Inquiry into Convictions of Kathleen Folbigg

Submissions on behalf of Kathleen Folbigg

PART C - SARAH

Cause of Death – Sarah

1. Sarah's death can be attributed to a number of alternative causes unrelated to homicide. In summary, Sarah's death could have been caused:

   (a) By obstruction caused by the uvula;

   (b) By laryngospasm triggered by the uvula;

   (c) By cardiac arrhythmia triggered by mild infection; or

   (d) Bed sharing.

2. We shall deal with each issue in turn.

Crown Opening[1]

3. With respect to Sarah, the Crown opened as follows:

   A post-mortem examination was conducted by Professor Hilton. Professor Hilton is the head of the New South Wales Institute of Forensic Medicine, which is basically what we commonly know as the morgue at Glebe. He found a 1.5 centimetre scratch on Sarah's upper arm and two tiny punctate abrasions, which are like prick marks, one below the lower lip and one near the midline of the chin. The lungs had collapsed. There were petechial haemorrhages. There was petechial bleeding in the lungs of the petechia. He found that the uvula, the dangly bit at the back of the mouth, appeared displaced, but he only discovered that when he dissected the back of her throat and removed it from the body and examined it and he said it could easily have become displaced during the dissection process or the movement of the part that he had

[1] Exh F T 39.21-.37, T 67.08-.19.

dissected. He considered that this was not the likely cause of death. He found various other features which he will describe to you.

…

The Crown case is that from these doctors, from these experts, you will be able to conclude the following. Firstly, none of these children died from the mysterious disease of SIDS[2]*. Next Caleb didn't die from a floppy larynx or any other natural causes. Next Patrick did not have a spontaneous epileptic episode when he had his ALTE, but he suffered brain damage from lack of oxygen which caused him to become epileptic. Next that his epilepsy did not cause him to suddenly stop breathing and die. Next that Sarah didn't die from a displaced uvula or any other natural cause. And lastly that Laura did not die from myocarditis or any other natural cause.* (emphasis added)

Evidence at Trial

4. Prior to her death, Sarah had been unwell with a cold like illness, for a week or so.[3] There was no sign of neglect[4] and no injuries to her on autopsy.

5. The autopsy report demonstrated five features that demonstrated health issues[5]

6. Professor Hilton performed the autopsy, and after a thorough investigation opined that the cause of death was SIDS[6].

7. With respect to Sarah, the evidence at trial was to the effect the presentation of the uvula to Professor Hilton at autopsy was likely to be an effect of post-mortem and incidental to the cause of death. He gave evidence the uvula was in the incorrect position.[7] It was red, which was consistent with infection or abnormal movements.[8]

[2] This shows another example of the misunderstanding of the medical condition. SIDS is not a disease, and it is in error to describe it as such.
[3] See ERISP Q 270, Report of Death to Coroner Exh H page 91.
[4] Exh H page 104.
[5] Autopsy report Exh H page 98.
[6] Exh H 98.
[7] Exh F T 662.49.
[8] Exh F T 622.10.

8. Prof Hilton gave the following evidence at trial[9]:

Q: Is that what you are saying, that the uvula, the dangly bits at the back of the throat, may have in fact been moved by the process of you dissecting the throat area?

A: Yes.

Q: So it may have been in its normal position when the throat was intact with the body?

A: Yes. The normal position, in a baby of this age, there isn't very much separation but there is separation and it may just have been a dissection artefact, if you like. (T 622) …

Q: So what you are saying is that by the time you came to examine the dissected throat, the epiglottis was in an abnormal position?

A: Yes.

Q: As compared to the epiglottis --

A: In the living position. The epiglottis was in an abnormal position when I did my examination in comparison to what I would anticipate is the normal anatomical position

…

Q: You have told us the uvula, the dangly bit at the back of the throat, was not enlarged?

A: It wasn't disproportionate to the uvula of a child of this age. Now, when I say that, there's obviously a spectrum from very small to quite big.

Q: You have told us that it was red?

[9] Exh F page 621.

A: It was red, yes.

Q: Is that consistent with a child having a mild infection?

A: A mild infection or some, perhaps, abnormal movements of the uvula, such as in snoring.

