Report of the Inquiry into the convictions of Kathleen Megan Folbigg

July 2019

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Acknowledgments

Acknowledgments

1. First and foremost, I express my appreciation for the assistance provided to the Inquiry by Ms Gail Furness SC, counsel assisting. Much of the evidence in the Inquiry involved complex scientific and medical evidence that required careful analysis and preparation for the Inquiry. Without her efforts, the Inquiry would have been much longer and less successful in understanding the issues. She was ably assisted by junior counsel, Ms Ann Bonnor and Ms Sian McGee, and prior to that Ms Georgina Wright.

2. I also acknowledge the assistance of Mr Jeremy Morris SC, Dr Robert Cavanagh and Ms Isabel Reed, appearing on behalf of Ms Folbigg. I also acknowledge other counsel who appeared in the Inquiry – Mr Christopher Maxwell QC for the DPP, Ms Margaret Cunneen SC for Mr Craig Folbigg, Mr Ian Fraser for NSW Ministry of Health, Ms Kate Richardson SC for Dr Allan Cala and Ms Ragni Mathur for Professor John Hilton. I also acknowledge Detective A/Inspector Ryan Coffey, Solicitor Advocate for NSW Police Force.

3. The Inquiry would never have occurred without the assistance of a team provided by the solicitor assisting the Inquiry, the NSW Crown Solicitor. That team was led by Ms Amber Richards, senior solicitor, who not only managed the logistics of the Inquiry, but also made a significant contribution to understanding the complexities of the evidence and in preparing the report. She was ably assisted by Clara Potocki, Jake Marusich, Connie Livanos, Kathleen McKinlay, Valentina Markovina, Deborah Rana and Shaun Towers. I thank the Crown Solicitor's Office for that support.

4. I also thank the Legal Aid Commission for providing the necessary assistance to Ms Folbigg.

5. I wish to express my appreciation to all of the experts who prepared reports for and gave evidence in the Inquiry for sharing their time and expertise. I wish to acknowledge particularly the genetics experts in this regard, who dedicated a large number of hours over many months to conduct and interpret complex genetic testing and present their findings in a way that could be understood by the Inquiry. I also wish to thank those experts that assisted the Inquiry in an advisory capacity, including forensic pathologist Professor Roger Byard, and those who assisted the Inquiry in identifying relevant literature.

6. I also record my thanks to Ms Ann Lambino, Registrar of the Forensic Medicine and Coroner's Court at Lidcombe, for her patience and gracious hosting of the Inquiry through three phases of substantive hearings. Thank you also to Mr Tim Musgrave, Justice IT, without whose efforts the smooth presentation of electronic and audio visual evidence would not have been possible.

7. Finally, I thank my associate, Ms Tonia Wood, for her support and assistance, as always.

Report of the Inquiry into the convictions of Kathleen Megan Folbigg

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Volume 1: Chapter 1

Chapter 1: The Inquiry

Introduction

1. On 21 May 2003 in the Supreme Court of New South Wales, Kathleen Megan Folbigg was found guilty by jury of the following charges in respect of her four children:
    a. the manslaughter of Caleb Gibson Folbigg, aged 19 days, on 20 February 1989;
    b. maliciously inflicting grievous bodily harm upon Patrick Allen Folbigg with intent to do grievous bodily harm, aged four months and 15 days, on 18 October 1990;
    c. the murder of Patrick Allen Folbigg, aged eight months and 10 days, on 13 February 1991;
    d. the murder of Sarah Kathleen Folbigg, aged ten months and 16 days, on 30 August 1993; and
    e. the murder of Laura Elizabeth Folbigg, aged 18 months and 22 days, on 1 March 1999.
2. Ms Folbigg was sentenced on 24 October 2003 to 40 years' imprisonment with a non-parole period of 30 years. This was later reduced on appeal to 30 years' imprisonment with a non-parole period of 25 years.[1]
3. Ms Folbigg has exhausted all avenues of appeal and remains incarcerated at Silverwater Women's Correctional Centre.

Petition

4. On 16 June 2015 the Governor of New South Wales received the petition prepared by the University of Newcastle Legal Centre and barristers Dr Robert Cavanagh, Nicolas Moir and Isabel Reed on behalf of Ms Folbigg ("the Petition").[2] The Petition was made pursuant to s 76 of the *Crimes (Appeal and Review) Act 2001* (NSW) ("the *CAR Act*"), which allows a petition to be made by or on behalf of a convicted person for a review of a conviction or sentence or the exercise of the Governor's pardoning power.
5. The Petition sought for the Governor to direct that an inquiry be conducted by a judicial officer into Ms Folbigg's convictions pursuant to s 77(1)(a) of the *CAR Act*. Section 77(2) of the *CAR Act* provides that the Governor may only direct an inquiry to be conducted if it appears that there is a doubt or question as to the convicted person's guilt, or as to any mitigating circumstances in the case, or as to any part of the evidence in the case.
6. The Petition submitted that further evidence had come to light since Ms Folbigg's unsuccessful appeals against her convictions, such that there was a doubt or question as to her guilt.[3]
7. The Petition relied upon the reports of four experts which were said to contain the fresh evidence:
    a. an undated report by Professor Stephen Cordner AM, Professor of Forensic Pathology (International) at Monash University;

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1 *R v Folbigg* [2005] NSWCCA 23.
2 Petition to the Governor of New South Wales for a review of the convictions of Kathleen Folbigg (26 May 2015).
3 The Petition, p 3.

b. a report dated 1 June 2015 by Professor Michael Pollanen, Chief Forensic Pathologist for Ontario;

c. a report dated 7 April 2015 by Professor Ray Hill, Professor of Mathematics at the University of Salford United Kingdom; and

d. a report dated 18 April 2014 by Dr Sharmila Betts, Clinical Psychologist.

8. The Petition characterised the fresh evidence as falling into two categories: evidence relating to the causes of death of the children, and evidence relating to the use made of Ms Folbigg's diary entries at trial.[4]

9. The Petition submitted that fresh evidence relating to the causes of death of the children leads to a "feeling of disquiet" regarding Ms Folbigg's convictions.[5]

10. In respect of Ms Folbigg's diary entries, the Petition submitted that although Sully J's conclusion on appeal that Ms Folbigg's diary entries were "damning" was rationally available, there is other equally compelling reasoning that allows for an innocent interpretation.[6]

Establishment of the Inquiry

11. On 22 August 2018, pursuant to s 77(1)(a) of the *CAR Act*, the Governor of New South Wales directed ("the Direction") that an inquiry be conducted into Ms Folbigg's convictions for the manslaughter of Caleb, the malicious infliction of grievous bodily harm upon Patrick, and the murder of Patrick, Sarah and Laura ("the Inquiry").[7] The Direction can be found at Annexure A to this Report.

