Dr O'Sullivan and whether it was heard or brought to the attention of midwife

[12] (Exhibit V)

[13] (T999)

Tsesmetzis, I do not know as she maintains she had no independent recollection of events. There is no evidence that, whatever the sound was, it was indicative of any particular condition and, in any event, it was transient. I can take this matter no further.

ASSESSMENT OF MANAGEMENT

26. The process I propose to follow is to examine, in chronological order of input, the course of management of Baby Summer by those primarily involved, with a view to determining whether there was, in each case, some deficiency in management/care and then to consider whether, if satisfied there was deficiency, it represented a causal or contributing factor in Baby Summer's death.

In considering whether there was some deficiency in medical or nursing management, which could be considered a causal factor in Baby Summer's death, I reiterate the principle enunciated by Callaway JA in '*Keown v Kahn*' is applicable. His Honour said:

> *"In determining whether an act or omission is a cause or merely one of the background circumstances, that is to say a non-causal condition, it will sometimes be necessary to consider whether the act departed from a norm or standard or the omission was in breach of a recognised duty."*[14]

DR HUAN O'SULLIVAN

27. Dr O'Sullivan was Ms Jamsek's obstetrician. He was well experienced, having been a Gynaecologist and Obstetrician for some 20 years. Having already disposed of what I will call the "Lexapro issue", I do not propose to deal with the antenatal aspects of Dr O'Sullivan's management of Ms Jamsek, but focus on the post natal period, which, as far as Dr O'Sullivan is concerned, is a period of approximately one hour.

At just after 3:45pm on 4th April 2010, Dr O'Sullivan attended Ms Jamsek in the birthing room. He previously reviewed Ms Jamsek at 8am, 11:45am and 1:30pm. Baby Summer was delivered by ventouse-assisted vaginal delivery at 4:01pm. The birth, at least from the perspective of the obstetrician, was an easy delivery only requiring two "pulls". Within a period of 30 seconds to one minute it was assessed that Baby Summer required oxygen to "*ensure a good transition to extra uterine life*".[15] She was taken to the nearby resuscitaire

[14] (1999), 1VR 69@76

[15] (T245.15)

where oxygen was administered by bag & mask. This process was undertaken for approximately 15-30 seconds, after which Dr O'Sullivan described Baby Summer as having a "*good change in colour*", and becoming pink with cyanosis of the lips not present. Dr O'Sullivan said that he was satisfied with Baby Summer's condition.

28. At 4.20pm, at the nurse's station, Dr O'Sullivan prepared his handwritten notes of the delivery, an exercise he estimated took about 10 minutes. From there, Dr O'Sullivan said he made a routine a referral to paediatrician Dr Noel Manikkam. Dr O'Sullivan stated it is his routine to refer all babies for paediatric check. He said that in this case he also bore in mind the fact that Ms Jamsek had experienced a protracted rupture of the membrane, which had the potential to lead to infection and, in those circumstances, made the referral at that time rather than leave it to the next day.

In viva voce evidence, Dr O'Sullivan related what he believes he conveyed to Dr Manikkam. For completeness, I include in this finding the following excerpt:

> *"I would have told Dr Manikkam about Sonja's history, her gestation, her presentation the day prior, on 3 April, with ruptured membranes, not in labour, the fact she was Group B streptococcus negative, the fact she'd had syntocinon commence that morning at 8 o'clock or shortly thereafter, Amoxil had been given in two doses as a broad spectrum antibiotic to cover for infection. The CTG, being slightly tachycardic towards the end of labour, the low grade fever, the straightforward ventouse, Summer's condition at birth with her Apgar scores, the fact she'd had some bag and mask oxygen momentarily, and that I was now satisfied that Baby Summer was in a satisfactory condition so that the call was to alert Dr Mannikam to her being here and to seek advice if there was any further advice that he might have particularly related to the possibility of infection although there was no sign of that at the time."*[16]

I note that, as part of the information conveyed, there was reference to the intravenous administration of Amoxil on two occasions earlier in the day.

Dr O'Sullivan stated he did not request, or necessarily expect, Dr Manikkam to review Baby Summer that day, saying he thought it would be okay for the review to take place the

[16] (T250.19)

following morning. He maintained he saw no urgency as he considered Baby Summer to be in a "*stable and good condition*".[17]

29. There has been contention about the APGAR scores allocated to Baby Summer shortly after delivery. Dr O'Sullivan maintains he wrote the three scores, which he says were settled/assessed in consultation with midwife Stephanie Tzemetsis. The APGAR scores allocated were 5 at 1 minute (whether that was before, during or after the bagging is unclear), 7 at 5 minutes and 8 at 10 minutes. There is contention on two bases:

    - Whether the scores allocated were appropriate to the condition of Baby Summer at the various times (i.e. were too high).
    - Whether the scores allocated claimed to be in the range of normal, are in fact outside that range.

    I address the first of these contentions. Dr O'Sullivan and Ms Tzemetsis, an experienced obstetrician, and a qualified, but inexperienced, midwife made their respective assessments on specific criteria when observing and handling Baby Summer. Others who are critical of the APGAR scores allocated either did not at the relevant time observe and assess Baby Summer, or in relation to those present, did not have the necessary qualifications, or indeed experience, to make a compelling judgement. I accept the APGAR scores allocated as reasonably reflective of Baby Summer's condition during that short period.

29. Those challenging the efficacy of the allocated APGAR scores, and the subsequent condition of Baby Summer, have placed significant reliance on what is depicted in photographs and videos taken of Ms Jamsek and Baby Summer in the period shortly after delivery. Obviously, that is not the optimal method of making an assessment of her condition. That material was provided to Dr Bernadette White, the expert engaged by the Court. Dr White advised, having viewed that material, that the additional material did not alter her previous opinion as to Dr O'Sullivan's management of Baby Summer. Dr White and others queried the quality of the photographs and videos to found a valid assessment of the baby's condition. Dr White did, however, say that she noted Baby Summer showed no signs of respiratory distress in the 4th video.

30. Dr Erickson was somewhat critical of the APGAR scores given, he described them as "*low and concerning*". In coming to that conclusion, Dr Erickson has apparently relied upon the

[17] (T251.28)

photos provided to him by Ms Jamsek. He concluded Baby Summer "*appeared to be quite cyanosed*". However, in viva voce evidence Dr Erickson suggested the APGAR scores were "*borderline low*" and conceded he had never before assessed cyanosis by way of photos and video.[18] In answer to a question from Ms Keeling, Dr Erickson conceded that Dr O'Sullivan, in the room with Ms Jamsek and Baby Summer, would be in a better position to judge whether Baby Summer was centrally cyanosed, if Dr O'Sullivan's judgement as to cyanosis was correct. Bearing in mind Dr O'Sullivan's experience, he claimed he had delivered, or been associated with the delivery of, approximately 8000 deliveries. I am satisfied Dr O'Sullivan would be in a position to make a valid judgement on APGAR scores.