Q: Doctor, in your view was the position of the uvula when you saw it in the dissected throat in its abnormal position, was that of any significance in terms of assessing the cause of death of this child?

A: It was of significance in a sense that it was there, I saw it and I commented on it. I've got grave doubts that it was necessarily there before the child died and before I started my dissection. It may have been, I just cannot tell.

Q: Do I take it from what you have just said that in your view, it was of no significance in terms of explaining the cause of death?

A: Well, it could have been. But the degree of uncertainty as to whether it was, what I was seeing was real or artefact, to my mind diminished its significance or caused me to question its significance. … (T 623)

…

Q: What did you find?

A: The uvula showed marked vascular congestion, particularly in the pharyngeal aspect adjacent to the base. I am reading from the report. (T 625)

9. Prof Hilton went on to say:[10]

Q. Doctor, in your view was the position of the uvula when you saw it in the dissected throat in its abnormal position, was that of any significance in terms of assessing the cause of death of the child?

[10] Exh F T 623.12-.28.

A. It was of significance in the sense that it was there, I saw it and I commented on it. I've got grave doubts that it was necessarily there before the child died and before I started my dissection. It may have been, I just cannot tell.

Q. Do I take it from what you have just said that in your view it was of no significance in terms of explaining the cause of death?

A. Well, it could have been. But the degree of uncertainty as to whether it was, what I was seeing was real or artefact, to my mind diminished its significance or caused me to question its significance.

10. Then on microscopy he observed:[11]

Q. Did you find any abnormal finding on any of these items when you examined them microscopically?

A. Yes.

Q. What did you find?

A. The uvula showed marked vascular congestion, particularly in the pharyngeal aspect adjacent to the base. I am reading from the report.

Q. Did that tell you anything at all about the cause of death?

A. It is a brick in a wall of diagnoses. It is indicative - it confirmed the reddening that I saw and described to the naked eye. The larynx showed a light mixed lymphocytic inflammatory infiltrate deep to the respiratory epithelium. That was a light degree of inflammation present in the larynx, that's the voice box. In the diaphragm there were two foci of individual muscle fibres which looked not quite healthy. The spleen was congested. There was a little bit of probably fatty infiltration of the liver, but that's not unusual. That's often accompanying the process of death.

[11] Exh F T 625.30 - T 626.53.

The rest of the sections were normal until the lungs and in one section of the lung there was some interstitial acute inflammatory infiltrate which could be seen around the occasional bronchial. Now, the bronchials are the smallest of the respiratory passages and there was just a very light inflammation around these little air passages. In another section of lung there was polymorpho nuclears. These are inflammatory cells within the lymph glands and again some interstitial infiltration by these inflammatory cells.

Q. If I could just take you to, does that conclude your findings on microscopic examination?

A. Yes.

Q. Firstly, going to the uvula. You found vascular congestion of one aspect of the uvula?

A . Yes.

Q. Is that consistent with a mild infection?

A. Yes.

Q. The kind of infection that might cause a mild sore throat?

A. Yes.

Q. The larynx showed a light inflammatory infiltrate?

A. Yes.

Q. To the epithelial layer?

A. Yes.

Q . Is that also consistent with a mild respiratory infection?

A. Yes.

Q. Did you also find that the salivary glands had an area of inflammation?

A. Yes.

Q. Is that also consistent with a mild infection?

A. Yes.

Q. Going to the lungs, you found a light inflammatory infiltrate of a part, a certain type of cell in the lungs?

A. Yes.

Q. Is that also consistent with a very mild respiratory infection?

A. Yes, indeed.

Q. And you also found some other abnormal finding within the lungs, which I won't repeat because they are just technical words?

A. This is a child who had mild manifestations of a respiratory tract infection…

Q: *In your view was it in any way related to the cause of death?*

A: <u>That's a very difficult question to answer, in a one size fits all sort of context. I don't know how much it takes to kill a child, that's the problem, and the more I see of babies that die unexpectedly, the less certain I have become of how much it takes to kill a child. I would normally not have expected this degree of inflammation to have contributed significantly to his child's death.</u> (T 628)

11. Professor Hilton note that two groups of germs were grown from the lung. One was streptococcus and the other was staphylococcus. The last organism was there is scanty proportions. The streptococcus was there in profuse growth.[12] Professor Hilton thought the staphylococcus was of no significance whatsoever because he thought it was a post-mortem contaminant.[13]

[12] Exh F T 627 V-X.
[13] Exh F T 628.