12. The Direction states "it appears that there is a doubt or question as to part of the evidence in the proceedings leading to the conviction of Kathleen Megan Folbigg on 21 May 2003."[8]

13. The Direction goes on to specify:

> *that doubt or question concerns evidence as to the incidence of reported deaths of three or more infants in the same family attributed to unidentified natural causes.*[9]

14. Pursuant to s 81(1)(a) of the *CAR Act* the Governor appointed me, the Honourable Reginald Oliver Blanch AM QC, formerly a judicial officer within the meaning of the *Judicial Officers Act 1986* (NSW), to conduct the Inquiry ("the Judicial Officer").[10]

15. Pursuant to s 81(2)(a) of the *CAR Act* I have the powers, authorities, protections and immunities conferred on a commissioner by Division 1 of Part 2 of the *Royal Commissions Act 1923* (NSW) ("the *RC Act*"). The Direction also conferred on me the powers and authorities conferred on a commissioner by Division 2 of Part 2 of the *RC Act*, except for s 17.[11]

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4 The Petition, p 2.

5 The Petition, pp 27-28, 32-34, 39, 56, 61-62, 68, 70; Exhibit Q, Report of Professor Stephen Cordner (undated) p 90.

6 The Petition, p 76.

7 Exhibit A, Governor of New South Wales, 'Direction pursuant to section 77(1)(a) of the *Crimes (Appeal and Review) Act 2001*' (22 August 2018).

8 Exhibit A.

9 Exhibit A.

10 Former Justice of the Supreme Court of NSW and Chief Judge of the District Court of NSW.

11 Exhibit A, the Direction.

16. Following my appointment as the Judicial Officer, the Crown Solicitor for New South Wales was instructed to act as solicitor assisting the Inquiry. The Crown Solicitor put together a team of legal and administrative staff to assist me in conducting the Inquiry, and instructed three barristers to act as counsel assisting the Inquiry, led by Gail Furness SC.

17. This chapter of the Report sets out the scope and conduct of the Inquiry, and my task as Judicial Officer.

Scope and conduct of the Inquiry

Advertisements and website

18. Following establishment of the Inquiry, advertisements requesting those with relevant information to contact the Inquiry and announcing the details of the first directions hearing were run. The text of each of the advertisements can be found in Annexure B to this report.

19. An Inquiry website was also established at www.folbigginquiry.justice.nsw.gov.au.

Directions hearings and scope of the Inquiry

25 October 2018

20. The first directions hearing was held at 10:00am on 25 October 2018. Counsel assisting, together with representatives of Ms Folbigg, NSW Health and the Commissioner of Police sought and were granted leave to appear before the Inquiry. The Direction and relevant judgments were tendered.[12]

21. Counsel assisting submitted that the scope of the Inquiry should be expanded to include:

    a. any new research or advances in medical science relevant to the causes of death of each child and the cause of the apparent or acute life threatening event ("ALTE") in respect of Patrick;

    b. expert medical opinion as to the causes of death of each child and the cause of the ALTE in respect of Patrick in light of any relevant new research or advances in medical science;

    c. any new research or literature concerning the incidence of reported deaths of three or more infants in the same family attributed to unidentified natural causes; and

    d. any other related expert medical evidence.[13]

22. I agreed such an expansion was appropriate and made an order accordingly.[14] This expansion of the scope of the Inquiry was to ascertain if there was any reasonable possibility that the four deaths and the ALTE could have been from natural causes.

12 December 2018

23. The second directions hearing was held at 10:00am on 12 December 2018. The Director of Public Prosecutions ("DPP") sought and was granted leave to appear. By this stage funding had also been approved to allow Ms Folbigg to instruct Jeremy Morris SC.[15]

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12 Exhibits A and B.

13 Transcript of the Inquiry, 25 October 2018 T5.42-50.

14 Transcript of the Inquiry, 25 October 2018 T1.42-2.1.

15 Up until this point Ms Folbigg had been represented by junior counsel Dr Robert Cavanagh and Isabel Reed.

24. I indicated to Mr Morris SC that if Ms Folbigg wished to give evidence about her diaries and her possession and dispossession of them, then I would be happy for her to be called but that she could not be compelled to attend.[16]

25. I also made a direction regarding Inquiry procedure and set a timetable for the hearing of the evidence relevant to Sudden Infant Death Syndrome ("SIDS")/Sudden Unexpected Death in Infancy ("SUDI"), forensic pathology, cardiology and genetics which required expert reports to be provided two weeks in advance of the relevant hearing.

26. The expert reports of Professors Cordner and Pollanen, the 2018 text *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* edited by Jhodie Duncan and Roger Byard ("Duncan and Byard, 2018") and the exhibits and transcripts from trial were also tendered.[17]

20 December 2018

27. At the third directions hearing at 2:00pm on 20 December 2018, Ms Folbigg's representatives indicated that Ms Folbigg may be willing to give evidence but they did not have final instructions on the matter.

28. In the circumstances I made a further order expanding the scope of the Inquiry to allow Ms Folbigg to give evidence about the diary entries, her possession of the diaries and her disposal of the diaries, should she wish to do so. Her evidence and the cross-examination of her was restricted to those particular issues.[18]

11 February 2019

29. Certainty as to whether Ms Folbigg would or would not give evidence had still not been achieved by the time of the fourth directions hearing on 11 February 2019. In fairness to those other persons with leave to appear who would need to prepare for the giving of such evidence, I made an order that the scope would not include Ms Folbigg's evidence unless the Inquiry was notified in writing by 17 March 2019 that Ms Folbigg wished to give evidence.

30. Representatives of Dr Allan Cala and Professor John Hilton sought and were granted leave to appear before the Inquiry. Dr Cala's representatives made an application for Professor Byard to be compelled to attend to give evidence before the Inquiry. In light of the tendering of Duncan and Byard (2018), Professor Byard's assistance to the Inquiry in an advisory capacity, and the absence of disagreement between the forensic pathologists that myocarditis was a possible cause of death of Laura, I declined that application.

