QUEENSLAND COURTS

CORONERS COURT OF QUEENSLAND

FINDINGS OF INQUEST

CITATION:	**Inquest into the death of Thea Flaskett**
TITLE OF COURT:	Coroners Court
JURISDICTION:	BRISBANE
FILE NO(s):	2023/4380
DELIVERED ON:	17 July 2026
DELIVERED AT:	BRISBANE
HEARING DATE(s):	Pre-inquest conference 18 June 2025 Inquest 1 – 5 September 2025; 19 September 2025 Written submissions following inquest 19 November 2025 – 31 March 2026
FINDINGS OF:	Stephanie Gallagher, Deputy State Coroner
CATCHWORDS:	Coroners: inquest, potential health care related death, in utero morphology scanning for risk, baby born with undiagnosed cardiac abnormality (transposition of the great arteries), reduced shunting, resuscitation, non-preventable, natural causes, death

REPRESENTATION:

Counsel Assisting:	G Diehm KC and S Ford
The family:	S Lane, instructed by Gilshenan & Luton
Metro North Hospital and Health Service:	A Freeman KC, instructed by MinterEllison
Mr Collins:	A Cappellano, instructed by Moray & Agnew
Dr Sinnott:	R Natrass, instructed by Avant Law
Ms Leddy:	R Oldfield, instructed by DLA Piper

Contents

Introduction

1. Thea Flaskett ('Thea'), daughter of Meg and James Flaskett, was born at the Redcliffe Hospital on 11 September 2023 at 12:07am. Having been born with an undiagnosed cardiac abnormality and following unsuccessful resuscitation efforts, Thea died approximately four hours later.

2. Thea's death was a reportable death for the purposes of s8(3)(d) of the *Coroners Act 2003* ('the Act').

3. Due to concerns raised by Thea's family, I determined, pursuant to s28(1) of the Act, that it was in the public interest to hold an inquest into Thea's death.

The investigation

4. During the coronial investigation, medical records for both Thea and Mrs Flaskett were obtained from Metro North Hospital and Health Service ('MNHHS'),[1] Retrieval Services Queensland and Imaging Queensland. Records concerning Mrs Flaskett were obtained from the Rothwell Family Practice. Those records encompassed the perinatal, intrapartum and postpartum care afforded to Mrs Flaskett and Thea.

5. Statements from a number of those involved in the care of Mrs Flaskett and Thea were also obtained. These included statements from sonographers, a radiologist, obstetricians, paediatricians, neonatologists and nurses. The Redcliffe Hospital also produced statements from the Director of Medical Imaging Services.

6. Six experts were engaged by the court to provide opinions. Two neuropathologists, an obstetrician and gynaecologist, a neonatologist, a paediatric cardiologist, and a specialist in obstetric and gynaecological ultrasound produced reports which were received into evidence. The court also received an expert opinion provided by a specialist neonatologist to the Office of the Health Ombudsman. The coronial investigation pursued enquiries identified from the records as well as those arising from concerns identified by Thea's parents, where appropriate.

Autopsy results

7. On 14 September 2023, Staff Specialist Forensic Pathologist, Dr Jack Garland, conducted an external and internal examination of Thea's body to establish her cause of death.[2]

8. The post-mortem examination showed that there were a number of injuries sustained to Thea's head, including haematoma and haemorrhage. Dr Garland also considered that there existed potential

[1] This included records from the Redcliffe Hospital and NeoRESQ.
[2] Ex A2.

histological features of persistent pulmonary hypertension. He noted that a paediatric pathologist could review the relevant slides if required.[3]

9. Dr Garland opined that Thea's underlying cause of death was Transposition of the Great Arteries ('TGA'):[4]

    1(a) Main disease or condition in neonate: Transposition of the great vessels.
    1(b) Other diseases or conditions in neonate: Umbilical phlebitis.
    1(c) Other maternal diseases or conditions affecting neonate: Acute chorionitis and maternal vascular malperfusion.
    2. Other relevant circumstances: Vacuum extraction birth due to abnormal cardiotocography.

10. In relation to the haemorrhages identified during the post-mortem, Dr Garland's conclusion was to the effect that they were not significant in terms of Thea's cause of death, though the possibility that they made a minor contribution to Thea's death, in the setting of the TGA, could not be excluded.

11. On 20 March 2024, following discussions with two paediatric pathologists and a neuropathologist, Dr Garland produced an amended autopsy report.[5]

12. On a review of the lungs slides and the neuropathology findings, it was determined that persistent pulmonary hypertension was unable to be diagnosed histopathologically after such a brief window of life. While the intracranial pathology was not considered to be the substantial pathology in Thea's case, consistent with Dr Garland's initial opinion, it could not be excluded that the haemorrhages made a minor contribution to Thea's death, in the setting of the TGA.[6]

13. Dr Garland revised his opinion as to Thea's death by including intracranial haemorrhage:

    1(a) Main disease or condition in neonate: Transposition of the great vessels.

    1(b) Other diseases or conditions in neonate: Umbilical phlebitis ***and intracranial haemorrhage***.

    1(c) Other maternal diseases or conditions affecting neonate: Acute chorionitis and maternal vascular malperfusion.

    2. Other relevant circumstances: Vacuum extraction birth due to abnormal cardiotocography.

[3] Ex A2, p 16.
[4] Ex A2, p 17.
[5] Ex A3.
[6] Ex A3, p 1.

14. Following receipt of the amended autopsy report, the court engaged the Victorian Institute of Forensic Medicine to provide an opinion about Thea's autopsy results and histopathology. Neuropathologist, Associate Professor Linda Iles, and forensic pathologist, Dr Sarah Parsons, each reviewed the material and produced reports.[7] I will say more about those opinions further in these findings.

15. I do note at this juncture though that Dr Parsons opined that Thea's cause of death should be assigned as:[8]

    1(a) Unascertained.

The inquest

16. Investigations undertaken between the pre-inquest conference and the inquest resulted in three of the eight contemplated inquest issues no longer requiring examination, with the concurrence of the parties granted leave to appear, including Thea's family. The issues not requiring further consideration were suspected maternal malperfusion as a contributing cause of death, the care provided during labour and whether a caesarean section should have been performed (rather than a vacuum extraction).

17. The issues examined during the inquest, in addition to the findings required by s45(2) of the Act, were confined to the following:

    Issue 1: With respect to the cause of Thea's death, specifically, was it:

    (a) transposition of the great arteries; and/or
    (b) some other cause.

    Issue 2: Should transposition of the great arteries have been detected on imaging by the 20-week scan or by any other clinical indicator or investigation which ought appropriately been undertaken?

    Issue 3: If the answer to the above question is "yes", what would then have happened and would Thea's death have been avoided as a result?

    Issue 4: Did any inadequacy in the post-delivery care contribute to Thea's death?

    Issue 5: Are any changes to the practices, procedures or policies of the health service appropriate to reduce the likelihood of deaths occurring in similar circumstances or to otherwise contribute to public health and safety?

18. The following witnesses gave oral evidence at the inquest:

[7] Exs B1.1, B1.1.1, and B1.2.
[8] Ex B1.2, p 4.

i. Dr Stephen Sinnott
ii. Amanda Leddy
iii. Craig Collins
iv. Dr Georgia O'Sullivan
v. Dr Pieter Koorts
vi. Dr Didier Tshamala
vii. Dr Melissa Luig
viii. Professor Edward Weaver
ix. Associate Professor Nick Evans
x. Dr Timothy Colen
xi. Dr James Kelly
xii. Dr Nicole Woodrow

19. I have not had regard to any other expert evidence other than from those whose reports were received in evidence.

20. At the inquest, legal representatives appeared on behalf of Thea's family, MNHHS, Ms Leddy, Mr Collins, and Dr Sinnot. The legal representatives for Thea's family withdrew subsequent to the hearing of the evidence and the provision of Counsel Assisting's submissions.

Consideration of issue 1: Cause of death

Underlying cause of death

21. Thea's family have submitted that I should find that the cause of Thea's death remains "unascertained". For the reasons which follow in more detail below, I am satisfied that the cause of Thea's death has been ascertained, on the evidence before me. In coming to the conclusions I have, I am mindful that the findings I should make are those that I am satisfied are correct, on the balance of probabilities. The findings I should make as to the cause of death are what is probable, not what is necessarily certain.

22. I am also conscious that in some coronial matters, such as this one, the postmortem evidence and the opinions expressed upon it, do not always of themselves reveal conclusive answers to questions that arise for asking. Evidence from clinicians with relevant expertise in managing labour and especially in managing neonates compromised at birth, can be critical in coming to a conclusion as to cause of death, answering questions pathologists cannot. Indeed, sometimes the opinions of clinicians may be wholly or partly at odds with the opinions of pathologists but yet be right to be accepted. The task for me is to evaluate the weight I should give to the opinions of each the expert clinicians and pathologists, in the context in which they are each offered and against the apparent facts of the case otherwise established. I have been particularly assisted by Counsel Assisting's detailed submissions in that task.

23. I am satisfied that Thea's underlying cause of death was TGA, a congenital heart defect. I note that of all of the experts qualified to express a view on this matter, Dr Parsons was the only one to not hold this view. As noted above, Dr Parsons did not conclude in favour of some other cause of death.

24. TGA is a major congenital heart malformation of the arteries leaving the heart, while the structure of the heart is otherwise normal. In TGA , the aorta, which should be connected to the left ventricle, is connected to the right ventricle, and the pulmonary artery ends up connected to the left ventricle, when it should be connected to the right ventricle.[9] Dr Parsons did not doubt that Thea had TGA.

25. As was explained by Associate Professor Nick Evans, Clinical Associate Professor in Neonatal Medicine at the Royal Prince Alfred Hospital:[10]

> "In TGA, the pulmonary artery and aorta are transposed so the pulmonary artery is connected to the left ventricle and the aorta to the right ventricle. The result is two circulations in parallel with deoxygenated blood going around the systemic (body) circulation and the oxygenated blood going around the pulmonary (lung) circulation. The only way that oxygenated blood can get to the body is if there is a connection between the two sides of the heart which allows the oxygenated blood to mix into the blood going to the systemic (body circulation). So TGA is incompatible with life unless there is a connection between the two sides."

26. Associate Professor Evans said the degree of cyanosis and compatibility with life can depend on the extent to which the foramen ovale[11] restricts the two-way flow between the heart's two atria. In Thea's case, she was born with TGA "almost certainly" involving a very restrictive foramen ovale, which resulted in her failing to establish normal or adequate oxygenation after birth. Essentially, this meant that not enough oxygenated blood was able to reach Thea's systemic circulation.[12] Unlike in many other cases of TGA, where life can be sustained long enough to get definitive treatment, Thea's prospects of surviving were diminished by an inadequate mixing of oxygenated and deoxygenated blood to enable survival.

27. Dr Melissa Luig, specialist neonatologist, gave evidence that in addition to the foramen ovale, the ductus arteriosus is relevant to the mixing of blood. While the baby is in-utero, the ductus should be open. Ordinarily, it remains open after birth for around 24 hours, even if the baby is unwell or

[9] Ex C1.11, [3.26].
[10] Ex B1.6, [41].
[11] In the fetus, the foramen ovale allows blood to flow from the right atrium to the left atrium so it can bypass the lungs and be returned to the placenta. The foramen ovale is a hole with a flap which acts as something of a one way valve, allowing blood to flow right to left but restricting flow left to right (Ex B1.6, [43]).
[12] Ex B1.6, [36] and [44].

preterm. The trigger for the ductus to close is good oxygenation[13] throughout the "whole of the bloodstream".[14] The width of that ductus after delivery is an important factor in how much mixing can occur – the wider it is, the more mixing can occur.[15]

28. In evidence, Dr Luig was taken to the postmortem evidence showing that in Thea's case, the foramen ovale was identified (at autopsy) as being patent with a size of one centimetre. Dr Luig considered one centimetre as being a reasonable size.[16] Her evidence concerning restriction of the foramen ovale was mostly consistent with the evidence of Associate Professor Evans, who explained that whether the foramen ovale is restrictive or not depends not on the size of the window, but on the extent to which the flap allows mixing between the two chambers. The restriction is not determined by the size of the hole but rather, the extent to which the flap restricted flow between the two chambers.[17]

29. Dr Luig said that Thea's rapid deterioration after birth was in "keeping with TGA with intact ventricular septum associated with either a restricted shunting or pulmonary hypertension or both".[18] She explained that shunting, which can occur through a patent foramen ovale, as "Some blood will flow between left and right so that some blood that's blue or deoxygenated can go to the side that's going to the pulmonary artery and become oxygenated so the oxygenation can only happen at the lungs."[19]

30. Dr Timothy Colen, paediatric cardiologist, strongly suspected that reduced shunting, whether because of a small atrial septal defect (another term for a patent foramen ovale[20]) and/or a restriction of the ductus arteriosus, were "very likely the major problems".[21] This contributed to Thea's rapid deterioration and death in the context of the TGA.[22]

31. According to Dr Luig, babies born with TGA but who do not have reduced shunting (and in turn have sufficient mixing of the blood) can survive until they reach a tertiary centre (if not born in one). The reduced shunting in Thea made the TGA in her high-risk and rare.[23]

Other contributory factors

32. Whether any other condition contributed to or was relevant to Thea's cause of death, was the subject of evidence during the inquest.