As to whether the APGAR scores allocated were sufficiently outside what could be considered 'normal', the weight of evidence would suggest they were not at sufficiently low levels to raise concerns about Baby Summer's condition. However, when looked at collectively, they were at the lower end of normal.

31. Although it is not a determinative issue, I think it is appropriate to make some comment about Dr O'Sullivan providing obstetric care to Ms Jamsek in relation to the births of her two subsequent children, Sienna in June 2011 and Spencer in November 2013.

On behalf of Dr O'Sullivan, it was put that in attending Dr O'Sullivan for the births of Sienna and Spencer, Ms Jamsek was satisfied that Dr O'Sullivan provided good obstetric care to her and her babies.

In evidence, Ms Jamsek rejected that suggestion indicating she was too busy having a "*breakdown*" to shop around and believed Dr O'Sullivan would likely "*take extra care*" because of what occurred in Baby Summer's case.

Ms Jamsek also stated that Dr Butt had advised her that Frances Perry "*was the best place to be*". Ms Keeling put to Ms Jamsek that one could still go to Frances Perry, but consult a different obstetrician. Ms Jamsek conceded she did not try to get another obstetrician.

I conclude that, at the time of the births of Sienna and Spencer, Ms Jamsek did not consider the care provided by Dr O'Sullivan, in relation to Baby Summer's birth, to be deficient. I further conclude that Ms Jamsek's dissatisfaction with Dr O'Sullivan's obstetric management of Baby Summer's birth developed over time, the longer she pondered the tragic circumstances of the demise of her first child.

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[18] (T924)

32. Dr Erickson engaged by Ms Jamsek to provide an opinion, opined there were "some deficiencies in the initial management". It is unclear to me whether he is referring to both when Summer was under the care of Dr O'Sullivan or Dr Manikkam, or both. In the event he, quite reasonably, provided the following qualification to his opinions; he wrote,

> *"In providing this summary I must note that it is extremely difficult to be certain of events that occurred in retrospect and from limited information. It is also worth noting that some management decisions which appear clear in retrospect may extremely complicated and difficult at the time. It is also clear from the notes provided to me that medical staff involved in Summer's care had Summer's interest at the forefront of their actions."*[19]

In short, applying the appropriate standard of proof, I see no reasonable basis to be critical of the medical management of Baby Summer by Dr Huan O'Sullivan.

MIDWIFE STEPHANIE TSESMETZIS

33. Ms Stephanie Tsesmetzis was the midwife allocated to Ms Jamsek in the birthing suite. Ms Tsesmetzis claims she has no independent recollection of the events relating to the birth and subsequent care of Baby Summer. Consequently, the information contained in her statement is based upon a review of the relevant medical records.

34. While I am satisfied Ms Tsesmetzis was appropriately qualified and trained, she was inexperienced; the 4th April 2010 was only her fourth unsupervised day in the birthing suite. Although I note Midwife Teponga, in her statement, said she viewed Ms Tsesmetzis as a "*very competent midwife notwithstanding her relatively junior status*". The reality is that, like many occupations/callings, expertise is accumulated by practical experience.

35. It was submitted by Mr Mukherjee, counsel for Ms Jamsek, that midwife Tsesmetzis' claim of having no recollection of events is "*implausible*". I asked Ms Tsesmetzis when she became aware that Baby Summer had died only hours after leaving her care. Ms Tsesmetzis said she believed she was told the following day. I asked that question of Ms Tsesmetzis because I would have thought that such an event, in her first week as an unsupervised midwife, would be indelibly etched into her mind so that the recollection of events would be vivid and would not diminish to the extent one would have no recollection, even some years

[19] Brief p62 Exhibit Y

after. Ms Tsesmetzis maintained her position of having no independent recollection due to the unexpected death of her father and the effluxion of time.

36. I must say I have significant reservations about Ms Tsesmetzis's claim/explanation, but I do not believe I can dismiss it completely, due to the vagaries surrounding the phenomenon of repression of the memory. However, I have concluded that Ms Tsesmetzis' inability to provide precise detail of what she observed is not critical, mainly due to the fact Baby Summer was seen by other more experienced midwifes during the relevant time. Furthermore, Baby Summer was reviewed by paediatrician Dr Noel Manikkam who, being advised Baby Summer "*doesn't look right*", requested that she be transferred to the Special Care Nursery for observation.

37. Ms Katrina Russell, another midwife on duty in the birthing suite (with two patients of her own) entered the room where midwife Tsesmetzis was caring for Ms Jamsek and Baby Summer to check a Hartmann's fluid flask; hospital protocol required two nurses check the flask. Ms Russell says she was in the room for only 3-5 minutes, during which time she observed Baby Summer. I have carefully examined Ms Russell's evidence. Conceding she was not a paediatrician, Ms Russell concluded Baby Summer "*wasn't responding like a normal baby necessarily*". She concluded Baby Summer was a "*tiny bit off*", "*nothing concrete*" but "*better to be safe than sorry*",[20] so suggested Ms Tsesmetzis do oxygen saturations and, if not within normal ranges, advise Dr Manikkam. Ms Russell considered Baby Summer "*quite pale*", "*floppy*", "*slightly lacking tone*", looked vacant, "*didn't seem to have much going on behind her eyes*". In short, Ms Russell had some concerns about Baby Summer's condition, at least to the extent that further investigation was warranted.

However, in spite of those concerns, Ms Russell went on to say she was "*very surprised*" Baby Summer was "*shipped out*" to the Special Care Nursery and stated that if she considered Baby Summer was "*compromised*" she would be the first to "*press the buzzer*" for escalation of management.[21] Ms Russell denied the claim made by family members that she said to family members "*this baby is sick- get her to the Special Nursery*". I include an excerpt from the viva voce evidence in which Ms Russell maintains she said,

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[20] (T502)

[21] (T515)

"We're going to do an 02 sat"- I– "I've suggested that we do an oxygen saturation then call the paediatrician if needed and then, just so you're aware, she may or may not need to go to the Special Care Nursery".[22]

I accept the message she conveyed was along those lines; not an indication that she thought Baby Summer should be immediately transferred to the Special Care Nursery. I believe the family misconstrued what Ms Russell said to them.

MIDWIFE HENSHALL

38. Ms Robyn Henshall, the midwife in charge of the Special Care Nursery at the relevant time, had direct care of Baby Summer from when she first went to the Special Care Nursery. Ms Henshall is a vastly experienced midwife, working in Special Care Nurseries for over 25 years at the time.