1 April 2019

31. Following the first phase of substantive hearings, a further directions hearing was held at 2:00pm on 1 April 2019 to determine the timetable for receipt of any further expert reports. Representatives of Mr Craig Folbigg sought and were granted leave to appear before the Inquiry.

Summonses issued

32. During the course of the Inquiry, the following summonses were issued to obtain material relevant to the scope of the Inquiry:

    a. NSW Police;

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[16] Pursuant to the Direction I do not have the power under s 17 of the *Royal Commissions Act 1923* to compel witnesses to answer questions or produce documents that may tend to criminate that witness.

[17] Exhibits C, D, E and F.

[18] Transcript of the Inquiry, 20 December 2018 T6.6-10.

b. Office of the DPP;
c. NSW Health Administration Corporation;
d. Sydney Children's Hospital Network;
e. Hunter New England Local Health District;
f. Medicare Services Australia;
g. Justice Health and Forensic Mental Health Network;
h. Legal Aid Commission of New South Wales;
i. Western Sydney Local Health District; and
j. Mr Folbigg.

Engagement of experts

33. At the outset of the Inquiry, those assisting the Inquiry attempted to make contact with relevant witnesses who gave evidence at Ms Folbigg's trial.

34. Forensic pathologists Professor Hilton and Dr Cala were contacted and prepared reports for and gave evidence at the Inquiry. Clinical geneticist Professor Bridget Wilcken AM also prepared a report for the Inquiry, but it was unnecessary for her to give evidence.

35. Professor Peter Berry (paediatric pathologist) and Dr Ian Wilkinson (paediatric neurologist) could not be reached and it is understood they have not practiced for several years. Dr Susan Beal AM (paediatrician) is unwell and Professor Peter Herdson (forensic pathologist) is deceased.

36. Forensic pathologist Professor Roger Byard AO PSM was also contacted by the Inquiry. I note that the text by Duncan and Byard (2018) is the most up to date text on many of the issues relevant to the Inquiry and has been of substantial assistance. Professor Byard indicated he was not prepared to give evidence before the Inquiry, but was willing to act in an advisory role. Professor Byard was accordingly engaged by the Inquiry as an expert advisor in relation to the literature in the areas of forensic pathology and SIDS/SUDI.

37. Those assisting the Inquiry approached Professor Cordner and gave him the opportunity to update or amend his report that was annexed to Ms Folbigg's Petition.[19] Professor Cordner also gave evidence before the Inquiry.

38. Those assisting the Inquiry also approached other experts to provide expert assistance relevant to the scope of the Inquiry. The following experts were engaged by the Inquiry to prepare reports and give evidence:
    a. Professor Rosemary Horne, infant sleep and SIDS specialist;
    b. Professor Dawn Elder, paediatrician;
    c. Dr Alison Colley, clinical geneticist;
    d. Dr Michael Buckley, genetic pathologist and molecular geneticist;
    e. Professor Edwin Kirk, genetic pathologist, clinical geneticist and metabolic specialist;
    f. Professor Matthew Cook, immunologist;
    g. Professor Jonathan Skinner, paediatric cardiologist and electrophysiologist;
    h. Associate Professor Michael Fahey, paediatric neurologist; and
    i. Dr Michael Giuffrida, forensic psychiatrist.

[19] In the Inquiry hearings, Professor Cordner confirmed he did not take up that opportunity. See Transcript of the Inquiry, 19 March 2019 T66.46-67.8.

39. The Inquiry also met with Professor Caroline Blackwell, microbiologist. Professor Blackwell was unable to assist the Inquiry due to her personal circumstances at that time, but recommended that the Inquiry approach Professor William Rawlinson AM, virologist. Those assisting the Inquiry met with Professor Rawlinson and he prepared a short written statement setting out his opinion regarding the viability of testing the children's samples for viruses. He was not called to give evidence.

40. The following experts were engaged by Ms Folbigg's representatives and gave evidence before the Inquiry:

    a. Professor Johan Duflou, forensic pathologist;

    b. Professor Robert Clancy AM, mucosal immunologist;

    c. Professor Caroline Blackwell, microbiologist;

    d. Professor Carola Garcia de Vinuesa, immunologist;

    e. Dr Todor Arsov, visiting fellow at the Centre for Personalised Immunology at the Australian National University; and

    f. Professor Monique Ryan, paediatric neurologist.

41. Ms Folbigg's representatives also obtained reports from the following experts who were not called to give evidence:

    a. Professor Paul Goldwater, microbiologist;

    b. Associate Professor Hariharan Raju, cardiologist and electrophysiologist;

    c. Dr Kathryn Waddell-Smith, genetic cardiologist; and

    d. Dr Michael Diamond, psychiatrist.

42. A list of the qualifications and involvement of each of the experts, both at trial and in the Inquiry, may be found at Annexure C of this report. A list of those experts who prepared reports and statements in the Inquiry may be found at Annexure D.

Genetic and cardiac testing

43. The length of the Inquiry was significantly governed by the time it took to obtain samples for genetic testing, to extract DNA from those samples, obtain genetic data and receive expert advice about the results. However, in my view it was important to do this because clearly a common genetic defect would be the most likely natural cause of the five events being examined, if such a defect existed.

44. The details of the genetic testing conducted by teams engaged by the Inquiry and by Ms Folbigg's representatives ("the Sydney Team" and "the Canberra Team", respectively) and the results of that testing are set out in Chapter 7 of this report.

45. Two sets of a range of cardiac tests were also performed on Ms Folbigg at the request of the Canberra and Sydney teams.

Non-publication directions

46. During the course of the Inquiry I made directions relating to the publication and non-publication of the following material:

    a. the genetic sequencing data, information resulting from the interpretation of that data (together "the information"), and any report given to the Inquiry about the information and oral evidence about those reports;

b. health records of Ms Folbigg produced to the Inquiry by NSW Justice Health, any report given to the Inquiry about those health records, and the oral evidence given before the Inquiry about these reports; and

c. at the request of Ms Folbigg's representatives an exchange between myself and Ms Folbigg's representatives on 17 April 2019 about a psychiatric report obtained by Ms Folbigg's representatives.