[13] T3-20, LL 20-24.
[14] T3-20, LL 30-34.
[15] T3-20, LL 10-16.
[16] T3-20, L 2.
[17] T4-24, LL 8 – 19.
[18] T3-24, LL 36-38.
[19] T3-24, LL 43-46.
[20] T5-9, LL 21-24.
[21] T5-11, LL 26-29.
[22] T5-17, LL 4-7.
[23] T3-43, LL 15-31.

The amended autopsy findings

33. As noted above, Dr Garland's revised conclusions on contributory factors to Thea's death were as follows:

    1(b) Other diseases or conditions in neonate: Umbilical phlebitis and intracranial haemorrhage.

    1(c) Other maternal diseases or conditions affecting neonate: Acute chorionitis and maternal vascular malperfusion.

    2. Other relevant circumstances: Vacuum extraction birth due to abnormal cardiotocography.

34. In Associate Professor Evans' opinion, none of umbilical phlebitis, acute chorionitis, maternal vascular malperfusion, nor vacuum extraction contributed to Thea's death.[24]

35. Professor Edward Weaver, obstetrician and gynaecologist, also disagreed with Dr Garland's conclusion that the vacuum extraction was relevant to cause of death. As he explained:[25]

    "I think that the bleeding that was – that – that – that was seen at autopsy was likely related to the normal labour process, that we know that that's a relatively common thing, and – and provided that the baby doesn't have any signs of a blood clotting disorder, that it's a – it – it usually is inconsequential, certainly with using a vacuum extraction or more commonly with using obstetric forces, you can – you can get –um – slightly more bleeding, but again, it – it's often irrelevant in terms of the prognosis for the baby. Um – so given the – from reading the autopsy reports, the volume of blood that was there seemed to be quite small, and I – I do not believe that it – that it – that it was associated with – um – any causation of – of Thea's death."

36. Professor Weaver said that in Thea's case, the mode of delivery had nothing to do with the outcome[26] and a vacuum extraction was a reasonable way to proceed.[27] Associate Professor Evans expressed a similar opinion.[28]

37. As to Dr Garland's addition of "intracranial haemorrhages" at 1(b) of his cause of death findings, Associate Professor Iles, who reviewed Dr Garland's findings, concluded that the haemorrhages observed were not

[24] T4 – 29, LL 1-15; T4-33, LL 16-23.
[25] T4-8, LL 5-24.
[26] T4-12, LL 20-21.
[27] T4-11, LL 28-29.
[28] T4-6, LL 27-40.

unexpected given the setting in which Thea was delivered. She opined that they were neither an independent cause of death nor a contributing factor.[29] Associate Professor Evans agreed with this.[30] He observed that haemorrhages are a "really common incidental finding in…newborn babies, whether or not they've had vacuums…"[31]

Pulmonary hypertension

38. The possibility of there being some thickening of the pulmonary arteries consistent with pulmonary hypertension was mentioned by Dr Garland in his addendum autopsy report. He concluded, however, that persistent pulmonary hypertension[32] ('PPH') was unable to be diagnosed on histopathology due to Thea's short window of life.[33]

39. Associate Professor Evans gave evidence that the thickening of the pulmonary arterioles as identified by Dr Garland was a feature of vascular remodelling associated with pulmonary hypertension. In Thea's case, it is possible that it "played into" her severely poor oxygenation – making it plausible that pulmonary hypertension "was an additional factor in Thea's rapid demise". [34]

40. Associate Professor Evans explained pulmonary hypertension in these terms:[35]

> "The normal state of the fetal pulmonary arterioles in utero is constricted, this limits blood flow to the lungs and diverts that blood to the placenta. One of the first and most important changes in cardiorespiratory transition at birth is that these arterioles dilate to allow blood to flow into the lungs. When this dilatation fails, that is called pulmonary hypertension of the newborn (PPHN) and blood flow into the lungs continues to be restricted and so further compromising oxygenation."

41. Pulmonary hypertension is classified (by some) as an associated abnormality to TGA, as opposed to a distinct pathology.[36] Associate Professor Evans said that while TGA was the dominant pathology here, pulmonary hypertension was certainly a significant *associated* pathology.[37]

[29] Ex B1.1, p 3.
[30] Ex B1.6, [19].
[31] T4-28, LL 26-33.
[32] Persistent hypertension is a specific condition in newborns. It is a *type* of pulmonary hypertension that has persisted in utero.
[33] Ex A3, p 1.
[34] Ex B1.6, [45] – [47]. See also: T4-21, LL 31-43.
[35] Ex B1.6, [46].
[36] Ex B1.6, [47].
[37] T4-21, LL44-46.

42. Associate Professor Evans gave evidence that, in some cases, PPH is seen in conjunction with TGA.[38] Literature cited in his report referred to a cohort of 112 babies, of which 14 babies with antenatally undiagnosed TGA also had PPH. Of the 14 babies, four died before they could be operated on.[39]

43. Associate Professor Evans referred to PPH as being a recognised comorbidity of TGA.[40] Dr Luig, although not classifying it as a 'comorbidity', agreed that there is an association between the two:

> "…pulmonary hypertension in association with transposition is a particularly severe condition and can be associated with transposition as part of the maldevelopment of the pulmonary vascular tree within the lungs, as part of the haemodynamics in foetal life, which are not normal."[41]

44. In Dr Luig's opinion, it was most probable that, having regard to the changes seen on Thea's lungs at autopsy, the severe degrees of cyanosis and metabolic acidosis, and the fact that the ductus had not closed, Thea had PPH.[42] Dr Luig explained that in Thea's case, the problems lay in both the lung vessels and the air spaces – both of which developed before birth. Where the air spaces were poorly aerated, meaning they were not opening well, blood was present in those spaces, and where Thea's lungs were very heavy, Dr Luig considered that another lung disease, in combination with PPH, may have been present. [43] She said that TGA when combined with severe PPH is a "fairly rare" condition.[44]

45. Dr Colen was also of the opinion in his report that PPH was present. When asked to comment on Thea's cause of death, he opined that the most likely cause of death was TGA with a high-risk feature such as pulmonary hypertension or small atrial septal defect (the latter of which has been discussed above).[45]

46. When giving oral evidence about this, however, Dr Colen could not definitively say that Thea had PPH. Rather, he said that it was a "possible cause" insofar as her not surviving.[46] He considered that reduced shunting was the major problem, having reviewed the neonatal resuscitation records before giving oral evidence.

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[38] T4-22, LL 1-4.
[39] Ex B1.6, [47].
[40] Ex B1.6, [47].
[41] T3-23, LL 31-34.
[42] T3-24, LL 1-11.
[43] T3042, LL 24-47.
[44] T3-46, LL 37-39.
[45] Ex B1.7, p 5; T 5-11, LL 1-8.
[46] T5-11, LL 19-29.

47. Dr Colen did not consider PPH as being a "complication" of TGA. He said it is a risk for every newborn,[47] and did not consider there to be a "real connection" between PPH and TGA – it was coincidental rather than causally connected:[48]

> "There are some papers written around persistent pulmonary – pulmonary hypertension in transpositions that suggest that the slight difference in fetal blood flow in a TGA normal versus a heart with slightly more oxygen potentially going to the lungs in fetal life may make babies more – ah – likely to have pulmonary hypertension with a TGA; however, pulmonary hypertension with a TGA is not common and every baby with TGA has that increased blood flow – increased oxygen going towards the lungs. So I – I'm not sure I believe there is a real connection."

48. He accepted, though, that for babies born with TGA, PPH does make them more vulnerable,[49] and Thea was high-risk given all of the conditions/factors that were present.[50]

49. Dr Colen said that death of a baby from TGA within four hours after birth is not generally expected. However, in Thea's case, a combination of TGA, possible PPH and reduced shunting led to her rapid deterioration and affected her chances of survival.[51] Further, that TGA combined with "possibly" PPH and limited shunting at birth is very rare.[52]

50. At the conclusion of his oral evidence, Dr Colen agreed to a summary that "Thea's case is that one of two things or a combination of them: she didn't have adequate mixing to allow that survival, whether it be through the atrial septal defect or any other means, and possibly, as well, that she had persistent pulmonary hypertension as a factor, as well, making her more vulnerable."[53]

Conclusions

<u>Underlying cause of death</u>

51. As indicated earlier in these findings, I am satisfied that Thea's underlying cause of death was TGA. Dr Luig, Professor Weaver, Associate Professor Evans, and Dr Colen, all agreed with this autopsy finding. These expert witnesses possess the requisite qualifications and experience to do so.

52. I note that Dr Parsons' conclusion that "Whilst the finding of transposition of the great vessels is a significant finding this does not usually cause

[47] T5-11, LL 37-42.
[48] T5-11, LL 44-47 to T5-12, LL 1-7.
[49] T5-11, L 42.
[50] T5-13, LL 45-47 to T5-14 LL1-11.
[51] T5-16, LL 39-45 to T5-17, LL1-16.
[52] T5-20, LL30-32.
[53] T5-23, LL 23-28.

death in the circumstances documented",[54] and gave her recommendation that the cause of death be assigned as 'unascertained'. That she reached that conclusion is consistent with the other expert evidence to the effect that TGA would not normally be expected to result in death this quickly. What the other experts were able to identify was that in Thea's case, unfortunately, there were one or more factors that made her condition unsurvivable, as is known to happen with other babies and explains the rapidity of her demise. As clinicians familiar with the variable course for babies with TGA, from their own respective experience as well as the literature in their fields, I am satisfied that I should accept TGA as the cause of death in Thea's case, due to her particular circumstances being high risk.

Contributory factors

53. Insofar as contributory factors, Dr Garland's conclusions as contained at 1(b), 1(c) and 2 are the subject of significant disagreement by the experts and cannot be accepted. The evidence of Professor Weaver, Associate Professor Evans, and Associate Professor Iles in this regard, is preferred by me.[55] The clinical experience of Professors Weaver and Evans are particularly persuasive to me, on these issues. They, I expect, see the cases where babies survive such identified pathology, whereas pathologists do not. Unlike the issue of TGA causing death, they did not identify any circumstance for Thea that meant she would not survive the identified pathology considered contributory by Dr Garland, with or without the effect of the TGA.

54. The evidence about the TGA having been complicated by reduced shunting is persuasive and should be accepted. Dr Colen, Associate Professor Evans and Dr Luig all agree that in Thea's case, reduced shunting was likely present and made the TGA high-risk and rare and it contributed to her rapid deterioration and death. That doesn't mean that reduced shunting is a separate disease or condition causing death. It is properly regarded as a circumstance that meant Thea could not survive TGA.

55. As to PPH, there is controversy as to whether it is associated with TGA, as in whether it is caused by TGA or may just in some cases be independently concurrent.

56. Notwithstanding this difference in opinion, Dr Colen, Associate Professor Evans and Dr Luig all provided cogent evidence as to PPH as being potentially contributory and as such a complicating factor for Thea's chance of survival. Their confidence as to its presence varied between them and at times for each of them in their expression.

[54] Ex B1.2, p 4.
[55] Dr Colen was unable to comment on Dr Garland's cause of death findings beyond TGA.

57. It should also be observed that Dr Georgia O'Sullivan's "most likely" differential diagnosis was hypoxic-ischemic encephalopathy ('HIE') with severe pulmonary hypertension, based on what she knew from her clinical involvement. In her experience, that is the most common cause for a term baby who, like Thea, had very low oxygen levels.[56] Albeit retrospectively, Dr O'Sullivan recorded the possibility of PPH in her notes for 11 September 2023:[57]

> "Our difficultly oxygenating Thea, I feel, was likely due to her poor cardiac output, although an undiagnosed cardiac lesion or severe PPHN is a possibility"

58. When asked whether PPH should be included with Thea's cause of death, Associate Professor Evans provided this response:[58]

> "Yes, I think you – I think you could. Ah – well, if – you see, because – this goes to the fact of this being a haemodynamic observation. So a pathologist can't diagnose PPHN because it's a living diagnosis. So what the pathologist has diagnosed is the vascular remodelling that you see in babies with PPHN. So that's why I refer to this as being a little bit of a semantic argument – um – but I think that what you could – you know, what you might reasonably put there is pulmonary vascular remodelling – ah – which is strongly associated with PPHN – um – and, you know, but hospitals – they won't have facilities at Redcliffe to do – um – cardiac ultrasound at that time and nor would you expect there to be those facilities in a hospital like Redcliffe. Um – ah – we're left speculating as to what that status was on the basis of the pathological observation."