39. In evidence, Ms Henshall corrected the time that Baby Summer came into her care; she said it was shortly prior to 7:30pm, not 8:30pm as she had stated in her statement. In any event, Ms Henshall said she initially undertook a set of observations of Baby Summer after attaching a cardiorespiratory monitor and an oxygen saturation probe. While Ms Henshall noted "*mild retraction*" she did not think Baby Summer was critically unwell. In fact in response to a question from Mr Halley, Ms Henshall maintained that at 7:30pm observations indicated Baby Summer was a "*healthy baby*". At the time, Baby Summer was only in Special Care Nursery for observation, she had not been formally admitted. It was planned that Baby Summer would be returned to the room with Ms Jamsek if stable after the next feed.

40. However, at 8:30pm, after being advised by Ms Henshall that Baby Summer was experiencing transient desaturations down to 84% (then increasing to 92%), Dr Manikkam decided Baby Summer would be admitted to the Special Care Nursery and not returned to Ms Jamsek.

41. At 9:30pm, Ms Henshall noted Baby Summer's mild rib retractions persisted, she was pink in colour, but remained quiet. However, shortly after finishing her 9:30pm feed, Baby Summer's oxygen requirements "*increased rapidly*" from 25% then to 40%. This obviously concerned Ms Henshall, who immediately notified Dr Manikkam of Summer's worrying

[22] Ibid

oxygen desaturations. Dr Manikkam asked for an urgent chest x-ray, ordered no oral fluids be provided and to prepare for an intravenous infusion and ordered antibiotics.

42. Dr Manikkam attended within 20 minutes and, as Ms Henshall commented in her statement:

> *"...whilst Dr Manikkam and I were looking at Summer, her legs and lower torso started to turn blue in colour. This happened before our eyes. It was very unusual and not something I have seen before, nor since this event."*[23]

Ms Henshall noted in the Progress Notes that at 10pm Baby Summer "*suddenly pinked up*", with oxygen saturation back to 100%. Ms Henshall maintains there was no delay in responding to Baby Summer's "*unusual pattern of cyanosis*". Again, this was evidence of Baby Summer's fluctuating condition. Ms Henshall confirmed that antibiotics were administered intravenously at 11pm.

43. Ms Henshall said that, having pondered in detail the events after they occurred, she considered the treatment/management of Baby Summer's symptoms "*at all times appropriate*". She further stated that her professional relationship with Dr Manikkam was such that, if she considered the treatment decisions were not appropriate, she would have raised her concerns with him. Ms Henshall advised that although her shift was to end at 10pm, she stayed on until 10:30pm when she handed over nursing care of Baby Summer to the oncoming night shift. Obviously, she was not involved in the care of Baby Summer thereafter.

44. I see no basis for any criticism of Ms Henshall, or other nursing staff on duty in the Special Care Nursery while Baby Summer was in their care; what was needed to be conveyed to Dr Manikkam was conveyed in a timely manner and what he ordered be done was done.

DR NOEL MANIKKAM

45. Dr Manikkam is the paediatrician who took over care of Baby Summer from obstetrician Dr O'Sullivan shortly before 5pm on 4th April, until approximately 11pm when her care was taken over by the NETS team he had summoned.

46. Dr Manikkam provided an initial statement (Exhibit "T") followed by a comprehensive supplementary statement (Exhibit "U") with annexures A-G.

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[23] Brief p59 (exhibit 2)

47. Dr Manikkam's initial input at about 5pm was to advise nursing staff to measure Baby Summer's blood sugar levels and advise him if they fell below 2.5mmol/L. In oral evidence, Dr Manikkam said he asked for BSL to be monitored because he was aware Ms Jamsek had experienced some prolongation of ruptured membranes, together with the fact that Baby Summer required oxygen very shortly after delivery. Shortly before 6pm, Dr Manikkam was advised, presumably by midwife Tsesmetzis, that Baby Summer's BSL was 1.5 mmol/L. Dr Manikkam asked the midwife to provide Baby Summer with 20-25ml of formula, further monitor blood sugar levels and to contact him if BSL didn't pick up.

48. At about 7:20pm, Dr Manikkam received a call from midwife Teponga advising that the "*baby didn't look right*" and the BSL remained low. Dr Manikkam attended virtually right away and observed Baby Summer at the resuscitaire being given oxygen by mask. Dr Manikkam was advised that midwife Teponga had previously observed Baby Summer to have blue legs, midwife Tsesmezis had previously observed some grunting and some external retractions and midwife Russell had concluded Baby Summer was "*vacant behind the eyes*".

    I was somewhat perplexed by one aspect of the evidence of Ms Teponga, an experienced midwife. She maintained that when she first observed Baby Summer's colour to be "*different*" to anything she had previously seen. The evidence of Drs Erickson and Butt was to the effect that what midwife Teponga saw may well have been peripheral cyanosis, likely to be acrocyanosis, which in any event, was quickly reversed by the provision of oxygen.

49. Dr Manikkam stated he undertook a formal neonatal examination of Baby Summer, concluding she was well, with good colour, with oxygen saturations of 100% in air, without signs of respiratory distress, BSL at 3.7mmol/L, but behaving like a 36-37 week baby (in terms of posture and muscle tone). This examination was undertaken in the presence of Baby Summer's father, Paul Chadwick. Dr Manikkam asked that Baby Summer be transferred to the Special Care Nursery for monitoring, not formal admission. He indicated that if Baby Summer fed well and remained stable, she could be returned to Ms Jamsek. Dr Manikkam said he then attended upon Ms Jamsek and "*reassured her that baby was well*".

50. In relation to the concerning signs observed by the midwives, Dr Manikkam stated:

"that's obviously all reversed because when I saw the baby, baby didn't have any of those concerns".[24]

He further concluded the improvement, which I accept occurred, may well have been due to the oxygenation provided immediately before and at the time he attended.

51. At 9:30pm, Dr Manikkam was advised that Baby Summer was now on 25% oxygen, indicating mild respiratory distress. Dr Manikkam ordered an urgent chest x-ray and to prepare for intravenous infusions. At about 9:50pm, when he attended and saw Baby Summer, Dr Manikkam said he suspected Baby Summer may have hyaline membrane disease, with "*unusual pattern of cyanosis*", well demarcated with the upper part of the body pink, but blue from the umbilicus down. He became somewhat concerned that what he observed could be an unstable cardiovascular system issue. He considered the prospect of coarctation of the aorta patent ductus arteriosus and possibly pulmonary hypertension. Dr Manikkam inserted an intravenous infusion of 10% dextrose. He claims Baby Summer's condition suddenly improved, with femoral pulses returning and the unusual pattern of cyanosis disappearing. However, shortly after 10pm, Dr Manikkam contacted NETS asking for transfer of Baby Summer to a tertiary hospital in the event that cardiac surgery would be necessary. The NETS team arrived at Frances Perry at 10:46pm; after formalities, care of Baby Summer was handed over to them.