47. I also made specific directions regarding media access and publication.

Substantive hearings

48. The substantive hearings were held in Courtroom 1 of the Forensic Medicine and Coroner's Court Complex in Lidcombe, New South Wales in three separate phases from 18 to 23 March 2019, 15 to 17 April 2019 and 29 April to 1 May 2019. Where possible, the expert evidence was heard concurrently to enable the experts to hear the responses of their colleagues.

49. A live stream of the audio from the Inquiry was broadcast on the Inquiry website and remains available.

50. All hearings were open to the public.

Those with leave to appear and cross-examine

51. The following persons sought and were granted leave to appear at the substantive hearings and to cross-examine witnesses:

a. Counsel assisting the Inquiry, Gail Furness SC, with Ann Bonnor and Sian McGee, instructed by Amber Richards on behalf of the NSW Crown Solicitor;

b. Ms Folbigg, represented by Jeremy Morris SC, Dr Robert Cavanagh and Isabel Reed, instructed by Stuart Gray of Cardillo Gray Partners;

c. NSW Health, represented by Ian Fraser, instructed by Blaise Lyons;

d. Dr Allan Cala, represented by Kate Richardson SC, instructed by Paulina Moncrieff of Norton Rose Fulbright;

e. Professor John Hilton, represented by Ragni Mathur, instructed by Tony Mineo of Avant Mutual;

f. The DPP, represented by Christopher Maxwell QC, instructed by Rachel Swift; and

g. Mr Folbigg, represented by Margaret Cunneen SC, instructed by Danny Eid of Danny Eid lawyers.

52. Counsel assisting and Ms Folbigg's representatives appeared throughout the Inquiry; representatives of NSW Health, Dr Cala and Professor Hilton appeared only at the first phase of hearings (evidence relevant to SIDS/SUDI, forensic pathology and immunology); representatives of the DPP and Mr Folbigg appeared only at the third phase of hearings (relevant to Ms Folbigg's diaries).

Exhibits, witness list and glossary

53. A list of the exhibits tendered in the Inquiry can be found at Annexure E to this report.

54. A list of the witnesses who gave evidence before the Inquiry and the transcript references to that evidence may be found at Annexure F. A glossary of medical terms is at Annexure G.

After the close of the evidence

55. The evidence in the Inquiry closed on 1 May 2019.[20] On 21 June 2019 Professor Vinuesa sent to the Inquiry a further statement in relation to a specific genetic variant which annexed a paper published in June 2019. The Inquiry sought the views of the Sydney Genetics team and Professor Skinner in relation to the new information available, and a supplementary report was prepared. On 12 July 2019 the Inquiry received a further report from Professor Vinuesa that had been "reviewed and endorsed" by other experts.

Submissions

56. At the conclusion of the final phase of the substantive hearings, the timetable for the provision of legal submissions was confirmed.

57. Counsel assisting provided written submissions to those with leave on 17 May 2019.

58. Representatives of Mr Folbigg and the DPP provided written submissions to the Inquiry on 24 May 2019.

59. Submissions on behalf of Ms Folbigg were due on 31 May 2019. Her representatives sought and were granted an extension of one week and provided written submissions to the Inquiry on 7 June 2019.

60. Submissions on behalf of Professor Hilton, Dr Cala and NSW Health were also due on 31 May 2019. However, in the event they did not file full written submissions, the representatives of Dr Cala sought leave to file short submissions in reply to the submissions of Ms Folbigg's representatives insofar as they were relevant to the interests of Dr Cala. I granted leave to respond in this way to Professor Hilton, Dr Cala and NSW Health, with any submissions in reply to be provided no later than 14 June 2019. Representatives of Dr Cala provided written submissions in reply on 14 June 2019. I granted an extension to the representatives of Professor Hilton of two working days and written submissions in reply were provided on his behalf on 18 June 2019. No submissions were received on behalf of NSW Health.

61. Given the relevance of the June 2019 genetics paper provided to the Inquiry by Professor Vinuesa, I received further written submissions on this issue from counsel assisting on 8 July 2019 and from Ms Folbigg on 12 July 2019.

Tasks of the Judicial Officer

Inquiries under Part 7 of the *CAR Act*

62. Part 7 of the *CAR Act* provides a mechanism in New South Wales for review of convictions otherwise than by appeal, including by the conduct of an inquiry into a conviction or sentence. An inquiry directed to be conducted by a judicial officer under s 77(1)(a) of the *CAR Act* under Part 7 is an inquiry "into the conviction or sentence".

63. Pursuant to s 77(2) of the *CAR Act*, a direction for an inquiry may be predicated upon the existence of a doubt or question as to the guilt of the convicted person. Consistently with the approach taken in previous inquiries of this nature, including under predecessor legislation, however, I do not consider that the Direction involves a revival of the presumption of innocence in favour of the convicted person.[21]

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[20] At the close of the substantive hearings, at the request of Ms Folbigg's representatives, the Judicial Officer directed that Ms Folbigg's representatives had until 7 May 2019 to seek the tender of any further documents. Further documents were tendered at this point.

[21] *Report of the Inquiry into the conviction of Patrick John O'Connor* (Ducker DCJ, 21 February 1995) (*"O'Connor Inquiry"*) p 18 (on basis of Wood J in *Report of the Inquiry into the convictions of Timothy Edward Anderson, Paul Shawn Alister and Ross Anthony Dunn* (Wood J, 14 May 1985) ("*Anderson Inquiry*") p 60); *Report of the Inquiry into the conviction of Andrew Kalajzich* (Slattery J, 30 May 1995) (*"Kalajzich Inquiry"*) p 20; *Report of the Inquiry into the conviction of Johann Pohl* (McInerney J, 2 November 1973) ("*Pohl Inquiry*") p 5.