59. The evidence satisfies me that the underlying cause of death was transposition of the great vessels, as I have said. I am satisfied that as the cause of death it was compounded by poorer oxygenation of the blood than most other babies born with that condition would experience, in the first few hours of life, because Thea had reduced shunting and possibly PPH. I prefer Dr Colen's evidence that idiosyncratic reduced shunting was the major problem, in all likelihood, if not the only complicating factor. PPH was possibly present however I am not satisfied to the sufficient degree to conclude that it should be included on the death certificate as another disease or condition in Thea, though if there was not reduced shunting as described, then an inference in favour of its presence would be drawn as explaining the clinical course. Not including PPH separately as another disease or condition would also be justified if it is properly regarded as a feature of TGA in Thea's case, rather than being coincidental, however I do not need to decide that controversy.

[56] T3-8, LL 36-44.
[57] Ex D1.5, p 62.
[58] T4-33, LL 31-42.

60. The death certificate should be amended to say:

> 1(a) Main disease or condition in neonate: Transposition of the great vessels.
>
> 1(b) Other diseases or conditions in neonate: Nil.

Consideration of issues 2 and 3: Should TGA have been detected and if so, what would then have happened and would Thea's death have been avoided

61. It is convenient to deal with these issues together, as Counsel Assisting submitted.

TGA detection and management

Detection and diagnosis

62. According to the Queensland Maternal and Perinatal Quality Council, and as cited by Professor Weaver in his first report, congenital heart defects (including TGA) have an incidence rate of between four to ten live births per 100. In Queensland, congenital heart defect cases account for more perinatal deaths than any other congenital abnormality and babies born with critical defects require urgent and complex neonatal care.[59]

63. Congenital heart defect conditions, including TGA, can only be diagnosed antenatally by fetal ultrasound. Ideally, this diagnosis would occur as a result of the 20-week morphology scan ('the morphology scan').

64. A morphology scan takes around 60 minutes, including when a trainee sonographer is involved.[60] It is a routine antenatal screening test performed to check a baby's growth, to review the early development of the fetal organs, determine the placental location, cervical length, the appearance of the umbilical cord and the amniotic fluid volume and it allows for estimates as to the baby's age and sex. It also assesses the heart rate and rhythm and attempts to screen for congenital heart disease.[61]

65. In performing a morphology scan, sonographers are expected to adhere to the Australian Society for Ultrasound in Medicine guidelines ('ASUM guidelines'). These guidelines,[62] which have evolved over time, outline the clinical indications and procedures for undertaking standard morphology

[59] Ex B1.4, p 6.
[60] T1-14, LL 46-49; T1-28, LL 18-20.
[61] Ex C1.11, [3.1] – [3.5].
[62] As attached to C1.9 at p 32 (Annexure F).

scans. They apply to all ultrasound practitioners and, amongst other things, list the minimum imaging and reporting requirements.[63]

66. For a baby's heart and vessels, the minimum examinations required are heart motion, position, axis, four chambers, intraventricular septum, foramen ovale, mitral ovale, tricuspid valve, left and right ventricular outflow tract, aortic arch or three vessel view and the ductus arteriosus.[64]

67. Normal fetal hearts show four chambers with left and right ventricles, as well as an intact septum without any holes. Indicators on morphology for TGA include the presence of only two vessels while taking the three-vessel view and the absence of an arrowhead, when an arrowhead appearance should not be possible with the presence of only two vessels or with parallel outflow tracts seen in TGA.[65]

68. As explained by Dr Nicole Woodrow, obstetric and gynaecological ultrasound specialist, the morphology scan aims to "tick off" normality - it is a screening scan not a diagnostic scan.[66] Diagnostic scans are performed when a condition or conditions are suspected and are in turn specifically looked for. Screening scans are conducted on the relevant population, here pregnant women, where no particular condition is suspected and the scan aims to establish that none of the many signs that might possibly be present for a large variety of conditions, are observed. The specificity of the former examination logically speaks to the higher risk in the latter examination of some pathology actually present not being detected.

69. Mr Collins explained how a morphology scan of the fetal heart is performed:[67]

> "…so normally I would have a look at the heart, and we start in tr – like, in transverse, or across the baby, and you scan up and down and – and check that the baby's got a heartbeat. That would be the first thing that you want to do. Um – that the – the heart has four chambers – um – that it's in the right place, it's the right orientation, that the aorta is in the correct position, that we don't have any big holes in the heart, the – um – usually use – um – Colour Doppler, and that – that's – that indicates flow in the baby. So we'll look at the direction of the flow, whether that's appropriate. And then by scanning up and down, we can look at the larger vessels on the top of the heart and see where they come out, whether they're in the
> correct position, and that they go – they go in the right directions."

[63] See pp 5-7.
[64] See pp 6-7.
[65] Ex C1.11, [3.41].
[66] T6-29, LL 33-45.
[67] T1-28, LL 42-49 to T1-29, LL1-3.

70. Where there is doubt about the presence of a normal structure, or concerns about cardiac structural abnormalities, held by either the sonographer or the reporting radiologist, further imaging along with a referral to a materno-fetal medicine specialist ('MFM') or paediatric cardiologist at a tertiary facility is required. The process then transitions from one of screening (as is the case with the morphology scan) to diagnostic.[68] Again, a diagnostic scan is where some particular condition is suspected and is looked for. In the context of a morphology scan, the description of it as a screening scan reflects that it is one undertaken where no particular condition is suspected but many different abnormalities reflecting many different underlying conditions are looked for.[69]

71. A fetal cardiology review is then undertaken with a view to diagnosis. This typically involves scans late in pregnancy to assess the size of the atrial septal defect[70] and to determine the risk of early balloon septostomy. Dr Colen, who is a specialist in this area, notes that even with this specialist intervention, diagnostic "prediction" is not 100% accurate.[71]

72. According to Dr Woodrow, no matter how good the scan is, there is nothing in a morphology scan that can completely rule out abnormal outflow tract morphology - there is always the possibility that there will be an undetected congenital heart defect.[72]

Management of diagnosed TGA

73. If a diagnosis of TGA is made or confirmed by or following fetal echocardiography, care is transferred to a suitable tertiary centre and arrangements regarding delivery location and timing are made.[73] Collaborative management between the paediatric cardiologist, MFM and neonatal teams then occurs.[74]

74. In Queensland, the baby would be delivered at the Mater Mothers Hospital or Royal Brisbane and Women's Hospital. [75] In TGA cases without high-risk, the baby is born in the presence of a neonatal team with a cardiology team on standby. Within 15 minutes of being born, an echocardiogram is performed to confirm anatomy, assess the size of the atrial septal defect/foramen ovale, and check the baby's oxygen levels.[76]

[68] T1-53, LL 21-25.
[69] T1-53.
[70] If the hole is very small after the baby has been born, the baby can be very unwell (TT5-9, LL 3-4).
[71] Ex B1.7, p 4.
[72] T6-36, LL 37-44.
[73] Ex B1.4, p 7.
[74] Ex B1.8, p 10.
[75] Ex B1.8, p 10.
[76] T5-12, LL 23-28.

75. If during the latter part of the third trimester, there is "echocardiographic evidence of restrictive flow across the foramen ovale and/or the ductus arteriosus and/or echocardiographic evidence of pulmonary vascular disease that could portend high risk for pulmonary hypertension", the assessment will be of "high risk" and a caesarean section will be recommended.[77]

76. If emergency intervention is required in any case of TGA, a balloon atrial septostomy can be performed. As explained by Associate Professor Evans, this involves:[78]

> "…passing a catheter with a balloon on the end through the umbilical vein and then through the foramen ovale. Once through the balloon is blown up and pulled sharply back through the foramen ovale such that it rips open a connection between the two atria. This will usually provide enough oxygenation to sustain the baby until the definitive corrective arterial switch operation can be performed."

77. Dr Luig, who manages all antenatally diagnosed cases of TGA at the Westmead Hospital, says that where intervention is required, a septostomy will be necessary on day one. An infusion of Alprostadil to prevent the closure of the ductus will also be required. Importantly, these are only temporising measures prior to surgery[79] (being surgery to correct the transposition[80]). Septostomy is only performed in major tertiary hospitals.

78. If the baby is not responding well to the septostomy and/or other forms of resuscitation are not working, the baby can be placed on extra-corporal membrane oxygenation ('ECMO'). This is a lifesaving treatment which involves the baby being placed on a heart-lung bypass machine.[81] ECMO is only available in major tertiary hospitals.

<u>TGA detection rates</u>

79. According to the 2016 – 2017 Qld Maternal and Perinatal Quality Council report, between 2007 and 2015, more than 60% of critical congenital heart defect cases were missed during fetal ultrasound screening.[82]

80. In respect of TGA specifically, there is divergence within the literature concerning detection rates. Some studies suggest that, internationally, antenatal detection rates for TGA are less than 50%.[83] Dr Colen, however,

[77] Ex B1.8, p 11.
[78] Ex B1.6, [54].
[79] Ex B1.8, pp 10-11.
[80] T5-13, LL 25-26.
[81] T5-12, LL 38-40.
[82] Ex B1.4, p 6.
[83] Ex B1.6, [34]; T4-21, LL 1- 3; Ex B1.5, p 4; Ex B1.3, p 5; Ex B1.5, p 4.

cited recent Queensland data which showed that between 2008 and 2017, there was a 64% overall antenatal diagnosis rate in Queensland.[84]

81. Irrespective of this variance in percentages, it became very clear during the coronial investigation and inquest that TGA detection rates are sub-optimal, despite advancements in technology. Whichever of the data sets is looked at, a large percentage of affected babies are not identified before birth.

82. Several of the experts offered opinions as to why this is the case.

83. Associate Professor Evans said that where a baby has TGA, it is more likely than not that it will not be detected on antenatal scanning. I have already noted the variable data as to that. Tempering his opinion to say that as many as third are not detected is not of great moment. He explained that the anatomy of the heart is unusually complex and difficult to examine with ultrasound (in the *in utero* context). It requires prolonged and specialised training and relies on reason for suspicion appearing on the imaging.[85]

84. Specific issues arise with respect to the scanning of the heart's four chambers. Associate Professor Evans gave evidence that in TGA cases, the four-chamber scan can appear normal, despite there being an abnormality.[86]

85. Professor Weaver provided several possible reasons for low detection rates. These included difficulties associated with the positioning of the baby at the time of the scan, the fact that a baby's vessels can be quite small, issues created by bone shadowing and the low incidence rate. He noted that even where the required views of the baby's heart are obtained, congenital heart conditions can still be missed.[87]

86. Dr Colen gave evidence that even some of the very experienced sonographers he has worked with can struggle to obtain ideal images due to the positioning of the baby. He observed that the placenta, spine, arms and bones can contribute in this regard.[88]

87. Dr Woodrow's evidence on this topic was particularly enlightening. She said that during morphology scanning of the baby's heart, the apparent structural anatomy can be deceiving – three vessels can be imitated despite only two being present.[89]

[84] Ex B1.7, p 4.
[85] Ex B1.6, [32], [34].
[86] T4-23, LL 24-30.
[87] T4-4, LL 36-46; T4-5, LL 21-23.
[88] T5-5, LL 8-17.
[89] T6-31, LL39 – 47.