52. In this case, I think Dr Manikkam could be seen as the 'main player'; the person against who most criticism, both direct and by implication, has been levelled. The contention, in broad terms, being that Baby Summer died from secondary persistent pulmonary hypertension due to sepsis which, it is claimed, manifested itself by 7pm, so that antibiotic therapy (administered at 11pm) should have been administered earlier, at least by 8:30pm if not sooner. It is also contended that Dr Manikkam made "*incorrect lines of diagnosis*" (p.3. Jamsek submission) in initially considering hyaline membrane disease as the reason for Baby Summer's fluctuating, transient condition, and after 10pm believing her worsening condition was cardiac related.

53. From the outset, Ms Jamsek has levelled strident criticism of Dr Manikkam's medical management of Baby Summer. Fundamentally, the contention is that Baby Summer's condition was parlous from the outset, she died due to infection which was not recognised when it manifest itself and was not treated in a timely fashion by Dr Manikkam. While it

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[24] T645

may not have been said directly, the clear implication is that Baby Summer should have been transferred to Royal Children's Hospital earlier.

54. Dr Manikkam refutes the contention that his medical management of Baby Summer was deficient. Dr Manikkam provided a comprehensive supplementary statement (Exhibit "U") in which he elaborates upon his management of Baby Summer and critiques the bases upon which Drs Butt and Erickson take issue with his management of the baby.

55. My principal focus is on seeking to determine the cause of Baby Summer's death, and then to consider whether there was some deficiency/omission in the medical management by Dr Manikkam of Baby Summer that caused or contributed to her death. This exercise has be undertaken without the benefit of retrospection.

In seeking to determine that important issue, I have carefully examined the statements and oral evidence of those with the necessary experience, expertise and qualifications to voice opinions. Dr Manikkam himself, who, unlike the others, was able to actually observe the clinical picture of Baby Summer as events unfolded, Drs Dodd, Erickson, Butt and Costello, the experts who gave evidence, noting that Dr Costello was the expert engaged by the Court. I will deal with the efficiency of Dr Manikkam's management of Baby Summer in the conclusion to my findings.

NEONATAL EMERGENCY TRANSPORT SERVICE (NETS)

56. I digress to address the input of NETS, primarily due to the fact that there is, in my view, little contention in relation to their performance. I was also interested to ascertain whether there had been, in the intervening years, any enhancement/refinement of the service. After some consultation with counsel, I took the decision not to call Dr Manly, the NETS doctor who actually attended Frances Perry and was directly involved in Baby Summer's clinical management prior to her admission to the Royal Children's Hospital shortly after 1am on 5th April. I thought it appropriate to invite Dr Michael Stewart, Medical Director of NETS, who was able to attend at short notice.

57. During Mr Mukherjee's examination of Dr Stewart, I sensed that there was some veiled criticism of the organisation, having regarded the time it took to attend and perhaps whether transfer should have occurred earlier than it did. Prior to that, I was not aware of any concerns regarding the performance of NETS, nor was it an issue that I had indicated at Mentions/Directions was within the scope/parameters of my investigation. In the event,

Mr Mukherjee allayed my concern by indicating it was not his intention to level criticism of the organisation, but merely to seek to determine whether in the intervening period there had been improvements in the response times.

58. Dr Stewart was able to indicate that there were different levels of emergency transfer; "critical" and "urgent", observing that his transfer service was not the equivalent of an emergency ambulance. He advised that, primarily due to improved funding, the service has been significantly enhanced with drivers previously offsite now onsite and increased numbers of doctors available. Dr Stewart advised that, previously, the usual time lapse between receiving the call and despatch was about 45 minutes, whereas presently it is in the order of 10-15 minutes. In relation to the NETS attendance upon Baby Summer, Mr Mukherjee asked Dr Stewart:

> *"Does it follow from your answers, from your earlier answers, to His Honour that if a similar situation was to take place today, that it is unlikely to take NETS in the order of an hour to arrive in a case like this?"*[25]

Dr Stewart replied,

> *"Ah it's a reasonable assumption yes."*[26]

This is of course all somewhat academic; I have considered the issue in the context of the situation that prevailed at the time. The retrieval team was onsite at Frances Perry less than one hour after receiving the initial call. The decision to try to stabilise Baby Summer, prior to transfer to the Royal Children's Hospital was, in my view, appropriate medical management.

Ms Ellis, for Dr Manikkam, took the opportunity to raise what I see as quite an important point with Dr Stewart. Dr Stewart confirmed that on arrival, after a handover, the initial airway medical management of Baby Summer was CPAP, not intubation and ventilation. Ms Ellis put to Dr Stewart that if the retrieval team considered Baby Summer's respiratory distress to be "dire" they would have immediately proceeded to intubation. Dr Stewart agreed, he considered there was no real need to intubate until the sudden collapse at 11:45pm during the attempt to obtain arterial blood gases.

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[25] (T1041)

[26] (T1042)

I see no reasonable basis to be critical of any aspect of the involvement of NETS in the management of Baby Summer.

THE EXPERTS

59. I turn to address the evidence of the experts as it relates to the two key findings I believe I have to make:

- Cause of death
- Whether the claimed deficiencies in medical management could be viewed as causal factors in the death of Baby Summer

DR DODD

60. Dr Malcom Dodd is a Senior Forensic Pathologist at VIFM. Dr Dodd undertook a full autopsy and all manner of auxiliary investigations. His comprehensive Autopsy Report is constituted by Exhibit "A". In spite of exhaustive investigations, Dr Dodd was unable to determine the cause of death and categorised it as "*unascertained*". He did, however, conclude the sudden and unexpected death of Baby Summer was due to natural causes. He noted the cardiovascular system was "*unremarkable for age and development*" and there was "*no evidence of inheritable genetic abnormality*".

61. Dr Malcolm Dodd was asked about the extensive congestion and haemorrhage he observed in Baby Summer's lungs, which he described as "*gross*". Dr Dodd suggested the gross congestion observed was a "*non-specific*" finding, but opined it was a secondary, rather than a primary effect, reflecting a very poor degree of oxygenation to the lungs.

Dr Dodd, noting that PPHN is a clinical rather than a pathological diagnosis, accepted that while his examination did not confirm pulmonary hypertension, nor did he rule it out.