12. At trial, Dr Beal suggested the swollen uvula could have been "at most, post-mortem artefact".[14] At the Inquiry, the evidence suggests this assertion was incorrect. The uvula could have become swollen due to sticking or getting caught behind the epiglottis.[15]

Crown Address

13. The Crown address did not deal with the potential link between sudden infant death and mild infection. This evidence has only been adduced at this Inquiry and it is one of the major medical developments in SIDS research, it having been established there is a statistical association in almost 50 per cent of cases. Further, the infectious disease and immunology opinions demonstrate the scientific link between infection and sudden death in infancy. This is addressed elsewhere in these submissions.

14. The Crown did address on the basis that Sarah was otherwise in good health. If the association between mild infection and sudden death in infancy is considered, then regardless of any other information before this Inquiry, the mild infection was not benign. It was potentially fatal for an infant.

15. Further, the Crown addressed on the uvula on the basis that:

    (a) Prof Hilton did not think it was the cause of death;[16]

    (b) Dr Byard and Dr Berry had never had experience of a child dying of a displaced uvula;

    (c) "*In all medical literature there has never been a child that has died from a displaced uvula …*", so in other words, what they were saying is this: "*If Sarah died from a displaced uvula, this was the first reported incident of such a death worldwide, so far as they are aware*".[17]

[14] T 1142.21.
[15] See T 241.24 - T 243.46.
[16] T 1340.47.
[17] T 1340.55 - T 1341.05.

16. With respect to Sarah, the Crown submitted as follows:[18]

Professor Berry said that if he was looking at Sarah's death in isolation he probably would have said SIDS but he would have had misgivings. Of course Professor Hilton was not looking at Sarah's case in isolation. He knew perfectly well what had happened to the previous children. The conclusion is that Sarah died from undetermined causes. She had a displaced uvula. That was the only thing that Professor Hilton could find that was in the slightest bit out of the usual, a displaced uvula. He was of the view that that was not the cause of death.

Dr Cala and Professor Berry and Dr Beal were of the view that that was not the cause of death. The defence expert, Professor Byard, and the Crown expert, Dr Berry, both said that they have never had a child who has died of a displaced uvula. Their colleagues have never had a child who has died of a displaced uvula. In all of the medical literature there has never been a case of a case of a child that has died from a displaced uvula; there was one child who had a very elongated and split uvula who died, that is referred to in a French article. So, in other words, what they were saying is this: If Sarah died from a displaced uvula, this was the first reported incident of such a death worldwide, so far as they are aware. (Emphasis added)

Judge's Summing Up

17. With regards to Sarah, the Trial Judge addressed the content of the diaries at some length. Putting that to one side for the purpose of examining the medical causation case, the Trial Judge summed up as follows:

Just coming back to the medical evidence briefly. The Crown submitted to you, particularly in his attack on Professor Hilton, that the punctate abrasions ought to have put him on the alert and ought to have been followed up, investigated.

Mr Zahra says that is only a distraction and that in the absence of photographs and in the absence of any precise description of what these abrasions were like, except that they were punctate, which I take to mean pointed - and that suggests that they were small -

[18] T 1340.37 - T 1341.04.

one has no idea what they were like and is left speculating. That is one thing that you ought not to do .

He says that there is no positive indication of smothering. He points out that Professor Hilton still prefers SIDS to smothering.

The Crown says he has tried to defend a position that he has held for a few years now.

None of the experts seems to think that the uvula could possibly explain the death , so this is put forward as a SIDS death. Not that the defence has to prove that it was a SIDS death, but that is what is suggested should be seen as the reasonable natural cause, a cause which is natural but which cannot be identified.