88. Dr Stephen Sinnott, who was the reporting radiologist for Mrs Flaskett's morphology scan, provided similar observations – noting that structures can appear normal despite TGA being present.[90]

89. Ms Leddy and Mr Collins also spoke of the difficulties and limitations associated with morphology scans. From their perspectives, speaking of cases generally, this includes maternal habitus, the positioning of the baby and placenta,[91] the inability of the ultrasound equipment to show anything "under two millimetres",[92] and the fact that the images generated show only a fraction of the full examination that had been performed in real time.[93]

90. Having regard to the opinions of the independent experts and the combined experience of the sonographers and radiologist, clearly, ultrasound scanning as a means of detecting TGA antenatally is not a perfect test[94] and TGA is not uncommonly missed.[95]

Survival rates

91. According to Dr Colen, where TGA is diagnosed prenatally, there is an estimated 90 to 95% chance of survival through the prenatal period and following surgical repair. Most of the risk lies with the surgery, and to that end, surgical mortality is between 2 and 5%.[96]

92. However, even where TGA has been diagnosed antenatally, additional risk factors can appear after birth which increase the risk of poor outcomes.[97] Such risks can include a small atrial defect, PPH, early gestational age and small size.[98]

93. When giving evidence about survival rates in TGA cases, Dr Luig recalled that in the preceding five years (in New South Wales), two babies, each of whom had been diagnosed antenatally, and who were not determined to be high-risk, did not survive.[99]

Thea's case

Morphology scan

94. Mrs Flaskett presented to the Redcliffe Hospital Imaging Department on 5 May 2023 for a 20-week morphology scan. The scan was performed by Ms Leddy, a trainee sonographer, and her supervisor, Mr Collins.

[90] T1-60, LL 4-11.
[91] T1-14, LL 34-40;
[92] T1-27, LL 43-49.
[93] T1-28, LL 3-5.
[94] Ex B1.6, [31] – [32]; T1-60, LL 9-10.
[95] T1-59, LL1-2.
[96] Ex B1.7, p 5; T 5-13, LL 10-23.
[97] T5-13, LL 28-32.
[98] Ex B1.7, p 5.
[99] T3-29, LL 31-45.

95. As at May 2023, Ms Leddy was undertaking training and clinical placement at the Redcliffe Hospital as a trainee sonographer. She was in her third semester of a two year post graduate diploma in medical ultrasound. She had worked as a senior radiographer since 2012[100] and became qualified as a sonographer in 2024.[101]

96. Ms Leddy's supervisor, Mr Collins, had been a radiographer for more than 37 years and a sonographer for 19 years.[102] At the time, he had been working at the Redcliffe Hospital as a radiographer/sonographer since 2007. He had also been employed as a guest lecturer at the Queensland University of Technology for the postgraduate ultrasound course.[103]

97. The supervisor/trainee process for morphology scans required both the trainee and supervisor to scan the patient. Mr Collins would observe Ms Leddy conduct an initial scan and take the required images. Where any images could not be obtained by Ms Leddy, or where Mr Collins was not satisfied with any images produced by Ms Leddy, he would rescan the patient and retake any images as required.[104] This was the process followed during Mrs Flaskett's scan on 5 May 2023.[105]

98. The equipment used for the morphology scan was a Philips Epiq machine. Both sonographers had worked almost exclusively with that machine and had no concerns as to its quality.[106]

99. At the time of Mrs Flaskett's scan, sonographers at the Redcliffe Hospital were required to adhere to the ASUM guidelines (discussed above) and the MNHHS morphology ultrasound scan guide.[107]

100. In his statement, Mr Collins explained that the MNHHS guide was based on additional imaging requirements requested by Specialised Obstetric and Gynaecological Imaging ('SOGI') – the obstetric reporting provider for Redcliffe Hospital. SOGI had also supplied the hospital with a second-trimester checklist.[108]

101. SOGI's requirements for the morphology scan exceeded those prescribed by the ASUM guidelines. In addition to the minimum views required by the guidelines, SOGI's protocol required cinematic sweeps of the heart in B-mode and in colour, and colour documentation of outflow tracts and the corpus callosum.[109]

[100] Ex C1.10, [4]-[5] and [8].
[101] T1-13, LL 24-25.
[102] T1-26, L 22.
[103] Ex C1.9, [2].
[104] Ex C1.10, [33]; T1-17, LL 23-32; Ex C1.9, [4](e); T1-29, LL 9-32.
[105] Ex C1.9, [4](e); T1-17, LL 43-44; Ex C1.10, [42] and [46]; T1-31, LL 8-11.
[106] Ex C1.10, [16]; T1-21, LL 34-38; Ex C1.9, [5]-[6]; T1-30, LL 44- 49; T1-38, LL 39- 46.
[107] Attachment D to Ex C1.9.
[108] Ex C1.9, [8] and attachment E.
[109] Ex C1.10, [21]; T1-29, LL 11-26.

102. Once all images are taken, a worksheet containing the sonographer's impressions is produced and completed by one of the sonographers. The worksheet, biometry graphs, still images and cinematic loops would then be sent to SOGI for review and reporting by the radiologist. If the radiologist was unsatisfied with the quality of the examination or imaging, a formal report requesting further imaging and a recalling of the patient would be sent back to the sonographers.[110]

103. Neither Ms Leddy nor Mr Collins can recall who completed the worksheet on 5 May 2023. In any event, they were both satisfied as to the quality of the imaging produced and the absence of any fetal abnormalities. As such, the worksheet contained the following summary of findings: [111]

> "The anatomy has been checked in detail and no major structural problem has been identified. The normal ultrasound findings today are reassuring but no scan can confirm complete normality of the fetus."

104. Dr Stephen Sinnott, Director of SOGI, was the reporting radiologist. He has been a subspecialised radiologist in obstetrics and gynaecology since 1999. Prior to SOGI, he had been the Director of Medical Imaging at Royal Women's Hospital. He also co-founded the Centre for Fetal Diagnosis and Treatment.[112]

105. Dr Sinnott explained in evidence his usual practice in his role as the reporting radiologist in morphology scans. His evidence in this respect was uncontroversial and there is no reason why I would not accept it.

106. On receiving the sonography material, Dr Sinnott reviews the clinical information provided by the referring doctor, the sonographer's interpretation as documented on the worksheet and the videos and images. [113] He does not rely on the worksheet,[114] and even if nothing is drawn to Dr Sinnott's attention by the sonographers, he stills looks for everything.[115] Following his assessment, he prepares a report and sends it to the hospital and any other necessary practitioners.[116]

107. Where Dr Sinnott has any concerns, he would usually contact the obstetric registrar at the hospital to discuss his findings and recommend an appropriate course of management. In cases where TGA is suspected and where he was highly suspicious, he would recommend a referral to a MFM department. Otherwise, he would request rescanning.[117] That rescanning

[110] Ex C1.10, [30], [48]; Ex C1.9, [4](e).
[111] See Ex D1.8.
[112] Ex C1.11, [2.1]; T1-52, LL 40-41.
[113] Ex C1.11, [3.10].
[114] Ex C1.11, [4.5].
[115] T1-56, LL 11-12.
[116] Ex C1.11, [3.10].
[117] T1-56, LL 35-48.

would be focused on the particular abnormality he was concerned about.[118]

108. Dr Sinnott considered that Thea's imaging accorded with the ASUM guidelines and SOGI's additional requirements. In his opinion, the imaging revealed appropriate heart development and the absence of abnormality, three vessels (being the superior vena cava, aorta, and pulmonary artery), the presence of an arrowhead and the absence of any ventricular septal defect.[119]

109. He did not detect TGA. His evidence was that:[120]

> "The presence of four chambers and three vessels combined with the presence of an arrowhead, and the absence of any obvious holes in the septum, caused me to conclude the imaging was consistent with normal heart development."

110. Under cross-examination, Dr Sinnott said that at the time of reviewing Thea's imaging, he saw no issues that prompted him to seek rescanning.[121]

Post-death reviews of the images

111. Ms Leddy, Mr Collins and Dr Sinnott each reviewed Thea's morphology scan imaging following Thea's death. This exercise was, of course, undertaken with the benefit of hindsight and the knowledge that Thea did in fact have TGA.

112. Ms Leddy, who was taken through a series of images during her evidence,[122] maintained that in circumstances where she still considered the three vessels and arrowhead were visible, none of the imaging revealed any indicators of TGA.[123]

113. Mr Collins, who was also taken through the images, said that even with the benefit of hindsight, the three-vessel view (which he could still see) indicated the absence of any heart abnormalities.[124] He was able to identify and mark the apparent three vessels in the relevant images for the court.[125]

[118] T1-57, LL 43-47.
[119] Ex C1.11, [4.11] – [4.44].
[120] Ex C1.11, [4.48].
[121] T1-69, LL 40-46; T1-71, 40-41.
[122] Being Ex D1.4, images 50, 51, 53, 71, 72, 73.
[123] T1-20, LL 17-22' T1-21 LL 15-29.
[124] T1-35, LL 4-7; T1-35, LL 22-23.
[125] T1-46 to 1-48.

114. Mr Collins could not explain how three vessels and an arrowhead could be visible in circumstances where Thea had TGA. He ultimately attributed it to ultrasound screening for TGA detection being imperfect.[126]

115. When giving evidence about his review of the imaging, Dr Sinnott maintained his original conclusion that the arrowhead appeared normal and there appeared to be three vessels.[127] The imaging was therefore consistent with normal heart development.[128]

116. Although Dr Sinnott identified some "suboptimal" images insofar as clarity and colouring was concerned, he clarified that this had no bearing on what appeared to be the depiction of an arrowhead and three vessels.[129]

117. Dr Woodrow was briefed by the court to review and comment on Mrs Flaskett's antenatal ultrasound examinations. In relation to the morphology scan on 5 May 2023, she opined that the fetal heart imaging was performed appropriately and to a satisfactory standard by a competent ultrasound operator. The machine technology was appropriate, the fetal structures were "visualised in a methodical approach according to ASUM guidelines", and a thorough attempt had been made to obtain a comprehensive evaluation of Thea's heart.[130]

118. Concerning the three vessel view images, Dr Woodrow said that the images attempted to demonstrate that view. She went on to say that despite some of the views being oblique and difficult to interpret, she could not see any definite abnormality.[131]

119. In her report, Dr Woodrow concluded as follows:[132]

> "The cardiac views at the routine morphology screening scan do not demonstrate any definite fetal abnormality. Unfortunately, transposition of the great vessels is not visible in the images I have reviewed."

120. During her oral evidence, Dr Woodrow was asked by counsel assisting whether she was able to explain why, when Thea did in fact have TGA, it was not detected. Dr Woodrow said:[133]

> "What I think is happening, theoretically, possibly, is that third vessel is probably either the ductus or the pulmonary and is coming in the middle of the aorta and the SVC. It's partially seen, and it's got to do with the

[126] T1-50, LL 4-15.
[127] T1-59, LL 33-45.
[128] T1-61, LL 11-16.
[129] T1-61 to 1-62.
[130] Ex B1.3, pp 11-12.
[131] Ex B1.3, p 11.
[132] Ex B1.3, p 5.
[133] T6-31, LL39 – 47.

slightest of angles in that oblique view. So it appears to be three vessels. What else could it be? Well, it could be a part of the – ah – one of the – ah – atria. It could be a small amount of fluid. I don't know what it was. But I wonder when this happened – it's got to do with our sweeps coming up from the four-vessel – four-chamber view and coming onto that three-vessel view. And then, unfortunately, it imitates falsely three vessels when there are, in fact, just two."

121. As to Mrs Flaskett's other antenatal scans, at the time of providing her report, Dr Woodrow had not seen the nuchal translucency scan report or imaging. That scan occurs at 11 to 13 weeks gestation.[134] Dr Woodrow was asked to review the images for completeness.

122. Having had the benefit of reviewing the nuchal translucency scan report and imaging prior to giving oral evidence, Dr Woodrow's opinion at the inquest was that the nuchal translucency measurement was one that indicated low risk for heart defects. Also, that the cardiac imaging showed a normal fetal heart.[135] These factors meant that it was appropriate for Mrs Flaskett's pregnancy to continue with "the usual antenatal care". Further scanning was not required until the mid-trimester morphology scan.[136]

123. Dr Woodrow also had no concerns with respect to the 14-week scan on 27 March 2023 or the third trimester ultrasound on 28 August 2023. In relation to the latter, she noted that "It is not expected at a third trimester study to obtain comprehensive views of the fetal anatomy as per ASUM guidelines due to a large gravid uterus, fetal position and shadowing from bone."[137]

124. Professor Weaver was asked to comment on Dr Woodrow's report. He agreed with Dr Woodrow's conclusions regarding the first and third trimester scans.[138]

125. As to the morphology scan, he observed that congenital abnormalities are missed during antenatal investigations for a variety of reasons, despite a practitioner's best intentions. He commented that given there was no suspicion during either the morphology scan or any subsequent scans that anything was wrong with Thea's heart, there was no indication to conduct further scans or refer Mrs Flaskett's care.[139]

Conclusions

<u>Should TGA have been detected?</u>

[134] Ex B1.3, p 8.
[135] Dr Woodrow did, however, explain that it is rare to identify TGA at 13 weeks' gestation because the three vessels appear present and can falsely imitate "what is going on" (see T6-27, LL 30-44).
[136] T6-24 to T6-27.
[137] Ex B1.3, p 5.
[138] Ex B1.5, p 4.
[139] Ex B1.4, p 8.