62. The most important aspect of Dr Dodd's evidence related to whether sepsis was demonstrated at autopsy. In answer to a question put to him by Ms Ellis, Dr Dodd agreed that if sepsis was fulminant, as the consensus of opinion suggested it was, so that it could be seen as a contributing factor in Baby Summer's death, then it would be likely it would be demonstrated at autopsy. He stated that as no indication of sepsis was seen under the microscope at autopsy; his view that sepsis was not a contributing factor in Baby Summer's death was confirmed.

63. Dr Dodd stated that VIFM protocols in relation to an infant death required "*a very exhaustive search for pathogens both viral and bacterial*" maintaining that bacterial and viral studies were non-contributing. Dr Dodd said:

> *"...there was no evidence at all that I could see under the microscope or after extended testing that there was any degree of sepsis whatsoever."*[27]

Dr Mukherjee was given leave to re-examine Dr Dodd on one particular point. In broad terms, he put to Dr Dodd that the antibiotics administered to Baby Summer both at Frances Perry late on 4th April and subsequently at Royal Children's Hospital early on 5th April may have supressed an infection. Dr Dodd responded:

> *"Sometimes negative microbiology can be a result of antibiotics within the blood that you're actually sampling and that's acknowledged."*[28]

For completeness, I think it is appropriate to include in the finding the relevant dialogue between Mr Mukherjee and Dr Dodd on the issue:

> *"And when you look back, can you as a pathologist can you tell or not tell whether it was the antibiotics or not?---No, not really. When we look at the tissue under the microscope if we see inflammatory changes and that would suggest an infective process. Yes?--- we didn't see any of that at any level in any organ. It's reassuring to see a whole lot of negatives here which is what we hope to see, it excludes infection, but you're correct, if antibiotics are in the blood that actually can inhibit the growth on a agar plate or in a blood culture bottle.*
>
> *Yes. And putting it very simply in lay terms, the antibiotics in that scenario would have done their job to cure the infection such that none was seen by the time it came to doing a culture result?--- Yes that's possible, yes."*[29]

64. In answer to a question from Ms Ellis, for Dr Manikkam, Dr Dodd agreed that if sepsis was said to be a contributing factor in Baby Summer's death, it would likely be fulminant and he saw no evidence of sepsis confirming his view that sepsis was not a cause of Baby Summer's death.

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[27] (T10)

[28] (T35)

[29] (T35)

65. The conclusion I have reached, in relation to the issue of sepsis being a contributing factor in Baby Summer's death, will be dealt with in the conclusions of this finding.

HINDSIGHT

66. Before turning to the evidence of Drs Erickson, Butt and Costello, I propose to say something about retrospectivity/hindsight. In his statement, Dr Erickson volunteered:

> *"In providing this summary I must note that it is extremely difficult to be certain of events that occurred in retrospect and from limited information. It is also worth noting that some management decisions which appear clear in retrospect may extremely complicated and difficult at the time."*[30]

67. Dr Butt gave a similar caveat/qualification saying:

> *"So I would imagine there are 100 babies like that every day; only one of them is going to end up in my care and it is easy for me to look back and say, well, that's the one we should have done something about, so we're coming from a different perspective. I have no doubt she has Pulmonary Hypertension. Then, the question is when do you think it was reasonable that it should be picked, not me."*[31]

And added,

> *"so it's easy for me looking back, to say if things had, you know, been done differently and so on, maybe the outcome wouldn't been different..." (my emphasis)*[32]

68. Dr Butt described the exercise he undertook to assist Ms Jamsek as:

> *"I'm trying to put the pieces of the jigsaw together to understand why she died."*[33]

Retrospectively (with the benefit of hindsight), trying to put the pieces of the jigsaw together, is reasonable to try determine what was the cause of Baby Summer's death, but, not appropriate to gauge the performance of those involved at the coalface in the medical management of Baby Summer.

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[30] Brief p64 (exhibit Y)

[31] (T969)

[32] (T974)

[32] (T986)

69. I wish to make it clear that I am not in any way critical of the process undertaken by Drs Butt and Erickson, they were merely seeking to provide answers; however, my task is considerably different. I am required to consider the whole body of evidence and determine whether any ACT or OMISSION could, on balance, be reasonably viewed as a causal and/or contributing factor in Baby Summer's death. In broad terms, I am required to put myself in the shoes of the clinician. I have to look at the circumstances as they unfolded without the not inconsiderable benefit of hindsight.

ASSOCIATE PROFESSOR BUTT

70. Associate Professor Warwick Butt, Director Paediatric Intensive Care Unit Royal Children's Hospital (RCH), provided a statement (exhibit "AA") and gave oral evidence. In short, in his statement, Dr Butt opined that the clinical diagnosis of Baby Summer's condition was "*severe pulmonary hypertension and cardiovascular collapse probably due to sepsis*". He further stated that the pulmonary hypertension was "*not recognised*" so that "*persistent acidosis hypercapnia and hypoxaemia continued and eventually led to profound right heart failure and cardiovascular collapse.*" He added that the severe degree of hypoxia and acidosis limited the active treatment able to be given at RCH, so that Baby Summer subsequently was palliated and died.

71. I include in this finding an excerpt from Dr Butt's statement because it goes directly to the issue I have identified as central to my deliberations; he wrote:

> *"Sonia asked if anything could have made a difference and I stated that earlier direction, treatment and transfer to RCH for ECMO may have made a difference. I did, however, stress that I felt the care given to Summer was appropriate and she unfortunately had a fulminant and rapidly progressive process."*[34]

Dr Butt accepted that his understanding of events and his opinions were based on an examination of medical records and presumably on information conveyed by Ms Jamsek. As he said, he was trying to put the pieces of the jigsaw puzzle together.

72. I turn to Dr Butt's oral evidence where, when examined by counsel for interested parties, quite often statements observations, conclusions and opinions are refined or elaborated

[34] Exhibit AA p.62

upon. When asked to explain as simply as he could, Dr Butt said Baby Summer died due to severe pulmonary hypertension which resulted in a,

> *"profound lack of oxygen for a prolonged period that impacted on her ability to provide blood flow and oxygen to her body."* [35]

Dr Butt was asked by Mr Mukherjee if severe pulmonary hypertension should have been recognised by 7pm. Dr Butt responded:

> *"...in retrospect it's very easy for me to say that I think she had substantial pulmonary hypertension at 7pm, or the beginnings of it" (my emphasis)*[36]

He went on to say:

> *"I'm sure she did have pulmonary hypertension then already, but that doesn't mean that what was done was inappropriate."*[37]

He elaborated by saying an "echo" could have confirmed it, but that would not be a standard practice. He added, however, that pulmonary hypertension was able to be diagnosed clinically when there was a clear demarcation-