64. Nor is such an inquiry a retrial on the basis of the evidence before it.[22] It does not impose any onus on the Crown to produce evidence to remove the doubt that gave rise to the inquiry and re-establish guilt.[23] Similarly, it does not impose any onus on the convicted person to establish that the conviction was wrongly procured.[24]

65. It is the convictions which are the subject of an inquiry. Therefore, an inquiry must commence with the fact that a conviction has been recorded and that questions or doubts have been raised sufficient to justify the Governor directing a judicial officer to conduct an inquiry, and to summon and examine on oath all persons likely to give material information. As such, I do not consider my role to be akin to that of a judge and jury in a retrial.[25]

66. An inquiry is not confined to an investigation of the questions or doubts raised in a petition, or to an investigation of new evidence. It may re-examine evidence in the trial including any matters previously dealt with on appeal.[26] Further, an inquiry may consider any information that may throw light on the convicted person's guilt, whether that information is favourable or unfavourable to the convicted person.[27] It is not fettered by tactical or forensic decisions at trial, or by the way the Crown or defence cases were conducted.[28]

67. My overall task is therefore to consider the evidence at the trial and the conduct of the trial, in light of the further evidence and submissions received in the Inquiry, in order to determine whether, overall, there is a reasonable doubt as to Ms Folbigg's guilt or as to any matter that may have affected the nature or severity of her sentence.[29]

68. It is on this basis that the Inquiry was conducted and that this report is made.

Outcome of an inquiry: s 82 of the *CAR Act*

69. Pursuant to s 82(1)(a) of the CAR Act, on completion of the Inquiry I must cause a report on the results of the Inquiry to be sent to the Governor of New South Wales. The report must incorporate a transcript of the depositions given in the course of the Inquiry.

70. Section 82(2) provides:

> *The judicial officer may also refer the matter (together with a copy of the report) to the Court of Criminal Appeal:*
>
> > *(a) for consideration of the question of whether the conviction should be quashed (in any case in which the judicial officer is of the opinion that there is a reasonable doubt as to the guilt of the convicted person), or*
> >
> > *(b) for review of the sentence imposed on the convicted person (in any case in which the judicial officer is of the opinion that there is a reasonable doubt as to any matter that may have affected the nature or severity of the sentence).*

71. It is clear that s 82(2) confers on me a discretion to refer the matter to the Court of Criminal Appeal if I form the opinion/s as set out in s 82(2)(a) or (b).

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22 *O'Connor Inquiry,* p 18; *Anderson Inquiry,* pp 60-62.
23 *Anderson Inquiry,* p 60.
24 *Anderson Inquiry,* p 61.
25 *See Anderson Inquiry,* p 61.
26 *Anderson Inquiry,* pp 66-67.
27 *Anderson Inquiry,* pp 66-67.
28 *Anderson Inquiry,* pp 68-70.
29 *O'Connor Inquiry,* p 18 (on basis of Wood J in *Anderson Inquiry*).

Report on the results of the Inquiry

72. Section 82(1)(a) does not specifically confine what "the results" of an inquiry might entail, for the purposes of the report.

73. Judicial officers in previous inquiries have not considered the results to be restricted to conclusions regarding the questions or doubts as to guilt or as to facts potentially affecting the sentence raised in the direction for an inquiry.[30] The legislation does not appear to impose any bar to reporting conclusions as to whether the judicial officer finds there to have been any procedural irregularity in the trial process.[31]

Reasonable doubt as to the guilt of the convicted person

74. Pursuant to s 82(2)(a) I may refer the matter to the Court of Criminal Appeal if I am of the opinion that there is a reasonable doubt as to the guilt of Ms Folbigg.

75. "Reasonable doubt" should be ascribed its ordinary meaning; that is, a doubt which I consider reasonable. It does not mean any doubt, no matter how slight.[32]

76. According to authority, previous inquiries under Part 7 of the *CAR Act* and predecessor provisions, "guilt" in this context means guilt in fact, but also includes guilt as established by each conviction. As such, a reasonable doubt as to guilt may arise from a procedural irregularity or error in the trial process.[33] If the question or doubt concerns a possible miscarriage of justice or involves the possibility that one or more of the convictions were improperly obtained due to an error in the trial process, I am to explore whether or not there was a mishap and report my conclusion as to its occurrence and significance in relation to guilt.[34]

77. It is for me to form my own concluded opinion as to whether there is a reasonable doubt, according to well-known principles applicable to this concept. It is not for me to determine whether a jury, properly instructed, might have, or would have, a reasonable doubt.

78. Reasonable doubt may be formed whether or not the threshold doubts or questions that gave rise to the Inquiry have been resolved and found to have had no substance, or the doubts or questions raised remain unresolved.[35] Because an inquiry is not confined to an investigation of the questions or doubts raised in a petition, or of new evidence, and may consider any information that may throw light on the convicted person's guilt, a reasonable doubt may be based upon any doubt emerging at the inquiry up to the time of presentation of the report.

79. That is, I may form a reasonable doubt as to guilt regardless of my finding specifically in relation to the doubt or question that gave rise to the Inquiry. Equally, notwithstanding the existence of doubts or questions in relation to parts of the evidence by reliance on other parts of the evidence, I may be satisfied that there is no reasonable doubt as to Ms Folbigg's guilt.

80. Further, because I am not bound by the rules of evidence in forming an opinion as to the existence of a reasonable doubt, I may have regard to all of the information and evidence received by the Inquiry. That said, I may give varying weight to different pieces of evidence received in the Inquiry according to my assessment of matters such as the credibility of witnesses, the soundness of opinions offered by experts or the nature of the evidence.

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30 *Suey Inquiry,* [5.12].

31 *Suey Inquiry,* [5.12].

32 *The Queen v Dookheea* [2017] HCA 36, [34].

33 *Eastman v DPP (ACT)* (2003) 214 CLR 318 (McHugh J, Gummow J agreeing at [12]-[15], [21]-[23]); *Sinkovich v Attorney General of NSW* [2013] NSWCA 383, [27].

34 *Anderson Inquiry,* pp 63-64; *Pedrana Inquiry*, [6.4]; *Suey Inquiry*, [5.12]. See also *Kalajzich Inquiry; Inquiry into the conviction of Alexander Lindsay* (Loveday J, 29 July 1991); *Inquiry into the convictions of Grahame Andrew Rogers* (Kinchington DCJ, 4 February 1999).

35 *Anderson Inquiry,* pp 63-64.

Reasonable doubt as to sentence

81. Pursuant to s 82(2)(b) I may also refer the matter to the Court of Criminal Appeal if I am of the opinion that there is a reasonable doubt as to any matter that may have affected the nature or severity of the sentence imposed on Ms Folbigg.