126. A finding that TGA *should* have been detected is not reasonably supported by the evidence. The literature cited by the expert witnesses and the evidence given during the inquest in that regard, supports a conclusion that antenatal detection rates for TGA are low, and missed detection, such as in Thea's case, is not uncommon, at least as a proportion of the cases in which TGA occurs.

127. That it was missed here was through no fault of the sonographers or radiologist involved in Thea's care. All were appropriately experienced and qualified, the scanning was done in a conventional way and adhered to the prescribed methods, protocols and guidelines, and the imaging was of such quality that rescanning was not required.

128. By 5 May 2023, both Ms Leddy and Mr Collins had extensive experience in performing morphology scans. Ms Leddy estimated that she had completed between 90 and 100 morphology scans (under supervision).[140] Mr Collins estimated that he would do around 150 morphology scans a year[141] – meaning by May 2023, he would have performed approximately 2,800 scans.

129. At the time of giving evidence at the inquest, Thea's case of TGA was the only case Ms Leddy had been involved in and was aware of. Mr Collins gave evidence that in his 20 years of experience, there was only one occasion in which he suspected a fetal heart abnormality that was subsequently diagnosed TGA. This occurred early in his career, prior to Thea.[142]

130. Dr Sinnott undertakes around 15 to 20 morphology scan reviews each working day and, prior to Thea's case, had detected TGA "a number of times".[143] As such, he was well-placed to identify TGA.

131. The failure to detect Thea's TGA was, I find, very clearly a consequence of the known limitations of antenatal screening. Here, the limitation lay in the observation of what appeared to be three vessels and an arrowhead. Neither Mr Collins nor Dr Sinnott could explain how those structures could appear normal when Thea had TGA. As commented by Dr Sinnott, "it doesn't make sense."[144]

132. Significantly, it was not only the sonographers and radiologist who could see what appeared to be three vessels and an arrowhead. As discussed above, even Dr Woodrow thought there appeared to be a three-vessel view.

133. No criticism is apt to be made of Ms Leddy, Mr Collins, or Dr Sinnott for failing to detect Thea's TGA.

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[140] Ex C1.10, [6].
[141] T1-26, LL 27-28.
[142] T1-26, LL 32-36.
[143] T1-57, LL 4-16.
[144] T1-62, LL 18-19.

If TGA had been detected, would Thea's death have been avoidable?

134. If TGA had been detected antenatally and the appropriate course of management was provided, there was no guarantee that Thea's death would have been avoided.

135. Dr Colen observed that undiagnosed TGA in isolation does not inevitably cause death and prenatal diagnosis would "not entirely have prevented her death", which I take to mean that survival would not have been ensured. He did add that the risk would have reduced significantly. Thea very likely[145] had additional high-risk factors that increased her risk and are difficult to treat. Those high-risk factors are not always discernible before delivery. He said that even in a high-level centre, those features may be associated with mortality. In his opinion, Thea's death was not preventable[146], though that observation responded to a part of Professor Evan's report that commented upon Thea's chances of survival, given she was born with an undiagnosed TGA at Redcliffe hospital, in circumstances where only an immediate septostomy could have saved her.

136. As opined by Dr Luig, for Thea to have survived, at a minimum she would have required:[147]

> "1. Diagnosis of the TGA with intact ventricular septum during the pregnancy and Meg's referral for delivery at a tertiary maternity centre near a childrens hospital, 2. Delivery occurring during the day time on a weekday at the tertiary centre with an interventional cardiology team ready at the time of birth to perform a balloon atrial septostomy, in addition to a neonatal intensive care team to provide the supportive care."

137. Thea's rapid deterioration, however, would have occurred irrespective of where she was born and no matter the obstetric and delivery circumstances. A septostomy and Alprostadil infusion would have provided partial benefits only – they may not have adequately improved her cardio-respiratory instability. Urgent "heroic" procedures, such as a ECMO, would very likely have been required.[148] Septostomy and ECMO were not available at Redcliffe Hospital, it not being a major tertiary hospital.

138. The only chance Thea had of surviving would have been if her TGA had been detected as a result of the morphology scanning. She would then have been referred to a tertiary hospital. There her best chance of survival would have been if further sub-specialist ultrasound had accurately detected low flow through the septum, as discussed by Dr Colen. It may or may not have done so. If it did, she would have been categorised as high risk, resulting in delivery being done by caesarean section, with the

[145] Ex B1.7, p 6.
[146] Ex B1.7, p 5.
[147] Ex B1.8, p 11.
[148] Ex B1.8, p 11.

full suite of emergency cares immediately available. In the total patient cohort, there is about a 95% chance of survival, however the presence of reduced shunting and/or PPH would have meant Thea would have been at a higher risk of being in the 5%. If she wasn't assessed as being high risk, the chances of being in the 5% would have been greater.

139. All that said, it is probable Thea would have survived, had her TGA been detected antenatally. Having been born at Redcliffe Hospital, however, in the circumstances she was, she had no chance of survival. My conclusion in that regard, of itself, involves no criticism of that hospital or its staff.

Consideration of issue 4: Post-delivery care

Thea's birth

140. At 7:30pm on 9 September 2023, Mrs Flaskett underwent an induction of labour at the Redcliffe Hospital by balloon catheter due to complaints of decreased fetal movements.[149] She was 38 weeks and four days gestation.

141. At 4:50pm on 10 September 2023, Mrs Flaskett was transferred to the birth suite. At around 11:30pm, Consultant Obstetrician and Gynaecologist, Dr Mamta Vyas, was contacted to review Mrs Flaskett in the birth suite. She arrived about five minutes later.[150] Paediatric Registrar, Dr James Kelly, also attended.[151]

142. On arrival, Dr Vyas reviewed the CTG and performed an abdominal and vaginal examination. She determined that the CTG had complicated variable decelerations and so Thea's delivery needed to be expedited.[152]

143. Having assessed the position of Thea's head, Dr Vyas considered that an assisted vaginal birth, by way of vacuum extraction, was the safest option.[153] She encouraged Mrs Flaskett to push with contractions and conducted "three pull vacuum delivery episiotomy".[154]

144. Thea was born at 12:07am on 11 September 2023.[155]

145. At the inquest, the court heard from Professor Weaver that a vacuum delivery was reasonable in the circumstances. He had no concerns with the decision or the way in which the vacuum extraction was performed.[156] Nothing during this period of care was outside what would normally be expected.[157]

[149] Ex C1.12, [13].
[150] Ex C1.12, [13].
[151] Ex C1.17, [20].
[152] Ex C1.12, [16].
[153] Ex C1.12, [18].
[154] Ex C1.12, [22].
[155] Ex D1.5, p 7.
[156] T4-6 to T4-7.
[157] Ex B1.4, p 7.

Thea's condition at birth

146. There were discrepancies in the APGAR scores recorded retrospectively at one and five minutes of Thea's life. To this end, Dr Kelly assigned scores of 9 at one minute and 8 at five minutes, whereas a midwife assigned scores of 7 and 7.[158]

147. In his statement, Dr Kelly explained that he based his one-minute score of 9 on his assessment of Thea when she came to the resuscitaire after five minutes of life:[159]

> "At this point she was scoring 1 for Respiratory Effort (the reason for her coming to the resuscitaire for assessment), but her initial heart rate at this time was still greater than 100, which scores a 2. If her heart rate had been below 100 at 1 minute of life with respiratory distress, this would not have improved without intervention, these scores would not be consistent with normal scores for tone and reflex irritability."

148. When asked about this in oral evidence, Dr Kelly clarified that his score of 9 at one minute was based on Thea having cried and being placed on Mrs Flaskett's chest.[160] His score of 8 at five minutes was prompted by Thea grunting – which is an indicator of respiratory distress.[161]

149. Dr Kelly could not explain how the midwife reached a score of 7 at one minute. He postulated it was because resuscitation was unsuccessful and the score was based on the outcome. He disagreed with the midwife's score in that regard, noting that a score of that level at one minute would have necessitated earlier resuscitation.[162]

150. In any event, the witness observations of Thea's condition at birth suggest that she was born in a reasonable, if not good, condition. I do accept the evidence of Dr Kelly that Thea's heartrate at 1 minute was likely greater than 100, given that it was when assessed at 5 minutes and there had been no intervention in between.

151. Dr Vyas recalled Thea being pink in colour. She placed Thea on Mrs Flaskett's abdomen and clamped and cut the cord one-minute post-birth.[163] According to Dr Kelly, where a baby cries and has good colour, the preferred practice, as occurred here, is to leave the baby on the mother's chest for skin-to-skin contact and delay cord clamping until one minute post-delivery.[164]

152. Dr Kelly recalled Thea crying immediately, developing good colour, and breathing. He described her as being "active" and had no concerns at that

[158] Ex D1.5; Ex C1.17, [24] – [25].
[159] Ex C1.17, [26].
[160] T6-7, LL 6-8.
[161] T6-6, LL 13-16.
[162] T6-10.
[163] Ex C1.12, [22] – [23].
[164] Ex C1.17, [21].

time.[165] When cross-examined by the legal representatives for the family, he disagreed that Thea was born blue rather than pink and also, when she was placed on Mrs Flaskett's chest, she was floppy.[166]

153. Dr Tshamala's evidence was that Thea's placement on Mrs Flaskett's chest and her delayed cord clamping meant Thea was in good condition. He said that if this was not the case, Thea would not have been given to Mrs Flaskett.[167]

154. Dr Luig, although not present for Thea's birth, was shown a photograph of Thea[168] at two minutes of age. She gave evidence at the inquest that the photograph supported the conclusion that Thea was born in a reasonable or good condition:[169]

> "It does. I can see her eyes are open. Her colour, to me, at that time, is like other babies that I leave with their mothers."

155. Professor Weaver also had regard to the photograph. He made the observations that Thea had started to "pink up" and "look like…she's making a…reasonable adaption to outside life".[170]

156. Associate Professor Evans' evidence was that the records of Thea breathing and making noises after birth indicate that she had respiratory effort. Whether or not she cried was of less importance to the assessment of her condition, in his view – it is the respiratory effort that is the focus.[171]

Thea's deterioration

157. Around 5 minutes of age, while Thea was on Mrs Flaskett's chest, she was observed to be grunting while breathing and developing respiratory distress.[172] Thea was moved to the resuscitaire where she was assessed by Dr Kelly. Her heart rate at that stage was greater than 100 beats per minute ('bpm') and her oxygen saturation reading was 78%. Continuous positive airway pressure ('CPAP') was commenced.[173]

158. At 15 minutes of age, Thea was observed to be pink with reduced tone. Her heart rate was between 60 and 100 bpm and it continued to fall despite positive pressure ventilation.[174] The treating team were also experiencing difficulties in obtaining accurate oxygen saturation readings (an alternative perspective is they were accurate but were low beyond

[165] Ex C1.17, [22] – [23]; T6-5.
[166] T6-12, LL 26-31.
[167] T2-8, LL 16-23.
[168] On display during the inquest at the request of the family.
[169] T3-42, LL 16-19.
[170] T4-9, LL 17-19.
[171] T4-31, LL 21-41.
[172] Ex C1.17, [27]; Ex C1.2, pp 2-3; Ex D1.5, p 28.
[173] Ex C1.17, [29]; Ex B1.6, [7]; Ex D1.5, p 28.
[174] Ex C1.17, [34]; Ex B1.6, [7]; Ex D1.5, p 28.

expectations). Intermittent positive pressure ventilation ('IPPV') commenced.[175]

159. Due to Thea's ongoing deterioration despite ventilation and increased oxygen support, on-call Consultant Paediatrician and Director of Paediatrics at Redcliffe Hospital, Dr Didier Tshamala, was contacted to attend.[176] This occurred at approximately 12:30am.