> *"blue from the umbilicus down and pink up top."*[38]

73. At the conclusion of Mr Mukherjee's examination of Dr Butt, I sought some clarification on what I considered to be an important issue relating to the prospect that the pulmonary hypertension experienced by Baby Summer was due to sepsis. I viewed it as important, primarily, because it goes to the issue of when antibiotic therapy should have commenced. I pointed out that, in his statement and in evidence, Dr Butt had opined that the pulmonary hypertension was PROBABLY due to sepsis, but later in oral evidence, when pressed, he said the sepsis COULD NOT BE EXCLUDED. I indicated that, ultimately, I would need to consider the issue on the balance of probabilities, not as a possibility. Dr Butt said he could not resolve that issue for me as there were *"pros and cons to the diagnosis of sepsis."*[39]

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[35] (T964)

[36] (T968)

[37] (T969)

[38] (T971)

[39] (T976)

74. It appeared to me that Dr Butt came to the view that sepsis was, or could have been, the cause of Baby Summer's severe pulmonary hypertension by a process of elimination of other possible causes. I stress I am not critical of that process if every other potential cause is eliminated.

DR SIMON ERICKSON

75. Dr Erickson is a paediatric and neonatal intensive care specialist at the Princess Margaret Hospital for Children in Perth, Western Australia. Dr Simon Erickson was approached by Dr Butt, with whom he had worked, to endeavour to provide to Ms Jamsek answers to how Baby Summer died. Dr Erickson provided a statement and gave oral evidence by way of video link. Firstly, I thank him for his patience in light of the technical difficulties experienced in relation to the link.

76. Dr Erickson, having examined all the material available, concluded Baby Summer died from severe PPHN. He helpfully explained the phenomenon:

> *"PPHN results in the failure of the pre-natal circulation to adapt to birth with persistent pulmonary hypertension, persistence if the ductus arteriosus with right-left shunting and resulting hypoxia and acidosis which, if untreated, may result in cardiac dysfunction and death."*[40]

Dr Erickson, also undertook a process of elimination and concluded:

> *"In summary, although I feel confident that Summer suffered from severe persistent pulmonary hypertension of the newborn (PPHN) the cause of this is less certain, although sepsis would appear the most likely."*[41]

In reaching this non-definitive conclusion, Dr Erickson accepted that there were aspects of the evidence which did not support his hypothesis. While he placed some significance on the fact Ms Jamsek experienced some prolonged rupture of the membranes, he made no reference to the antibiotic therapy provided to Ms Jamsek by Dr O'Sullivan prior to birth. Dr Erickson also accepted that the pathological investigations did not support sepsis as being the cause of Baby Summer's PPHN.

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[40] Brief p64 (Exhibit "Y")

[41] Brief p66 (Exhibit "Y")

When requested to provide an opinion, Dr Erickson was asked whether he considered the initial antenatal management of Baby Summer was appropriate. I have taken his response to relate to the period from birth to the handover to NETS, as he made no criticism of their performance.

77. Dr Erickson maintained there were some "deficiencies" in initial management. He suggested the onset of respiratory distress and oxygen requirements warranted immediate intravenous antibiotic therapy. In that respect, it would appear Dr Erickson placed some reliance on the photographs tendered in evidence, in respect of which the preponderance of evidence suggests were, at best, inconclusive in relation to cyanosis. However, Dr Erickson said:

> *"It is possible, although difficult to know whether earlier antibiotic therapy would have altered outcome as Summer's clinical demise was extremely rapid and unresponsive to aggressive therapy."*[42]

This is an issue I will return to later in this finding.

78. Undertaking the same process that I have adopted in relation to the experts who were not actually involved in the treatment of Baby Summer, but have provided opinions, I turn to Dr Erickson's viva voce evidence. I am required to consider the totality of his evidence after he has been examined by counsel for the interested parties.

79. At the outset, Dr Erickson accepted that his claim in relation to the initial BSL persisting for three hours was incorrect and that the return to normal was in the order of two hours; however, I do not think that issue is critical.

I note that, in oral evidence, Dr Erickson volunteered the same qualification about the difficulty in providing expert opinions in retrospect from "*second hand information*"[43] and medical reports. He observed:

> *"...if you're not there, you don't really know what happened."*[44]

80. The crux of Dr Erickson's opinion as to the cause of death is encapsulated in the following excerpt, which was in response to Mr Mukherjee's invitation to "in a nutshell" say why he thought Baby Summer's death was due to PPHN, he stated:

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[42] Exhibit Y p64

[43] (T841)

[44] (T847)

"...that's based on the- the clinical features that- that Summer displayed, so she did appear to have intermittent symptoms based on the medical notes. That at times she was seen to look relatively well, and at times quite hypoxic, um, so it's based on that. It's based on the clinical features of, um, the lower limb lower limb cyanosis which- which was consistent with right to left shunting across the patent ductus arteriosus, which is, um, something that would be seen in persistent pulmonary hypertension in a newborn, but- but, um, the main support for that diagnosis was the echocardiographic findings at Royal Children's Hospital, Victoria, which showed that there was a large patent ductus arteriosus with right to left shunting across the duct, so shunting from the right side of the heart to the left side of the heart, and um, there was some dysfunction of the- of the cardiac function, particularly the right ventricle so those features are by definition persistent pulmonary hypertension of the newborn with right to left shunting after birth across a patent ductus arteriosus."[45]

Dr Erickson went on to say that PPHN is "*an extremely serious condition...and has quite a high mortality rate*".[46]

81. Dr Erickson formed the view that the "more likely" cause of Baby Summer's PPHN was sepsis, which he stated is a "very common cause" of PPHN, but conceded there were features that "*go against the diagnosis of sepsis*".[47] In that respect, Dr Erickson was taken to Dr Dodd's autopsy findings and asked about the prospect of the intravenous administration of antibiotics to Baby Summer at 11pm and 12:30am on the 5th April (the following morning) resulting in sepsis not being evident at autopsy. He replied:

"I don't think they would have had- at that stage had any influence on, um, the autopsy findings. I think it was- if sepsis was the cause then they wouldn't- they were administered too late in the- in the condition of Summer to- to make any difference to any of the findings." [48]

82. Dr Erickson maintained his view that, at the time Baby Summer was admitted to the Special Care Nursery, she should have been administered antibiotics, as her fluctuating condition and transient symptoms "*should have been treated as due to sepsis until proven*

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[45] (T848)

[46] (T848)

[47] (T849)

[48] ?

otherwise".[49] Dr Erickson explained the bases (what he referred to as "red flags") upon which he held that view, placing significant reliance upon low blood sugar levels. He conceded that the BSLs did come back up fairly quickly (up to 3.7mmol/L after an oral feed), but maintained that for a period of 1 to 2 hours the BSL was quite low. Dr Erickson reiterated his view that, by 8:30pm, there were sufficient concerning and clinically observable features as to administer antibiotics, even prophylactically, as the "*downside of giving antibiotics unnecessarily is very low*".[50] However, Dr Erickson accepted a contention put to him by Mr Halley that prior to 8:30pm he would not be prepared to say the management of Baby Summer by Dr Manikkam was other than reasonable.