The other two factors that might make an expert doubt that this was a SIDS death is the age of Sarah. She was, I think, ten and a half months old. You will remember the classic, if I can put it that way, age range is two to six months, although that does not mean that it could not have been properly called a SIDS death, and the fact that Sarah was on her back and not her front. Now, they seem to be the arguments concerning the death of Sarah (Emphasis added)

Evidence before the Inquiry

18. By the time of the Inquiry, Prof Hilton had changed his professional opinion considerably from his evidence at trial.[19]

    (a) There were other reported cases in the literature of the uvula causing sudden infant death as it became stuck in or to the epiglottis;

    (b) That the structures of the upper respiratory tract in infants is subject to considerable change. In this regard, the paper by Marom et al "*Otolaryngological aspects of sudden infant death syndrome"* International Journal of Pediatric Otorhinolaryngology 76 (2012) 311-318 was important new evidence regarding that risk;

[19] T 324.34 – T 325.07.

(c) The uvula of Sarah was inflamed which could have been a result of its striking the epiglottis. In other words, it may have been red not through infection but by repeated contact with the epiglottis;

(d) Had Sarah's uvula struck the epiglottis, it was possible the uvula could block the airway. If this occurred, she could die;[20]

(e) Alternatively, it was established at the Inquiry that trauma inflicted upon the uvula as a result of sticking the epiglottis (as could happen with snoring[21]) could give rise to a laryngospasm giving rise to obstruction and death

(f) Alternatively, the uvula may have reflected an infective response to a bacteria or mild viral illness.

19. In half of all SIDS deaths, there is a history of a mild viral illness in the days leading up to the death.[22] This information about the connection between mild infection and sudden death in SIDS was not adduced at trial and was not understood at trial. This scientific information has since been consolidated by a great deal of research as set out in the reports of Professor Blackwell (Exh T and U), and the report of Professor Clancy (Exh W and AT) and the Report of Professor Goldwater (Exh AU).

20. The death of Sarah presents three significant advances in medical knowledge that presents a significantly different evidential landscape than the one at trial. These three areas raise a distinct possibility of a natural death due to a number of features:

(a) The uvula becoming stuck behind the epiglottis and causing death;

[20] T 234.01-.15, T 234.32-.34,
[21] T 234.29.
[22] Exh J, page 4- this was thought to be due to genetics and the cytokine response. See also Professor Horne's evidence at T 38.15 and T 49.05-.15, and the triple risk model in Exh D Chapter 30.

(b) the uvula becoming irritated and triggering laryngospasm (tightening of the larynx and obstruction);[23]

(c) mild infective process triggering the cytokine response and sudden death (this is addressed elsewhere and will not be repeated here).

21. At the Inquiry, the evidence of expert microbiologist (Prof Blackwell) and immunologist (Prof Clancy) is that "mild infection" can trigger a sudden arrhythmia that can cause sudden death. This phenomenon is addressed in Byard Chapter 30 and supported by the studies referred to by Professor Horne that demonstrate 50 per cent of SIDS deaths demonstrate mild viral infection in the days preceding death.[24]

22. Professor Blackwell thought there was not much evidence the swollen uvula was blocking her breathing.[25]

23. However, she opined that the swollen uvula was indicative of an infective process, and, as such indicated that the inflammatory response had been triggered. This leaves open the possibility of an infective process as a cause of death.

24. Dr Cala expressed some concern about the swollen uvula in his report (Exh M p 12).

25. Professor Hilton's opinion has been summarised elsewhere. The nett effect of his evidence is that the uvula could have caused an obstruction of the epiglottis, which could cause a failure of breathing, or alternatively, it could have triggered laryngospasm, which would abate after death and leave no signs.[26] In this regard, he relied on the article about the otolaryngolical changes in infants and their relationship to sudden unexpected death. The forensic pathologists were reluctant to speak of the cause of death in circumstances

[23] T 243.16-.40.
[24] Ex J page 4, T 38.15, T 49.05-.15.
[25] T 316.15.
[26] See T 175.33 - T 176.33, T 233.43 - T 234.14, T 233.25 - T 235.21, (noting Cala who would not commit at T 176.38 - T 241.25), Duflou who was uncertain T 176.38- T 241.25, Cordner T 177.36-.43, T 279.60 - T 281.30.

where they were dealing with an issue outside their field.[27] Yet it is clear from the literature and the evidence of the forensic pathologists that the certainty expressed by Prof Hilton at trial was a matter from which he resiled, and the uvula as found on autopsy, and its appearance gave rise to a readily available alternative natural cause of death in Sarah.