160. The call to Dr Tshamala was made by a nurse, who put the phone to Dr Kelly's ear. As Dr Kelly explained in his statement that he provided Dr Tshamala with a "brief history which included onset of respiratory distress at 5 minutes of life, hypoxia and falling heart rate despite escalating respiratory support, and anticipation of cardiac arrest, and requested that he attend the hospital".[177] Dr Kelly confirmed during oral evidence that he was continuing to provide interventions to Thea whilst on the phone.[178]

161. When giving oral evidence about this phone call, Dr Tshamala said that the situation was obviously fairly urgent and so he did not want to waste time questioning or asking for details. He was given enough information to know that there existed an emergency situation requiring his immediate attendance. He told Dr Kelly to "Keep going with the resuscitation. I'll be there soon."[179]

162. During the phone call between Dr Kelly and Dr Tshamala, Thea's heart rate decreased to below 60 bpm, causing CPR to be commenced.[180] It became clear to Dr Kelly, during the CPR, that Thea would require retrieval to a tertiary level facility as well as tertiary level investigation into the potential cause for her deterioration and cardiac arrest.[181]

163. At 12:31am, a neonatal resus code '666' was called and Dr Kelly asked for the neonatal retrieval team, NeoRESQ, to be contacted.[182] NeoRESQ provides a service whereby babies are retrieved from non-tertiary hospitals in Queensland and transported to a tertiary hospital. Advice services are also provided.[183]

164. Dr Kelly did not request input from NeoRESQ because he was occupied with preparing for intubation and intravenous access.[184] He was focussed on resuscitation and considered he had the requisite skills and knowledge to follow the neo-resuscitation algorithm.[185]

[175] Ex C1.2, p 3.
[176] Ex D1.5, p 28; Ex C1.2, p 3; Ex C1.17, [36] T 6-7, LL 21-32.
[177] Ex C1.17, [37].
[178] T6-7, LL 34-42.
[179] T2-5.
[180] Ex C1.2, p 3.
[181] Ex C1.17, [40].
[182] Ex D1.5, p 28; Ex C1.17, [39].
[183] T2-32.
[184] Ex C1.17, [42]; Ex C1.2, p 7.
[185] T6-9, LL 31-39.

Initial resuscitation efforts

165. While waiting for Dr Tshamala to arrive, Dr Kelly led the resuscitation efforts. He successfully intubated Thea with an endotracheal tube at 12:36am, following which he attempted to establish intravenous access.[186] At this time, Thea was displaying poor to minimal respiratory effort.[187]

166. Dr Kelly was unable to establish either peripheral or umbilical intravenous access. As such, he administered adrenaline through the endotracheal tube. This occurred at 12:43am and resulted in Thea's heart rate increasing to 60 bpm as well as spontaneous respiratory activity.[188]

167. Between 12:45am and 12:47am, CPR and ventilation continued and one milligram of Konakion was administered. Thea's heart rate increased to 120 bpm.[189]

168. At 12:48am, nurse Claire Parry spoke to 'Carly' at Retrieval Services Queensland ('RSQ'). Ms Parry had not been present during Thea's birth and so had limited information she could provide to Carly. This is evident from the audio recording of that conversation.[190]

169. At 12:49am, Carly contacted Dr Pieter Koorts, the on-call coordinating neonatologist for NeoRESQ. She provided Dr Koorts with the limited information she had and then connected Dr Koorts to Ms Parry. Amongst other things, Dr Koorts was advised that Thea was 25 minutes old, had been born by vacuum delivery and had required CPR, intubation, adrenaline and an endotracheal tube. Ms Parry also told him that her heart rate was 120 bpm and improving, but the team were struggling to obtain saturations.[191] Dr Koorts was not told that Thea had been born in good condition.

170. Nevertheless, Dr Koorts considered, from the information provided, that Thea potentially had birth asphyxia, with PPH, resulting in poor saturations.[192] Although limited information had been provided to him, he considered it to be adequate for the purposes of making a presumptive diagnosis.[193]

171. Despite not having been told that Thea had been born in good condition, Dr Koorts gave oral evidence to the effect that such information would not have changed his working diagnosis.[194]

[186] Ex C1.17, [43]; Ex D1.5, p 28.
[187] Ex C1.2, p 7.
[188] Ex C1.17, [43]; Ex D1.5, p 30.
[189] Ex D1.5, p 30.
[190] Ex D1.7.1.
[191] Ex D1.7.3; Ex C1.14, [10].
[192] Ex C1.14, [13].
[193] T2-36, LL 10-12.
[194] T2-36, LL 14-35.

172. Having decided he had sufficient information to make a presumptive diagnosis and dispatch a retrieval team, Dr Koorts considered there was no further advice he could provide at that stage. Accordingly, a NeoRESQ team was arranged to be sent to Redcliffe Hospital on a priority 1 (ambulance with lights and sirens). The ambulance was scheduled to collect the team from the Royal Brisbane Women's Hospital in 35 minutes' time[195] and a bed was organised at the Mater Hospital.[196]

173. Dr Tshamala arrived at the hospital at 12:58am and took over management of Thea. He received a brief handover from Dr Kelly, advising that intubation had been successful and good respiratory support was being provided, but they were unable to gain intravenous access.[197] He was told that Thea was born in good condition with delayed cord clamping and that she had been skin on skin with her mother for the first five to six minutes of life.[198] He was aware that a NeoRESQ team was en route.

174. At 1am, Dr Georgia Sullivan, the NeoRESQ on-call neonatal advanced trainee, was contacted to attend the retrieval of Thea from Redcliffe Hospital. She was expected to be at the Royal Brisbane Women's Hospital within 30 minutes.[199] The only information Dr O'Sullivan had at that stage was that "Thea was a term infant with suspected Hypoxic Ischaemic Encephalopathy ('HIE') and was intubated and ventilated on 100% oxygen."[200]

175. At 1:06am, Dr Tshamala managed to establish intravenous access through an umbilical venous catheter. Thea was observed to be pale, with poor tone and she was grimacing. Her oxygen saturations were 72%.[201] Dr Tshamala's working diagnosis at this stage was that Thea had a lung infection or "any other congenital…respiratory disease".[202]

Transfer to neonatal unit

176. Thea was transferred to the neonatal unit at 1:11am for continued critical care. There, she was ventilated.[203]

177. The inability to obtain accurate oxygen saturation and ongoing issues with hypoxia continued.[204] According to Dr Kelly, transillumination of Thea's chest showed no sign of pneumothorax and a chest x-ray showed no pneumothorax or malposition of the endotracheal tube. The chest x-ray did, however, show that the umbilical catheter had been inserted into an umbilical artery rather than the umbilical vein. It was removed, and Dr

[195] So as to afford the on-call retrieval team sufficient time to get to the hospital from their homes.
[196] Ex D1.7.5; Ex D1.5, p 30; Ex D1.5, p 51; T2-50.
[197] T2-6, LL 16-27.
[198] T2-9, LL 16-23. See also: T2-16, LL 23-30.
[199] T3-4, LL 11-17.
[200] Ex C1.15, [13].
[201] Ex C1.2, p 4; Ex D1.5, p 30.
[202] T2-9, LL 28-36.
[203] Ex C1.17, [44]; Ex D1.5, p 56.
[204] Ex C1.2, p 4; Ex C1.17, [45].

Tshamala managed peripheral intravenous access. Despite the trialling of multiple ventilation modes, and multiple fluid boluses (inadvertently but inconsequentially given as 10% dextrose), there was no improvement in Thea's oxygenation.[205]

178. At 1:41am, Dr O'Sullivan and nurse Courtney Davidson departed the Royal Brisbane Women's Hospital.[206] Three minutes later, Dr O'Sullivan spoke to Ms Parry at the Redcliffe Hospital and advised that the retrieval team was 25 minutes away.[207] When asked whether this presented an opportunity for Dr O'Sullivan to provide advice to the Redcliffe Hospital team, Dr O'Sullivan gave evidence of a tension between the benefit of obtaining more information versus not distracting the treating team:[208]

> "Once again, I wouldn't have been providing advice because that's not my role over the phone, but us having more information prior to our arrival only would prepare us more. We obviously don't want to be taking up the time of the team that are actively trying to treat the baby and don't want to be providing any interruptions, particularly if I'm not able to provide advice, so I certainly wasn't wanting to take up any more of their time knowing how busy they would have been."

179. Like Dr Koorts, Dr O'Sullivan was not apprised of the information about Thea having been born in good condition. With the benefit of that information, HIE may have been a less likely primary diagnosis, however any differential diagnoses she may have reached by virtue of that information would not have changed her management approach. Nevertheless, she accepted that it would be preferable to be told such information.[209]

Arrival of NeoRESQ

180. Dr O'Sullivan and Ms Davidson arrived at 2:05am and met with Dr Tshamala. Dr Kelly had, moments earlier, been called to another delivery.[210]

181. Dr O'Sullivan said in her statement that on arrival, she received a minimal handover from Dr Tshamala, namely, that "Thea was a term baby with no significant antenatal history, intubated shortly after birth and required cardiopulmonary resuscitation and adrenaline, that the blood gases were poor and that they had been unable to oxygenate her."[211] When giving oral evidence about this, Dr O'Sullivan stated that it would have been helpful to have more information about Thea's condition at birth and, ideally, the entire history of Thea's deterioration.[212]

[205] Ex C1.17, [45]; Ex C1.2, p 7.
[206] Ex D1.5, pp 30 and 66.
[207] Ex D1.7; Ex D1.5, pp 30, 52, and 64.
[208] T3-5 to T3-6.
[209] T3-6 to T3-8.
[210] Ex C1.17, [46]; Ex D1.5, pp 59-62 and 66.
[211] Ex C1.15, [15].
[212] T3-6 to T3-8.

182. Despite having found out after Thea's death that Thea had been born in good condition, and having retrospectively considered further differentials, Dr O'Sullivan gave evidence that the information would not have changed her approach at the time, in circumstances where Thea was not born in a tertiary hospital.[213] Associate Professor Evans' evidence on this topic was that the information would not have made any difference.[214]

183. Dr O'Sullivan's observation of the situation on arrival was that Thea was clearly critically unwell. Further, it was assessed that Thea had severe hypoxia, poor perfusion, and was very encephalopathic.[215] Dr O'Sullivan's most likely differential diagnosis at that stage was HIE with severe PPH.[216]

184. Thea's heart rate at this stage was 150 bpm and her oxygen saturations were 57% on 100% of oxygen from the ventilator. She had no spontaneous movement, no grasp, no suck or gag reflex and her pupils were not reactive to light. [217]

185. At 2:45am, Dr O'Sullivan phoned Dr Koorts. This phone call was not recorded, however both Dr O'Sullivan and Dr Koorts addressed it in their statements. Dr O'Sullivan says that she relayed the information about Thea being critically unwell to Dr Koorts and he agreed with her plan to move Thea onto a Fabian ventilator[218] (which Dr O'Sullivan had brought with her) and conduct high frequency ventilation, commence nitric oxide, and move the endotracheal tube to a more desirable position.[219]

186. Thea was placed on the Fabian ventilator and nitric oxide was commenced. A brief improvement was seen in her saturation levels, however they quickly dropped and a saturation trace on Thea's hand was unable to be obtained. What happened next was best described by Dr O'Sullivan in her statement:[220]

> "…I was then informed of the gas results from 0145 just prior to our arrival which showed a pH 6.72 and lactate of 17.7, indicating she was acidotic and critically unwell.
>
> We were then told that while a full blood count and blood culture had been taken, no antibiotics had yet been administered. My recollection is that I asked at this point to the local team if they could be drawn up and given however I have not documented this in my notes or said specifically who I asked. At this stage Thea's perfusion was worsening and her saturations were beginning to drop further. An urgent chest X-ray was ordered to ensure there were no acute changes after the ETT

[213] T3-10.
[214] T4-27, LL 16-18.
[215] Ex C1.15, [17] – [18].
[216] T3-8, LL 35-37.
[217] Ex C1.15, [14] and [17].
[218] A Fabian ventilator provides more powerful support than a standard ventilator.
[219] Ex C1.15, [18].
[220] Ex C1.15, [18] – [19].

retape and commencement of high frequency ventilation – the ETT was then at a good position and the lungs were well inflated."