82. A convicted person's role in or degree of responsibility for the acts perpetrated are examples of matters that may affect the nature and severity of the sentence handed down.[36]

Counsel assisting's submissions

83. Counsel assisting made submissions to the effect of the above.

Ms Folbigg's submissions

84. Below are my views in relation to specific submissions made on behalf of Ms Folbigg.

Effect of direction

85. Ms Folbigg submitted that in light of the Direction for the Inquiry made pursuant to s 77 of the *CAR Act*, there is a doubt and that doubt needs to be assessed against the criminal standard.[37]

86. In my view, and given the statutory context and analysis conducted by previous inquirers discussed above, it would be an error to take the Direction for the Inquiry as itself raising a reasonable doubt as to guilt. The statutory threshold for an inquiry in s 77 imposes quite a different standard and includes the "appearance" of "questions". It is not confined to guilt but includes any mitigating circumstance and any part of the evidence. The whole purpose of an inquiry is to inquire into the initial appearance of a doubt; it would turn that function on its head if Ms Folbigg's submission was accepted.

Presumptions

87. Ms Folbigg submitted that the proposition that the Inquiry does not involve a revival of the presumption of innocence should be rejected, on the basis that, were it intended to be removed or excluded by the legislature, one would expect clear statutory language to this effect.[38] Ms Folbigg submitted that in the event I rejected that proposition, the Inquiry should not approach its task with a presumption of guilt.[39]

88. For reasons set out above, in my view, a Part 7 inquiry is premised neither upon a presumption of innocence nor one of guilt. In the latter respect, I agree with Ms Folbigg's submissions.

89. In view of the language, context and purpose of the statute governing an inquiry, I would expect that if there were to be any particular presumption, the statute would make that clear. It does not. In my view this is entirely consistent with the function of reviewing the conviction on behalf of the Executive by inquiring into relevant matters upon which the conviction is based, in order to produce a report of the results and determine whether there is, overall, a reasonable doubt as to guilt.

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36 *Suey Inquiry,* [19.22], [20.2].
37 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [15].
38 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [30].
39 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [31].

90. I have approached my analysis of the evidence in this Inquiry, and my findings, without applying a presumption of innocence or one of guilt. In a number of instances in her submissions, Ms Folbigg asserted that evidence or submissions received in the Inquiry were premised upon a presumption of guilt.[40] I do not agree. Testing propositions by cross-examination, for instance, does not itself amount to a revival of a presumption. Nonetheless, I have borne the tenor of Ms Folbigg's submissions in mind in ultimately weighing the evidence.

Issues of onus

91. Ms Folbigg submitted that once a doubt is identified it must be demonstrated why that doubt is unreasonable and that:

> *to comply with the s. 77 direction, the obligation is upon the Crown to adduce all relevant evidence on the question of guilt, rather than proceeding with some pre-determined conception as to the evidence. If it fails to do so, then the capacity of the Crown to assert its compliance with the direction of the Governor is open to question.*[41]

92. Again, I do not consider this submission to be consistent with the *CAR Act*. Questions of onus are foreign to this type of inquiry.[42] If a doubt is identified either by a direction or in the course of an inquiry, it is to be investigated as part of the inquiry and reported on to the extent that it remains after further examination.

93. Ms Folbigg appears to have suggested in a number of instances that the Crown bore some onus in the Inquiry.

94. There is no obligation under the *CAR Act* upon the Crown, in the sense that the interests of the Crown are represented by the DPP, to adduce or produce evidence (unless summonsed or required to do so by the Inquiry) on the question of guilt.

Task of the Judicial Officer in relation to the convictions

95. Ms Folbigg submitted that my task is similar to that of the Court of Criminal Appeal and I should not agglomerate all five charges and consider the matter as one.[43]

96. In the *Anderson Inquiry*, Wood J (as his Honour then was) distinguished the functions of an inquirer under predecessor provisions from the appellate role of the Court of Criminal Appeal. His Honour stated:

> *[I]f I were to conclude at the end of the Inquiry that at the trial there was a miscarriage of justice in some respect, yet the jury would certainly have returned the same verdict if the matter complained of had not arisen (R v McDonald, Court of Criminal Appeal 23rd February 1984), Marie v The Queen 52 ALJR 631 at 635), I do not believe that I could discharge my function by a simple conclusion that there was no doubt or question. Unlike the Court of Criminal Appeal, I do not believe that I could myself have resort to a process akin to an application of the proviso to Section 6(1) of the Criminal Appeal Act 1912. In such a case I consider that I would have to report in relation to the questions or doubts concerning the matter or matters involving a miscarriage of justice, and for the benefit of the Executive express my opinion as to their significance for the finding of guilt.*

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40 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [31], [231]; Part B, [105]; Part C – Caleb, [101]; Part D, [27]-[29], [120], [168], [297], [307].

41 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [33(b)].

42 *Anderson Inquiry,* p 61.

43 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [22], [24].

> *I also do not consider that I am constrained within the well recognised tests applied in the consideration of criminal appeals concerning, for example, fresh evidence: Ratten v The Queen (1974) 131 CLR 510; and R v Eustace (Court of Criminal Appeal, 18 June 1981); or concerning the failure to object to evidence in the Crown case: R v Visser (1983) 3 NSWLR, 240; or concerning error in the presentation of the defence case: Re Knowles (Supreme Court of Victoria 25 May 1984; and Green v The King (1939) 61 CLR 167 at 175.*
>
> *Further, it does not seem appropriate to decide the question whether there was a doubt or question arising out of an error in the trial process by an application of the test, established in Mraz v The Queen (1955) 93 CLR 493 (at 514), that is whether by reason of the error a petitioner may have lost a chance of acquittal that was fairly open to him. This would be a material aspect, but it would not conclude my inquiry.*[44]

97. I respectfully agree. Particularly in relation to the last paragraph, considerations that may arise on an appeal may be material in an inquiry but would not conclude the inquiry. I am not constrained by the well-recognised tests applied in the consideration of criminal appeals, such as in respect of fresh evidence.[45] The fact that some issues have previously been the subject of consideration and decision as part of an appellate process is not determinative of my opinion (although I may consider this to be relevant).[46] Following its determination, an appellate court has the ability to deal directly with the convicted person, for example by releasing them or reducing their sentence. These powers are not available to me.

98. I agree with Ms Folbigg's submission, however, that her convictions ought not be considered as one. All of her convictions having been the subject of the Direction, I must inquire into each conviction. For reasons set out in Chapter 3, I consider that the appropriate approach is to apply principles orthodox to a circumstantial criminal case where multiple counts are involved, and also apply accepted principles as to whether coincidence and tendency reasoning ought to be employed and, if so, how.

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44 *Anderson Inquiry*, pp 67-68.