26. It is clear from evidence adduced at this Inquiry that:

    (a) There had been previous reported incidents of sudden death from a displaced uvula;

    (b) There were sound otolaryngolical reasons for the risk of such death due to the considerable changes in the structures of the upper respiratory tract in early infancy.[28]

    (c) Not only is a displaced uvula a risk of death, but so is a laryngospasm which can be triggered by the uvula sticking to the epiglottis while snoring. This can cause death through airway obstruction. This was either not known at trial (it being a matter for specialist otolaryngolical opinion) and appears to have been the subject of articles within that field since the trial or was not the subject of evidence a trial.

27. It is submitted that had the evidence that was available at the Inquiry regarding deaths from a displaced uvula, laryngospasm, and infection been available to the jury, then the Crown address and the judges summing up would have taken on a wholly different complexion, and that there was a readily available alternate natural cause of death that would have to be excluded by the Crown, and it could not do so.

28. On this basis alone, the Inquiry would have a reasonable doubt regarding Kathleen Folbigg's guilt with regard to her conviction for murder of her daughter Sarah. Further, once the doubt was established with respect to Sarah, there would be a doubt about the conviction on the other counts by reason of

[27] Duflou T 177.23-.33, T 180.12; Cala T 243.40, T 244.43, T 233.25 - T 235.21.
[28] See Marom et al, supra.

the Crown's invocation of the method of reasoning arising from the evidence of "three or more deaths in the one family".

29. Finally, had that evidence been presented at the pre-trial application, there is a real question as to whether the application to try the five counts together is open to doubt and whether the coincidence evidence would have been admitted into evidence due to the prejudicial effect outweighing the substantial probative value.

30. Findings should be made to this effect.

Bed Sharing - Sarah

31. This is an additional matter that has arisen through this Inquiry.

32. It is clear from the evidence at this Inquiry that bed sharing is a high risk for sudden infant death.[29] It is clear from the evidence of Prof Blackwell that heat permits the growth of staphylococcus aureus which is associated with sudden infant death. It is also clear that children who are kept too warm are at risk of sudden infant death.

33. The evidence given at trial was ambiguous as to which bed Sarah was in at the time she died. It is important for the police to capture a witness' recollection as close as possible to the evens to prevent conscious or unconscious reconstruction or pollution.

Risk of Bed Sharing

34. The literature is replete with this information and it was confirmed by Prof Horne[30] and Dr Elder.[31] The reason for the risk is either accidental suffocation or overheating.[32] While the forensic pathologists opined the overheating of a child was "a theory", Prof Elder told the Inquiry that

[29] T 25.30, T 26.14, T 28.26, T 28.36, T 30.50-T 31.01, T 33.47, T 43.43, T 70.41.
[30] T 24.14.
[31] T 30.46 - T 31.01, T 32.20.
[32] See forensic pathologists at T 245.48 - T 246.27.

overheating was an issue and that with a change in practice, the outcomes have been modified.[33] Accordingly, this theory has had an effect when put into practice so the Inquiry would place greater weight on it. Overheating can cause the propagation of staphylococcus aureus which has been demonstrated to be related to sudden infant death.[34]

35. Findings should be made to this effect.

36. Mr and Mrs Folbigg used to bed share from time to time with their children because they would sleep better. This was particularly the case with Sarah.[35]: She stated in her ERISP:

> *She would sleep quite happily if she was in the bed with us. Yeah, she would sleep for hours and hours on end if she was in the bed with us but not if she was in a bed by herself.*[36]

37. The ERISP[37] was conducted six years after Sarah's death[38]. Mr Folbigg's evidence at trial was given ten years[39] after the death of Sarah. His recollection was also likely to have been affected by the effluxion of time.