187. At 3am, Dr O'Sullivan again phoned Dr Koorts. During that phone call, Dr Koorts agreed that Thea needed central access to commence inotropes to assist with cardiac output and blood circulation. While this was being prepared, Thea's heart rate dropped to under 20 bpm.[221]

188. A code requesting more assistance was called and CPR commenced. Fluid boluses of sodium chloride were given and adrenaline was administered. Some spontaneous circulation returned and CPR was able to be ceased. Thea was, however, still severely hypoxic.[222]

189. Following a further phone call between Dr O'Sullivan and Dr Koorts, it was agreed that ongoing resuscitation efforts were likely futile and it was extremely unlikely that Thea would survive.[223]

190. After consultation with the family, active resuscitation measures were ceased. Thea passed away.[224]

Expert opinions on post-delivery care

<u>The working diagnoses</u>

191. Dr Luig was asked whether, prior to NeoRESQ's involvement, the Redcliffe Hospital team ought to have appreciated that there was a reasonable chance they were dealing with a cyanotic cardiac condition. Dr Luig gave evidence that HIE, which was being considered at the time, would be the most likely diagnosis "for the situation that the team were trying to manage". TGA would not be high on the list of differential diagnoses, in circumstances where there was a term baby, the mother received the standard obstetric care and most paediatricians, neonatologists, and retrievalists would not "be aware of this kind of presentation with this kind of rapidity of collapse".[225]

192. Dr Luig went on to explain that the severity of Thea's condition meant that it was "out of the normal" for undiagnosed TGA. Most times, babies born with undiagnosed TGA do not also have respiratory distress, as Thea did.[226] Under cross-examination, she said it would have been very difficult to reach Thea's diagnosis in a non-tertiary setting but even so, TGA combined with severe PPH is a rare condition and treating Thea, even in a tertiary hospital, would have been extremely difficult.[227]

[221] Ex C1.15, [20].
[222] Ex C1.15, [21].
[223] Ex C1.14, [20].
[224] Ex C1.14, [20].
[225] T3-28 to T3-29.
[226] T3-29, LL 18-29.
[227] T3-46, LL 31-44.

193. Associate Professor Evans said that with a baby like Thea, who was not responding to resuscitation in the sense of oxygenation, TGA or cyanotic heart conditions more generally, would be "way, way down the list". The "commonest" reasons would be lung-related issues and HIE would be one of the possibilities.[228]

Retrieval and resuscitation attempts

194. In relation to the timing of Thea's resuscitation, Dr Luig opined that where Thea's respiratory efforts deteriorated at around 15 minutes of age, the timing of the escalation was appropriate and within expected standards.[229] So too was the timing of the request for retrieval services.[230]

195. As to the resuscitation efforts themselves, Dr Luig considered that, aside from some deviations that had no bearing on the outcome, the hospital complied with the Queensland Clinical Guideline for Neonatal Resuscitation.[231] She did not believe that any concerns regarding the initial resuscitation, empty oxygen cylinder, depth of the endotracheal tube, or the use of incorrect intravenous fluid (dextrose rather than sodium chloride or Hartmann's solution) would have had any impact.[232]

196. Dr Luig was, however, slightly critical of the conversations between Redcliffe Hospital staff and NeoRESQ in the period that lapsed before NeoRESQ's arrival. To this end, she gave evidence that in Thea's case, the first two hours were critical, however the first conversation with clinical detail did not occur until 2:45am – approximately two hours after the retrieval request was activated.[233]

197. In Dr Luig's opinion, there should have been "doctor to doctor" conversations between the hospital and the retrieval service because part of the retrieval process is the clinical advice and planning between the time of the request and the arrival of the retrieval team.[234] The more sick the baby, the more important the clinical advice during this period.[235]

198. When asked what further information could have been provided to NeoRESQ, Dr Luig cited differences between the Queensland "call sheet" and the one used in New South Wales – specifically, that the Queensland version does not have "as many boxes to fill in with different pieces of clinical information as the one we use in New South Wales and, in particular, APGAR scores are not on there to be filled in."[236]

199. Notwithstanding this criticism though, Dr Luig said that any alteration in the thinking and differential diagnoses could only have come from

[228] T4-26, LL 8-45 T4-27, LL 1-8.
[229] Ex B1.8, p 12.
[230] T3-36, LL 13-19.
[231] Ex B1.8, p 10.
[232] Ex B1.8, p 18.
[233] T3-26, LL 21-29.
[234] T3-26.
[235] T3-40, L 3.
[236] T3-39, LL 1-7.

visualising Thea rather than hearing about her condition over the phone.[237] Further, that there was no advice that could have been provided to the hospital that would have changed the outcome.[238]

200. Associate Professor Evans was also asked about the appropriateness of the resuscitation efforts and whether any aspect of that care impacted upon Thea's death. He opined that Thea "was resuscitated to a high standard and her outcome was determined by her underlying congenital cardiac abnormality not the standard of her resuscitation."[239] In oral evidence, he clarified that he was referring to the efforts of both Redcliffe Hospital and NeoRESQ.[240]

201. According to Associate Professor Evans, given Thea's underlying cardiac condition and other vulnerabilities, there was nothing that could have been done by the hospital or NeoRESQ that could have saved her life.[241] He said that there was nothing that happened during labour or delivery that could have either adversely or positively affected Thea's prognosis.[242]

202. Concerning the exchange of information between the hospital and NeoRESQ, Associate Professor Evans said that while the initial call made by the nurse to RSQ was "not good," in that the nurse did not have sufficient information, in Thea's case, this did not make any difference to the outcome. He also noted that there were logistical issues such as staffing at that time of night, which meant that the person with the most information was the person dealing with the critical situation and so they could not "break away" to pass that information on.[243]

Use of prostin

203. During the inquest, evidence was heard about whether an infusion of prostin (also known as prostaglandin and Alprostadil) may have benefited Thea. This was primarily prompted by Dr Colen's report, in which he observed that the only additional potential treatment that could have been offered for Thea, was an infusion of prostin.[244]

204. As explained by Associate Professor Evans, prostin is used to maintain patency of the ductus arteriosis, while the baby is moved to a cardiac centre.[245] In diagnosed TGA cases, where the baby's saturations are borderline, prostin is used preventatively to ensure there is no change in oxygenation while the baby is in transit.[246] Notably, the ductus arteriosis does not usually close in a newborn until between 12 and 24 hours of life.

[237] T3-39, LL 23-33.
[238] T3-40, LL 22-36. See also: T3-48, LL 27-30.
[239] Ex B1.6, [49] – [50].
[240] T4-25, LL 11-20.
[241] T4-25, LL 22-29.
[242] T4-32, LL 35-38.
[243] T4-27 to T4-28.
[244] B1.7, p 6.
[245] T4-24, LL 28-42.
[246] T4-25, LL 5-9.

205. When asked about prostin in the context of Thea's case, Associate Professor Evans' evidence was that where Thea's ductus arteriosis would have been still open, prostin would not have made any difference.[247]

206. Dr Tshamala gave evidence that, in his view, Thea would have benefited from prostin.[248] Under cross-examination though, he accepted that prostin would not have affected the outcome in Thea's case because the ductus was open in the first few hours of life.[249] He agreed that commencing prostin within one hour of life was not an option at the time.[250]

207. Dr Koorts gave evidence that he would not have advised the hospital to start prostin because PPH was the primary diagnosis and TGA was low on the list.[251] In his opinion, there was nothing about Thea's case that indicated prostin was necessary.[252]

208. Dr O'Sullivan's evidence on this topic was that the team did not have time to get to the stage where drugs such as prostin could be considered. In any event, she thought it unlikely that if it had been administered, it would have made any difference.[253]

209. Dr Luig's opinion, as framed in her report, was that a prostin infusion soon after birth would have provided partial benefit only. It would not, however, have adequately improved Thea's cardio-respiratory instability.[254]

210. Expanding on this in oral evidence, Dr Luig noted that at autopsy, Thea's ductus arteriosis was patent – suggesting then that it had remained patent in her life after birth. Where prostin is used to maintain patency and does not result in an increase in saturations, given Thea's short life, Dr Luig did not believe prostin would have made any difference.[255]

211. As to Dr Colen's evidence at the inquest, despite raising the possibility of prostin in his report, his ultimate position was that prostin would not have made a clinical difference for Thea.[256]

Conclusions

212. The evidence before me raises only two possible areas of concern in relation to issue 4, namely, the discrepancy in the APGAR scores recorded at one and five minutes post-birth, and the adequacy of the information exchanged between the hospital and NeoRESQ prior to the arrival of Dr O'Sullivan.

[247] T4-24 to T4-25.
[248] T2-12, LL 4-6.
[249] T2-27, LL 30-32.
[250] T2-28, LL 16-17.
[251] T2-42, LL 8-10.
[252] T2-45, LL 33-36.
[253] T3-11, LL 25-34.
[254] Ex B1.8, p 11.
[255] T3-27.
[256] T5-21, LL 11-14.

213. Dealing first with the APGAR score discrepancy, it is important to bear in mind, as heard during the inquest, that APGAR scores are visually informed, and the assessment is a somewhat subjective one. They are a marker or indication as opposed to an absolute number. In Professor Weaver's opinion, due to interobserver variability, there will inevitably be variances in APGAR scores assessed by different people.[257]

214. Professor Weaver also explained that the one-minute score indicated that Thea needed careful watching. If the score is lower at five minutes, which it was, "then the baby definitely needs some sort of, you know, assistance with the extra oxygen or other, you know, stimulation or other, you know, assessment to see why the baby isn't really pinking up, having good tone, being vigorous crying and all the other things that newborns do."[258] This is what occurred and what prompted Thea's transfer to the resuscitaire.

215. I am satisfied that the APGAR score discrepancy is of little moment in Thea's case. The evidence leads me to conclude that Thea was born in a reasonable, if not good, condition, and that remained the case for her first five minutes of life. She was pink in colour, delayed cord clamping was possible, her eyes opened, she was breathing and making noises. It is recorded that she cried but her parents have disputed that.

216. Dr Luig gave some evidence that is particularly important for the resolution of the contention that Thea was in fact born in a poor condition, meaning that there was an inappropriate delay in her resuscitation. She said that when babies are born with previously undetected TGA and then experience collapse, the APGARs at 1 and 5 minutes are normal and the collapse occurs soon after then.[259] That the course described by Dr Kelly should be accepted as occurring is consistent with that clinical experience, when it is known that Thea had TGA.

217. Turning to Dr Luig's criticisms of the information exchanged between the hospital and NeoRESQ prior to the arrival of Dr O'Sullivan, whilst more information could have been provided to Dr Koorts and ideally, a more detailed and clinical-based conversation would have been appropriate earlier than 2:45am (when Dr O'Sullivan first spoke to Dr Koorts), Dr Luig herself said that these things would not have altered the outcome. There was no advice that could have been provided to the hospital, and without being able to visualise Thea, there could not have been any alteration in the differential diagnoses being considered.

218. It follows, therefore, that the communication between the hospital and the retrieval service was not such that it in any way contributed to the Thea's death.

[257] T4-8, LL 32-40; T4-9, LL 37 to T4-10, L 3.
[258] T4-9, LL 31-35.
[259] T3-21, L 25.

219. As to the remaining aspects of Thea's post-delivery care, I find that in circumstances where Thea had undiagnosed TGA and was not born in a tertiary hospital, both the Redcliffe Hospital and NeoRESQ staff did everything they could, and ought to have done, to try to save Thea's life.

220. Dr Luig and Associate Professor Evans both observed that Thea's presentation would have meant TGA was low on the list of differentials for the staff involved in her care. Respiratory related issues, such as HIE, were the more likely scenario, and so it was appropriate for this to have been the primary consideration at the time.

221. Notwithstanding what the hospital and retrieval staff thought they were dealing with, Associate Professor Evans' opinion, which is largely consistent with Dr Luig's opinion, is that Thea was resuscitated to a high standard, and nothing more could have been done to save her life. Thea's outcome was determined by her underlying congenital cardiac abnormality - not the standard of her post-delivery care. The Redcliffe Hospital neither had nor could be expected to have had the equipment, or the staff with the skills to use it, that would have been available at the Mater Hospital or the RBWH that would have afforded Thea the only chance of survival she could possibly have had.

Consideration of issue 5: Any appropriate changes to the practices, procedures or policies of the health service

222. Counsel Assisting have appropriately identified four matters for consideration in relation to this issue – training of sonographers, the suitability of the ultrasound technology at the Redcliffe Hospital, the adequacy of morphology scan guidelines, and the use of live-stream cameras within the hospital's neonatal resuscitation bays (coupled with the possibility of changes to the NeoRESQ intake form).

Training

223. When asked about increasing success in TGA detection rates, Dr Colen said that the training of local sonographers to improve, firstly, their acquisition of images and secondly, their understanding of what they are seeing in the images, has been an important step.[260] However, even some of the "very experienced" sonographers he works with can struggle to obtain ideal images.[261]

224. Dr Woodrow also mentioned training. She opined that practitioners should be given dedicated time at tertiary hospitals with fetal cardiologists who

[260] T5-4.
[261] T5-5, LL 6-10.

see abnormal hearts all the time – "the more number of abnormal scans you should see, the better your mid-trimester scan is going to be."[262]

225. Ms Leddy was asked whether, since Thea's death, she had undertaken any further training in relation to morphology scanning. Ms Leddy said she had done CPD courses and a day of training "talking about MFM specialling in obstetrics".[263]

226. Mr Collins was asked the same question. He stated that while he had not undertaken any additional training, he now has a much lower threshold for "bringing patients back for heart scanning".[264]

227. Professor Weaver also gave evidence on the topic of training – but in the context of radiologists. He told the court that following an audit of antenatal detection rates for congenital heart disease in 2013 or 2014, the Queensland Maternal and Perinatal Quality Council wrote to the College of Radiologists, which oversees ultrasound training of Australian radiologists. In doing so, the council suggested that particular views of the fetal heart obtained during morphology scanning required improvement and so the college should "look at their training".[265]

228. According to Professor Weaver, the college undertook to consider imaging recommendations stipulated by the International Society of Obstetricians and Gynaecologists and provide further training. Since doing so, detection rates have improved.[266]

Ultrasound technology

229. While no concerns were expressed in relation to the quality of the Philips Epiq machine used during Mrs Flaskett's morphology scan, some of the witnesses cited technology as being a likely factor in missed TGA detection generally.