83. In response to a proposition put to him by Ms Ellis, Dr Erickson accepted that "*the only real point of different management*"[51] between what Dr Manikkam did, and what he believes should have been done, was Dr Erickson would have administered intravenous antibiotics at 8:30pm; the clear influence being that otherwise he considered Dr Manikkam's management of Baby Summer to be reasonable.

84. Dr Erickson, noting that Baby Summer's condition was "grave" by the time she arrived at Royal Children's Hospital, with "no obvious cause of Baby Summer's condition", considered there was "some uncertainty" as to whether ECMO could have reversed the situation. In the event, the judgement call by clinicians at Royal Children's Hospital did not pursue that option.

DR SIMON COSTELLO

85. Dr Costello, a paediatrician/neonatologist engaged by the Court to provide an independent expert opinion provided statements[52] and also gave viva voce evidence.

86. In relation to the central issue, Dr Costello was asked if, in his opinion, the overall medical management of Baby Summer was "timely and appropriate". His response was:

> *"In my opinion there was prompt attention to this infant's issues. It was reasonable to give a formula feed after the initial low blood sugar level, given that there was no evidence of respiratory distress in a term infant who was likely to tolerate an oral*

[49] (T855)

[50] (T869)

[51] (T919)

[52] (Exhibits AE, AF and AS)

feed. There was improvement in the blood sugar level and normalisation by 3 ½ hours. Once respiratory distress emerged, Dr Manikkam attended promptly and arranged for the appropriate investigations and initial course of management with intravenous antibiotics."[53]

87. On the 8th day of the inquest, Dr Costello gave oral evidence where he was examined by counsel for the various parties. Interestingly, Dr Costello stated he has not seen a case like this where a baby has died without a specific cause being found. In that respect, he previously said he was "confident"[54] Baby Summer died due to PPHN, I have assumed, in saying he has never seen a case like this before where the cause of death is unascertained, he refers to what precipitated the PPHN. Dr Costello said that, while he could not give a definitive answer, sepsis would be the "first cause" he would go to, the one that is most likely. However, when pressed by Mr Mukherjee as to whether he agreed with Dr Erickson that sepsis was the "most" likely cause of Baby Summer's PPHN, Dr Costello elaborated and said:

"sepsis would have to be a consideration." [55]

I see a difference between <u>most likely</u> and a <u>mere consideration</u>. It seems to me Dr Costello was not prepared to express a firm view about sepsis being the most likely cause, due to the negative results at autopsy and other matters.

88. Dr Costello opined that as Baby Summer was of 37 week gestation it was reasonable to conclude that the desaturations observed prior to 9:30pm were due to transient tachypnoea of the newborn, or perhaps mild respiratory distress syndrome and it was appropriate to treat the baby accordingly. But he agreed the clinical picture had changed by 9:30pm, with oxygen requirements steadily increasing between 8:30pm and 9:30pm.

Dr Costello considered it would have been appropriate to initiate antibiotic therapy sometime between 8:30pm and 9:30pm because by then, among other things, there was a,

"<u>trend</u> towards a deterioration in the baby's respiratory status"[56] *<u>(my emphasis)</u>*

89. It was put to Dr Costello that he was "struggling to formulate"[57] in his mind clear conclusions as to the precise underlying cause of Baby Summer's death. Dr Costello

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[53] (Exhibit AE)

[54] (T1070)

[55] (T1077)

[56] (T1088)

accepted that he could not come up with a specific cause, but maintained his view that basically Baby Summer's left ventricle function was severely compromised, as demonstrated in the echocardiogram subsequently undertaken at RCH. Dr Costello reiterated that, in light of the rapid deterioration, coupled with Dr Dodd's findings at autopsy, the prospect of Baby Summer's demise being due to sepsis "*would have to be a consideration*". It seemed to me that Dr Costello was reluctant to go as far as to say it was the probable cause. I perceived some equivocation, perhaps even internal contradictions in Dr Costello's evidence. This demonstrates the dilemma I face, with highly qualified, experienced paediatricians having difficulty confidently formulating a precise cause for the death of Baby Summer.

90. In examination by Ms Ellis, Dr Costello was taken to other issues, which she described as "*troubling parts of the jigsaw puzzle*"[58]. Dr Costello accepted that the IT ratio (the higher the ratio the more indicative of a bacterial infection) and CRP were not necessarily definitive but "useful tools" to assist in the decision-making process of when to institute antibiotic therapy. Dr Costello opined that the IT ratio of 0.18 (prior to the subsequent test at the RCH at 1:50am, which demonstrated an IT ratio of 0.21), whilst an additional tool, together with other tests and clinical assessment, would not have necessarily added to the index of suspicion that Baby Summer's condition was due to sepsis. Dr Costello indicated that all these issues, in combination, were the bases of his reluctance to agree to PPHN due to sepsis being the fundamental reason for Baby Summer's rapid deterioration and death. Apparently, when the parts of the jigsaw puzzle were in place it did not demonstrate a clear picture to Dr Costello.

EVIDENCE OF FAMILY MEMBERS

91. I wish to make it clear that I have not entirely dismissed the evidence given by Ms Jamsek, Ms Jamsek's mother, Mrs Sandra Sharp, both of whom gave evidence at the hearing, and Mrs Marion Evans (who provided a statement), and all of whom said they raised concerns about Baby Summer's condition with midwife Tsesmetzis in the period shortly after the birth. Their evidence in that regard is uncontradicted, as Ms Tsesmetzis maintains she had

[57] (T1075)

[58] (T1099)

no recollection of events. I note, however, that at the time neither Ms Jamsek, Mrs Sharp or Mrs Evans raised their specific concerns with Dr O'Sullivan or more senior nursing staff.

92. More significantly, and with respect, neither Ms Jamsek, Mrs Sharp nor Mrs Evans, have the necessary qualifications or experience to voice opinions on medical matters. While I have heard what they say, it is all about how much weight one attaches to their opinions. To support their concerns, reliance was placed upon the photographs and videos in evidence. However, virtually all the experts agreed that the photos and videos did not clearly demonstrate that Baby Summer was in parlous condition at the time.