38. In these circumstances, the most important evidence is that contemporaneously generated documents assume great importance. Detective Senior Constable Ward[40] was stationed at Singleton and was a crime scene investigator. He received a telephone call from Constable Saunders at about 3:30 am on 30 August 1993 and was asked to attend the scene in relation to the death of Sarah.

39. He arrived and had a discussion with Constable Saunders who advised him of a history of prior deaths in the family, the age of the child and the prior medical history of Sarah, including that of a recent cold and the time of her last meal.

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[33] T 27.08-.10.
[34] See evidence of Professor Clancy and Professor Blackwell addressed elsewhere in these submissions.
[35] Exh E, ERISP Q.
[36] ERISP page 64.
[37] 23 July 1993.
[38] 30 August 1993.
[39] 2 April 2003.
[40] Exh BQ.

He took a history of the use of the sleep monitor and the time of discovery of the child. All of this information was undoubtedly correct and was contained in his statement. This information was obviously provided by one or both of the parents to Constable Saunders and Constable Saunders relayed it to Detective Senior Constable Ward.

40. Importantly, Constable Ward records the following in his statement:

That on the evening of the 29 August, 1993, the parents went to bed about 9.30pm, but at some time removed the monitor from Sarah and moved her into the bed with them. (Emphasis added)

41. This is relevant and contemporaneous evidence that at the time of their attendance, Constable Saunders was told by the parents that Sarah was in the matrimonial bed with her parents prior to death.

42. As a result of this information, Detective Senior Constable Saunders took a series of photographs but principally of the matrimonial bed.

43. It should be inferred from the third line in this document and by the reference "*Maitland CSS Jobs 93/652 …*" that on return to the station, he created a job file and ascribed that number to it. It should also be inferred that at the time of the creation of his statement on 14 December 1999, he had access to that job file for the purpose of preparing his statement and producing the photographs he took on 30 August 1999.

44. Accordingly, it should be inferred the information contained within his statement is accurate and reflected what he was told at the time of his attendance and that this information reflected what police were told by the parents on the night.

45. It matters not that Mr Folbigg gave different evidence at the trial or that Mrs Folbigg gave a different account in her ERISP. Both occasions were some years after the event.

46. The first account given to Police who first attended on the night in question by one or both of the parents was that Sarah was initially put to bed in her own bed. Sometime during the night she awoke and she was placed in her parents' bed.[41]

47. If one gives any credit to the statement of Detective Senior Constable Ward , then this evidence must raise a reasonable doubt about the death of Sarah given the sleeping arrangements at the time. It is impossible to ignore this clear, unambiguous contemporaneous statement by a police officer in the course of his duties as a crime scene examiner.

48. This evidence was not adduced at trial. Detective Sergeant Ward was not called to give evidence. If this account is accepted, then (although it is "*difficult in sorting out what's actually happened then through sleep*"[42] it is relevant as a potential natural cause of death for two reasons, one related to the advances in science and the other unrelated:

    (a) If she was bed sharing, then there was a risk she was overheated, which has an effect on bacteria reproduction (as per Prof Blackwell) or otherwise for some unspecified reason has a practical effect on survival outcomes.[43] The statement by Prof Blackwell that sudden death is related to bacteria is supported by clinical signs of infection in the lungs, spleen and uvula of Sarah, and this in turn supports a conclusion the bacteria found at autopsy was pathogenic rather than contaminant;

    (b) It gives rise to a potential cause of death by accidental smothering.

49. This piece of evidence is critical and on this matter alone, the Inquiry would conclude there is the requisite doubt about the conviction with regards to the charge of the murder of Sarah. If Sarah was in the matrimonial bed, then there

[41] This was a common practice at the Folbigg household- see Record of interview. In considering the evidence at the record of interview, it must be borne in mind that there was ample opportunity for Kathleen and Craig to have spoken about the events of that night, and the opportunity for unwitting reconstruction. The record of interview was held some years after Sarah's death.
[42] Cala at T 70.33.
[43] Prof Elder at T 27.08-.10.

was an alternative natural cause of death that the Crown could not exclude. Had that evidence been adduced at trial, the Crown could not have addressed in the manner he did that there was no alternative available innocent explanation, nor could the judge have given a summing up that failed to mention this issue. The evidential landscape at the Inquiry is very different from that presented at trial.