45 *Anderson Inquiry,* p 67.

46 *Anderson Inquiry,* p 68.

Report of the Inquiry into the convictions of Kathleen Megan Folbigg

July 2019

Volume 1: Chapter 2

Chapter 2: Overview of the Folbigg family

Introduction

1. This chapter of the Report provides a brief overview of the Folbigg family and the events leading to Ms Folbigg's trial.

Kathleen Megan Folbigg

Early life

2. On 14 June 1967, Kathleen Megan Folbigg was born Kathleen Megan Britton to Kathleen and Thomas Britton in Balmain, New South Wales.[1]

3. Her mother was murdered by her father in December 1968. Mr Britton stabbed his wife several times following a domestic dispute and served approximately 15 years in prison before being deported to England.[2] At the time of her mother's death Ms Folbigg was 18 months old.[3]

4. Ms Folbigg was then cared for by Mr and Mrs Platt, her mother's sister and her husband, in western Sydney.[4] Ms Folbigg had stayed with Mr and Mrs Platt, who also lived with Ms Folbigg's maternal grandmother, for extended periods prior to her mother's death.[5]

5. On 9 January 1969, Ms Folbigg was made a ward of the State and officially placed in the care of Mr and Mrs Platt.[6] She was known to the Platts as "Lisa."[7]

6. In May 1970, during a home visit Mrs Platt reported that the family was experiencing difficulties with Ms Folbigg's behaviour.[8] She was said to be aggressive, preoccupied with her genitals and masturbation, and had difficulty learning the basic requirements of hygiene, acceptable manners and behaviour.[9]

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1 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 4; Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 2; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) pp 1, 11.

2 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 12; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 11; Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 3; 3 April 2003 T191.7-15.

3 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 11.

4 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 2.

5 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 2; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 26.

6 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 2; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 26; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 15.

7 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 2; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 26

8 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 2; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 15; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 27.

9 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 2; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 27.

7. Ms Folbigg was assessed at the Yagoona Child Health Centre.[10] The medical officer there considered it likely that she had been abused by her father during infancy, and that Mrs Platt senior was a "destructive influence" on Ms Folbigg.[11]

8. Mrs Platt reported in June 1970 that Ms Folbigg's behaviour was deteriorating and causing difficulties in her marriage.[12] On 11 June 1970, Mr and Mrs Platt confirmed that they were no longer able to care for Ms Folbigg.[13]

9. On 29 July 1970, Ms Folbigg underwent psychological and educational assessment at Bidura.[14] She was assessed as having an IQ of 77, which was classified as "borderline retarded".[15] However, this was noted to be a "doubtful assessment" as Ms Folbigg did not respond to conversation and was restless and inattentive.[16] She was found to be a "very disturbed little girl" who exhibited numerous behavioural difficulties.[17]

10. On 12 September 1970, Ms Folbigg was placed in the care of a Mr and Mrs Marlborough.[18] This placement worked well: a strong relationship developed between the Marlboroughs and Ms Folbigg, and they expressed an interest in adopting her.[19]

11. It was generally accepted that Ms Folbigg presented "as a very happy well adjusted girl" who was very much a part of the family.[20] In 1983, Ms Folbigg changed her surname to Marlborough.[21]

Education

12. Ms Folbigg attended Kotara Public Primary School near Newcastle and later Kotara High School.[22]

13. In year two Ms Folbigg was formally assessed as having an IQ of 110 and her school attendance and behaviour was reportedly good.[23] In year five she was described as being "inattentive, disruptive and defiant".[24]

14. Ms Folbigg appeared before Worimi Children's Court in 1982 for shoplifting.[25]

15. Ms Folbigg successfully completed her Year 10 School Certificate and trial Higher School Certificate exams, but left school approximately six months before completing the Higher School Certificate in Year 12.[26] Ms Folbigg gave evidence that this occurred in the context of a breakdown of her relationship with her foster mother, rather than anything to do with her educational achievements.[27]

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10 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 34; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 15.
11 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 2; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 27.
12 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 2; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 27; Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 5.
13 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 2; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 27.
14 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 2; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 27.
15 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 2; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 27.
16 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 2; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 27.
17 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 3; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 27.
18 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 2.
19 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 3; Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 5.
20 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 4.
21 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 3.
22 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) pp 6, 9.
23 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 34; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) o 17.
24 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 35; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 17.
25 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 35; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 17.
26 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 13.
27 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 9; Transcript of the Inquiry, 1 May 2019 T790.34-46.

Relationships

16. Ms Folbigg reported remembering little of her early years, prior to growing up in the Marlborough family home.[28] She described Mrs Marlborough as controlling to an excessive degree, with a focus on household chores.[29] She said she had an isolated childhood with few friends and restricted social activities outside of the family.[30] She also reported being physically punished by her foster mother for misbehaving, such as being hit with a feather duster handle, a wooden spoon and a belt.[31] She described Mr Marlborough as a good provider but a "shadow in the background" and a "very closed book".[32]

17. Ms Folbigg described herself as a "lone wolf" at school until she entered her first relationship at age 15, which lasted two years.[33] She said that, at that stage, she was able to join her boyfriend's peer group and she made lasting friendships with three or four girls from that group.[34]

18. Ms Folbigg left home at the age of 17 following a breakdown in her relationship with Mrs Marlborough.[35] She said that Mrs Marlborough was unpredictable, moody and controlling of her social life.[36] Mrs Marlborough also didn't approve of her boyfriend at the time.[37]

19. Ms Folbigg then met Craig Folbigg on the dance floor of a disco at age 18.[38] Mr Folbigg came from a family of eight children.[39] His mother had died when he was 15.[40]

20. Mr Folbigg was five years older than Ms Folbigg and worked as a forklift driver at BHP.[41] Ms Folbigg described Mr Folbigg as "extremely charming" with the "gift of the gab" and said that she thought of him as a "knight in shining armour" with a good job and good income.[42]

21. Mr and Ms Folbigg were married in 1987 when Ms Folbigg was aged 20 and Mr Folbigg aged 25.[43]

Married life

22. By 1988 Ms Folbigg was employed as a waitress at an Indian restaurant and Mr Folbigg was in a clerical role at BHP.[44] Together they rented a flat in Georgetown, Newcastle.[45]

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28 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 5; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 12.
29 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 6.
30 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 6; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 12.
31 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 7.
32 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 7.
33 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) pp 7-8; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 13.
34 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 8.
35 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 9.
36 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) pp 7-9; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 13.
37 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 13.
38 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) pp 9-10; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 13.
39 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 10.
40 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 10.
41 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) pp 8-9.
42 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 13.
43 1 April 2003 T28.25-35
44 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 11.
45 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 11.