CONCLUSION

93. From my perspective, the critical issues in this case are:

- 'did Baby Summer die due to PPHN?'; and
- if so, 'was that condition brought about by sepsis?'; and
- if it was, 'when would that be manifest so that intravenous antibiotic therapy should have been administered?'

The other significant question following on from that is:

- if the rapid deterioration and death of Baby Summer was due to sepsis, would the administration of antibiotics, when sepsis became apparent, have altered the outcome?
- could the condition have been reversed, such that Baby Summer could have survived?

As to those critical findings the applicable standard of proof is determinative. The "*Bringinshaw Test*" must be applied; I have to be comfortably satisfied as to determining facts.

94. In submission Mr Mukherjee for Ms Jamsek, relying on aspects of Dr Costello's evidence, suggested the preponderance of evidence supports the contention that Baby Summer died due to "*secondary pulmonary hypertension caused by sepsis*". I suggest that, in that part of his evidence, while Dr Costello appears to be agreeing with Dr Erickson's view that PPHN caused by sepsis is likely, when one views the totality of his evidence, his opinion is not as

clear as is claimed; indeed, some aspects of Dr Costello's evidence appear to be contradictory.

95. On behalf of Dr Manikkam, Ms Ellis submitted that the weight of evidence, even if I was satisfied death was due to PPHN, is not sufficient to safely conclude that on balance that condition was due to sepsis, only that is a possibility, perhaps even likely.

96. Ms Keeling, for Dr O'Sullivan, also submitted that the evidence is not sufficient to conclude Baby Summer's death was associated with infection. In fact, Ms Keeling maintained that the cause of Baby Summer's death should, as Dr Dodd concluded, remain unascertained; claiming that if I am satisfied Baby Summer had PPHN, it was secondary to an unascertainable cause, so that as a consequence of an "underlying unknown problem" PPHN cannot be considered to be the cause of death; an interesting submission!

97. Ms Keeling, in submission, carefully analysed the evidence in support of the contention that sepsis was the underlying cause of Baby Summer's death. I have undertaken the same exercise. In summary, Ms Keeling submitted that when one looks at the entirety of the evidence, at its highest, it does not reach the required standard to conclude that sepsis was the underlying cause of death.

98. Having given the matter earnest consideration, conceding some vacillation, I find myself comfortably satisfied on the balance of probabilities that Baby Summer died due to PPHN, but I am not comfortably satisfied that that condition was caused by sepsis. In my considered view, there are just too many aspects of the evidence, particularly that of Dr Dodd, which do not support a finding of sepsis being the underlying cause of Baby Summer's death.

99. I should not be taken as saying sepsis was not a possibility, it is. What I am saying is that the evidence does not, to my satisfaction, lead me to the view that it was more probable than not. The distinction, although somewhat subtle, is in fact real.

100. If I am wrong in coming to that view, and the underlying cause of Baby Summer's death was indeed sepsis, I consider it appropriate to make a formal finding as to whether the administration of antibiotics, when it was reasonable to consider underlying infection being a distinct possibility, would have altered the outcome.

101. Firstly, I do not accept that Dr Manikkam decided to order antibiotics only after being advised to do so by NETS. I accept that he took that decision himself and ordered the antibiotic therapy, which was subsequently administered at about 11pm. It is, however,

unclear to me why it took an hour after he attended for antibiotics to be actually administered.

102. I do not consider there were sufficient concerning features of Baby Summer's presentation prior to 8:30pm to warrant antibiotics being provided. On this issue, there was broad consensus amongst the relevant experts, Drs Butt, Erickson and Costello.

103. Dr Erickson initially said it would be "*difficult to say*"[59] if the earlier administration of antibiotics would have made a difference. He said that not providing antibiotic therapy prior to 8:30pm was a "*reasonable approach*".[60] Importantly, Dr Erickson opined that it was possible, but not probable,[61] that administration at 8:30pm may have altered the outcome; noting on several occasions that the deterioration was a relentless process. Put at its highest, Dr Erickson stated that the prophylactic administration of antibiotics at 8:30pm, when he suggested it would have been appropriate to do so,

> *"may have tempered the deterioration and you know there's a small possibility that the baby may have been able to have been managed more aggressively by the time it arrived at the Royal Children's and may have survived."*[62]
>
> *"But it's very difficult to- you know, it's very difficult to predict and I would say it's possible but probably not probable (my emphasis)"*[63]

104. In his statement, Dr Butt said:

> *"Sonia asked if anything could have made a difference and I stated that earlier detection, treatment and transfer to RCH for ECMO may have made a difference. I did however stress that I felt the care given to Summer was appropriate and she unfortunately had a fulminant and rapidly progressive process."*[64]

He did, however, say that he may have "*done things differently*" in regard to the administration of antibiotics, but opined that, even if administered at 8:30pm, or even 7pm, it would have been "*unlikely to have made a difference*".[65]

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[59] (T869)

[60] (T871)

[61] (T872)

[62] (T873)

[63] (T873)

[64] Brief p62 (exhibit "AA)

[65] (T1003)

105. I consider an argument can be made that antibiotics should have been administered prior to 11pm, perhaps even as early as 8:30pm, but not before. I think it would have prudent to "cast the net widely" (if I may use that expression). In my view, that is the only basis that Dr Manikkam's management of Baby Summer can be reasonably questioned.

106. In summary, there was no compelling evidence that the administration of antibiotics at, or after it was reasonable to consider infection as a cause for Baby Summer's fluctuating condition, would more likely than not, have salvaged the position. On that basis, that omission in the medical management cannot in my view, be seen as a causal factor in her death.

In the final analysis, Baby Summer died due to a fulminant, relentless, natural cause, aptly described by Mr Mukherjee as a "complex clinical phenomenon". Her management was significantly complicated by intermittent fluctuation of symptoms.

COMMENT

107. I am satisfied the extent of investigation, at least since I took over carriage of the matter, has been sufficient to satisfy community expectation. There was a fulsome eight-day inquest, during which my assistant played an active part and each of the interested parties was ably represented by counsel. Examination of witnesses was extensive, so that each party had an opportunity to put matters supporting their respective positions.

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

Ms Jamsek, Senior next of kin

Dr Manikkam

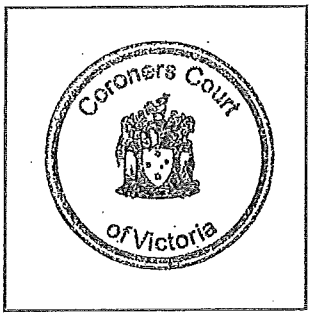
Dr O'Sullivan

Frances Perry House

Ms K Russell

Coroner's Investigator

Signature:



PHILLIP BYRNE
CORONER
Date: **21 June 2016**