50. Further, this evidence would have had an effect on the capacity of the Crown to obtain a joint trial or to tender evidence of coincidence. If one child is sleeping with her parents (which could cause sudden death) and the other three are in their own beds, then the existence of similar facts giving rise to the admission of coincidence evidence is significantly eroded. In short, there is no coincidence between Sarah and the other three.

Submissions of Counsel Assisting

51. As to paragraph [106], the petechial haemorrhage could have been caused by hypoxia due to upper airway obstruction.

52. The evidence referred to in [113] was given before there was any consideration of the uvula being a possible cause of upper airway obstruction, either by way of sticking the epiglottis or laryngospasm. This has been dealt with earlier in these submissions at length.

53. As to the submission at [127], the wrong question is identified. It is not a case of attributing the cause of death to infection. The issue is whether it is a reasonably available alternative natural cause of death, and if so, can the Crown exclude it? Sarah had the combination of clinical history, clinical signs, positive autopsy findings with respect to infection which gives rise to a likelihood the blood results were not contaminant. Even putting aside the contamination question, Sarah had infection. Infection can cause arrhythmia. Infection can combine with genetic variants to cause arrhythmia.

54. The analysis of likelihood as against a characterisation that she was at low risk of SIDS has problems. Firstly, 50 per cent of SIDS victims have mild infection, so the submission is wrong. Secondly, it is dangerous to apply epidemiological material dealing with population studies to an individual when attempting to deal with causation.

55. The submissions of Counsel Assisting should be rejected. There are reasonably available natural causes of death that the Crown could not exclude. As such, any coincidence evidence is seriously undermined. As such, the evidential landscape has changed considerably since trial. A reasonable doubt is established as to whether joint trials should have been ordered, coincidence evidence admitted and the guilt of Ms Folbigg for the death of her daughter Sarah.

Submissions of the DPP

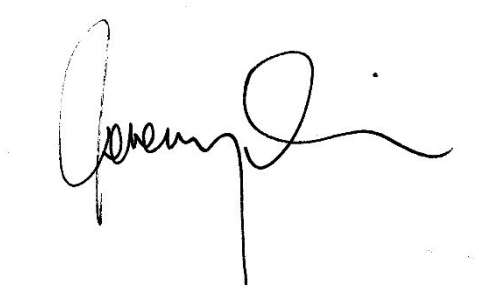
56. The DPP repeats the submissions of Counsel Assisting and it is submitted, embrace the same errors.

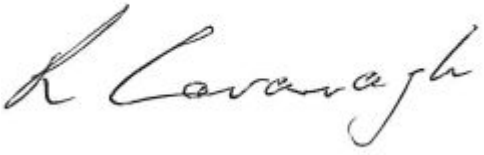
Summary

57. There are a number of alternative natural causes of death for Sarah. That her death has been described as "undetermined", there is a world of difference between a cause of death that is a "*debating point possibility*"[44] and one which is readily available, but which cannot be determined with scientific certainty. The Crown has the obligation to exclude a reasonable cause of death. It cannot do so, given the evidence at the Inquiry, and this gives rise to a reasonable doubt about the conviction by the jury, and her guilt. Findings should be made to this effect.

[44] *R V Follbigg* [2005] NSWCCA 23 at [143] which needs to be read in the context of findings and observations at [63], [80]-[81], [91], [103], [128], and [143].

Dated: 7 June 2019





Jeremy Morris SC
13th Floor St James' Hall
169 Phillip Street
SYDNEY NSW 2000
Tel: (02) 9335.3040
Fax: (02) 9335.3099
DX 266 Sydney
jeremymorris@stjames.net.au

Robert Cavanagh
Sir Owen Dixon Chambers
Sydney/Newcastle
1/360 Hunter Street
Newcastle NSW 2300
Tel: (02) 4925 2371
robert.cavanagh7@hotmail.com

Isabel Reed
Dame Roma Mitchell Chambers
P O Box 681
NEWCASTLE NSW
Tel: 0411.240.650
Isabel_reed@hotmail.com

Counsel instructed by Stuart Gray, Cardillo Gray Partners