230. Professor Weaver opined that technical difficulties associated with bone shadowing, obesity and positioning of the baby are relevant in this regard, as they can make obtaining the required views challenging.[267]

231. Dr Sinnott suggested that "better ultrasound machines with better colour imaging would likely improve the detection rate of some fetal anomalies."[268] His preferred machine is the GE Voluson,[269] which he says is superior due its software and hardware updates that enhance obstetric and gynaecology ultrasound imaging.[270]

[262] T6-33, LL 10-17.
[263] T1-22, LL 1-6.
[264] T1-37, LL 1-5.
[265] T4-4, LL 15-20.
[266] T4-4, LL 20-27.
[267] T4-4, LL 36-45.
[268] Ex C1.11, [10.7].
[269] Ex C1.11, [3.18].
[270] Ex C1.11, [6.2].

232. According to Dr Sinnott, as a "learning" from Thea's case, IMED loaned a high-end GE Voluson machine to the Redcliffe Hospital, and a senior sonographer from IMED provided training in relation to the use of the machine and improved scanning.[271]

233. Prior to, during and following the inquest, the court received three statements from Ms Tanya Oliver, Director of Medical Imaging at Redcliffe Hospital. Ms Oliver advised that the Philips Epiq machine used during Mrs Flaskett's morphology scan had a lifespan of eight to 10 years. Having been installed in 2015, the machine was scheduled for replacement in 2023.[272]

234. Following Thea's death, a procurement process was undertaken. This resulted in three new machines being purchased – one additional Canon Aplio i800 and two Philips Epiq Elites.[273] Ms Oliver says these machines scored the highest in qualitive ranking.[274] The selection criteria for the panel included image quality and usability in areas including obstetrics and gynaecology.[275]

235. In relation to the new machines, Mr Collins and Ms Leddy have both noted slight improvements, but Mr Collins maintained that the Philips Epiq had been more than adequate.[276]

236. Despite the tender process not resulting in the purchase of a GE Voluson, Dr Sinnott is satisfied that the hospital's new machines are suitable,[277] and he expects that they will improve the quality of colour imaging.[278]

237. According to Dr Woodrow, the Philips Epiq Elite is considered high end and is used extensively. In her opinion, the most important things are continued maintenance, accreditation with DIAS and the provision of funding to change the machines every seven years.[279]

Morphology scan guidelines

238. As has been discussed earlier in these findings, when it came to Mrs Flaskett's morphology scan, Ms Leddy and Mr Collins appropriately adhered to the ASUM guidelines as well as the additional imaging required by SOGI, which had been incorporated into MNHHS' morphology ultrasound scan guide.

239. Dr Colen explained that the ASUM guidelines have evolved over time to incorporate additional cardiac images with a view to improving TGA

[271] Ex C1.11, [6.2], [10.2], [10.3].
[272] Ex C1.13, [11].
[273] Ex C1.13, [18].
[274] Ex C1.13.1, [14].
[275] Ex C1.13.2, [13].
[276] T1-21, LL 44-47; T1-36, LL 29-35.
[277] T1-58, LL 35-36.
[278] T1-63, LL 9-13.
[279] T6-33.

diagnosis. He recalls that the last iteration of those guidelines occurred roughly eight to 10 years ago.[280]

240. Dr Woodrow told the court that the guidelines issued by "the British Society" no longer include a requirement for arch views during morphology scans. The ASUM guidelines are better in this respect, because arch views are still required and according to Dr Woodrow, this provides a "double opportunity" to see the outflow tracts and, in turn, detect TGA.[281]

Live-stream cameras

241. In New South Wales, paediatric resuscitation bays have live-streaming capabilities. Dr Luig was involved in piloting and establishing this program, which is called Vision for Life. It involves the use of cameras and microphones that facilitate live-streaming and simultaneous oral communications between on-site and off-site clinicians. As explained by Dr Luig:[282]

> "…there will be a camera on the ceiling that's pointing at the bed, and that camera will allow you to see the child and also see the monitors and equipment around the child, because seeing the monitors and equipment and what is being done for the child is very important. So it's a telly health where it's a – it's like cameras that watch you walk down the street but it's watching the child. And so if you're taking a call you can dial in and tell them to switch on their camera and you can see the child, often as you're talking to them the referring doctor or nurse as well. And the same thing is in place for non-tertiary special care units. There will be one or two patient bays where – which are dedicated for resuscitation for a very sick child – baby – where a camera will be on the ceiling for the same purpose, so you can see."

242. When asked whether this may have been advantageous in Thea's case, Dr Luig told the court that through the use of cameras, Thea's respiratory distress may have been apparent. If Dr Luig had been involved, she may have wanted to confirm whether PPH was present and what equipment was available in that regard. She may also have looked at Thea's colour, where the oximeter was, and which limb it was on. In turn, she might have asked the nurses to put two oximeters on - one on the right hand and perhaps one on Thea's leg. As she noted "it's very difficult to be a good doctor if you can't see the patient."[283]

243. Dr Koorts gave evidence that as at September 2023, video-conferencing was available but it was still in its infancy. He said it is used more often now, however it involves dialling into a conference room using a computer

[280] T5-4, LL 8-14.
[281] T6-30, LL 11-28.
[282] T3-37, LL 24-49.
[283] T3-38, LL 8-27.

and a camera – "in essence, it's the same thing as sitting down in front of the computer."[284]

244. Dr Luig was asked whether there exist any limitations with the use of the camera technology, and she was taken to Dr Koorts' opinion that "there are a lot of people standing around the bed and so it would be difficult to get a good view of what is happening". She explained that the cameras are positioned on the ceiling and the monitors on the walls. The cameras are not fixed into position – the view points can be adjusted and there is an option to zoom in.[285]

Conclusions

245. There is no evidence before me that the training of sonographers and radiographers is inadequate or requires any change at the health service level. Ms Leddy, Mr Collins and Dr Sinnott all adhered to the requisite guidelines and there is nothing to suggest that their training or experience in any way contributed to the missed TGA detection in Thea's case. As Dr Colen pointed out, even the most experienced of sonographers can struggle with fetal heart imaging. Any improvements in training that can be achieved apply to all sonography services. My impression is that the profession is consciously trying to improve detection rates, including by developments in training. Dr Woodrow's suggestion that sonographers have time in tertiary hospitals looking at abnormal scans seems intuitively sound and should be considered by hospital and health services for ongoing training. I will stop short of making an isolated recommendation for it because the model of training, already the subject of continuous development, is best designed wholistically, I expect.

246. As to the role technology can contribute to low detection rates, I am satisfied that the health service has acted appropriately in replacing and updating the hospital's ultrasound equipment. The new equipment is of high quality and has not attracted criticism from any of the experts. There were recommendations to use equipment that was the best for obstetric cases, made to the hospital after Thea's case, however it has elected to source equipment that was judged best overall for obstetric and other patients' needs. It is beyond the scope of this inquest to examine whether such a choice in the allocation of resources is appropriate, especially when the hospital established that the machines it was choosing produced high quality images in obstetric cases.

247. Concerning morphology scan guidelines, as noted above, the health service's guidelines adopted the additional requirements stipulated by SOGI. As such, the guidelines exceed those published by ASUM and for that reason, it cannot be said that they are, in any way, deficient. I have not heard any evidence to suggest that any changes to the guidelines

[284] T2-40, LL 8-26.
[285] T3-38, LL 37-43.

could be made so as to reduce the likelihood of similar deaths or to otherwise contribute to public health and safety and indeed there rather was endorsement of the current guidelines in preference to the British equivalent.

248. An appropriate change for consideration would be the installation and/or use of superior live-streaming capabilities in the health service's paediatric resuscitation bays, as described by Dr Luig. While there may not have been any direct correlation between the use of that technology and Thea's prospects of survival, I accept Counsel Assisting's submission that, in other cases, it could be life-saving. I note there is some facility at Redcliffe Hospital and other like hospitals with NeoRESQ for video conferencing but my impression from the evidence is that it is inferior to the NSW system. I will confine my recommendation to MNHHS and NeoResq reviewing the NSW live streaming resources to determine if there are improvements to facilities in Queensland which would be appropriate to make. Similarly, I will recommend a like review be made of the intake form used in NSW be made to see if improvements can be made to the one used in this State.

Conclusions

249. The weight of the evidence supports a finding that Thea's death was not preventable, her TGA not having been detected antenatally.

250. Thea was born with undiagnosed TGA complicated by reduced shunting and/or PPH.

251. The reduced shunting, in particular, made Thea's case high-risk and rare,[286] and the presence of PPH, if it was, would have increased her vulnerability.[287] These compounding factors determined her trajectory. She simply stood no chance of survival.

252. Ideally, Thea's TGA would have been suspected during Mrs Flaskett's morphology scan on 5 May 2023 and gone on to be diagnosed by a materno-fetal medicine specialist or paediatric cardiologist soon thereafter.

253. As discussed above in respect of issue 2 though, there are limitations to morphology scans, and no matter the quality of the scan, the possibility of an undetected congenital heart defect will always exist.[288] As explained by Associate Professor Evans, the anatomy of the heart is unusually complex and difficult to examine with ultrasound, in utero. It requires prolonged and specialised training and relies on cause for suspicion to be seen.[289] Accordingly, TGA is not uncommonly missed.[290]

[286] T3-43, LL 15-31.
[287] T5-23, LL 23-28.
[288] T6-36, LL 37-44.
[289] Ex B1.6, [34].
[290] T1-59, LL1-2.

254. In Thea's case, the artificial and erroneous appearance of three vessels and an arrowhead, as seen not only by the sonographers and radiologist but also Dr Woodrow, meant that cause for suspicion was not seen.

255. If Thea's TGA had been diagnosed antenatally, and she was born in a tertiary hospital, her survival was not guaranteed. As observed by Dr Colen, undiagnosed TGA in isolation does not inevitably cause death, and prenatal diagnosis would not entirely have ensured the prevention of Thea's death. Her additional high-risk factors, which are not always discernible before delivery, increased her risk and would have been difficult difficult to treat.[291] Thea though would probably survived, had TGA been detected in the morphology scan, in my view.

256. The Redcliffe Hospital and NeoRESQ staff did everything they could in the circumstances and with the resources they were presented with. Despite resuscitating Thea to a high standard, her outcome was determined by her underlying congenital cardiac abnormality - not the standard of her post-delivery care.

Findings required by s. 45

Identity of the deceased – Thea Flaskett

How she died – Thea died shortly after birth as a result of complications associated with undiagnosed Transposition of the Great Arteries.

Place of death – Redcliffe Hospital REDCLIFFE QLD 4020 AUSTRALIA

Date of death– 11/09/2023

Cause of death – 1(a) Main disease or condition in neonate: Transposition of the Great Arteries.
1(b) Other diseases or conditions in neonate: Nil.

Comments and recommendations

257. In accordance with s46 of the Act, a coroner may, whenever appropriate, comment on anything connected with a death investigated at an inquest which relates to public health or safety, the administration of justice or ways to prevent deaths from happening in similar circumstances in the future. Comments may take the form of recommendations to government or to particular agencies to make or consider systemic changes or

[291] Ex B1.7, p 5.

amendments to existing policies or procedures. I have canvassed the relevant matters under Issue 5 herein.

258. I recommend that MNHHS and NeoRESQ make inquiries as to the resources used in NSW public hospitals for live-streaming engagement of the peers of NeoRESQ consultants, in paediatric resuscitation bays in hospitals they serve, to permit adequate visualisation of the patient and attending staff, as well as oral communication. I also recommend that NeoRESQ and MNHHS enquire as to the detail required to be completed on the intake form for the like neonatal retrieval service in NSW, to see if amendments to the form used in Queensland would be apt.

I close the inquest.

Stephanie Gallagher
Deputy State Coroner
BRISBANE