23. Noting that Mr Folbigg came from a large family, Ms Folbigg said that she considered it an "inevitable and natural process" that they would have children.[46] They stopped contraception and achieved pregnancy without difficulty.[47]

Caleb Gibson Folbigg

24. Ms Folbigg recalled enjoying being pregnant for the first time and described herself as being protective of the pregnancy.[48] She stopped Mr Folbigg from smoking indoors and improved her diet.[49] Mr Folbigg said that he never smoked in the house again, and never smoked around any of the children in a confined space.[50]

25. It was a healthy pregnancy, but a difficult labour.[51] Ms Folbigg had an epidural anaesthetic and a forceps delivery and was unable to hold her new baby for some time.[52] Caleb Gibson Folbigg was born at 40 weeks on 1 February 1989 at a healthy weight.[53] Ms Folbigg was 21 years old at the time and described herself as feeling completed, with a husband, home and baby.[54]

26. Two of Ms Folbigg's school friends provided support to Ms Folbigg, and her foster parents visited occasionally.[55] However, it was mainly Mr Folbigg's family who were present and provided assistance following the birth of Caleb.[56]

27. Ms Folbigg reported that she was unable to breastfeed Caleb as she had "inverted nipples."[57] She noted that "short of his feedin' problem that he had, he sort of didn't have a, didn't have sniffles or colds or any of that sort of thing".[58]

28. Caleb died on 20 February 1989 when he was 19 days old.

29. The autopsy report and death certificate gave the direct cause of death as SIDS.[59]

Patrick Allen Folbigg

30. Near the end of 1989 Mr and Ms Folbigg discussed having further children, and Ms Folbigg soon fell pregnant.[60]

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46 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 11.
47 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 11.
48 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 13.
49 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 13.
50 10 April 2003 T530.24-35.
51 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 13.
52 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 13.
53 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 4; Exhibit H, Forensic pathology tender bundle, Neonatal record of Caleb (5 February 1989).
54 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 13.
55 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 13.
56 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 13.
57 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 14.
58 Exhibit E, ERISP of Kathleen Folbigg Q38.
59 Final autopsy report of Caleb (9 May 1989) p 9; Death certificate of Caleb (20 February 1989)
60 2 April 2003 T106.45-50, T107.13-21; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 15.

31. Ms Folbigg was anxious about reducing any SIDS-associated risks for her new baby and bought brand new bedding and carefully prepared the nursery for its arrival.[61] She and Mr Folbigg also set about doing renovations to their house as they had been told that SIDS could be associated with low socioeconomic status and housing issues.[62]

32. Patrick Allen Folbigg was born on 3 June 1990 at 39 weeks and a healthy weight after an uneventful delivery.[63] Ms Folbigg was aged 22 at the time.[64]

33. In light of Caleb's death, Patrick underwent a sleep study when he was one and a half weeks old, which was "entirely normal".[65] Further tests, including an ECG and a barium test for reflux, were also normal.[66]

34. Mr Folbigg returned to work shortly after the birth and Ms Folbigg described herself as being fearful and said that she tried to cope by establishing a routine.[67]

35. On 18 October 1990, at age four months and 15 days, Patrick had an ALTE.

36. Mr and Ms Folbigg were told that Patrick had suffered brain damage with visual impairment and seizures. Patrick was admitted to hospital again on 4 November, 14 November and 22 December 1990 following further seizures. Ms Folbigg said she accepted that it was her duty to care for him. She said it was hard work but that she and Mr Folbigg were so relieved he had survived.[68] She noted that she had a lot of family support, but their marriage became somewhat strained after the ALTE as 99 per cent of her time was spent looking after Patrick.[69]

37. On 13 February 1991 Patrick died.

38. His death certificate recorded the cause of death as asphyxia due to airway obstruction (one hour) and epileptic fits (four months).[70] The autopsy report recorded a diagnosis of encephalopathic disorder leading to intractable seizures, the underlying cause of which was not determined, and cardiac arrest at home.[71]

Post Patrick's death

39. Ms Folbigg reported that she was severely depressed following Patrick's death and had no drive to do anything. She felt she had worked so hard to ensure that this would not happen and that her preparations were not good enough or that she must have made a mistake in her care of Patrick. She felt that she was not a good person.[72]

61 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 15.
62 2 April 2003 T106.52-107.11.
63 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 5; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 15.
64 Exhibit H, Forensic pathology tender bundle, Birth certificate of Patrick (17 January 2000).
65 14 April 2003 T587.32-588.3.
66 14 April 2003 T512.45-55.
67 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 15.
68 Exhibit E, ERISP of Kathleen Folbigg Q149.
69 Exhibit E, ERISP of Kathleen Folbigg Q466.
70 Exhibit H, Forensic pathology tender bundle, Death certificate of Patrick (13 February 1991).
71 Exhibit H, Forensic pathology tender bundle, Autopsy report of Patrick (14 February 1991) p 3.
72 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 16.

40. Ms Folbigg responded to the situation by focussing on external events and challenges. She decided that they should sell their Mayfield house and they moved to Thornton.[73] Mr Folbigg secured a steady car sales job and Ms Folbigg commenced employment at the retail store BabyCo.[74] Following much discussion, they decided to try for another child.[75]

Sarah Kathleen Folbigg

41. Ms Folbigg described becoming a perfectionist during her pregnancy with Sarah and became preoccupied by decreasing her food intake and increasing her fitness.[76]

42. Her pregnancy was uneventful and Sarah Kathleen Folbigg was born at term on 14 October 1992 after an uncomplicated delivery.[77] She weighed 3,020 grams. Ms Folbigg was aged 25 years.[78]

43. Sarah had a sleep study performed when she was three weeks of age, which was normal.[79]

44. When she was taken home, Sarah had an apnoea blanket and an apnoea alarm, although their use ceased before her death.[80]

45. Ms Folbigg described having trouble attaching to and bonding with Sarah until she was at least six months old as she was terrified of losing her. She also struggled to get Mr Folbigg to "step up" with sharing childcare responsibilities.[81]

46. Mr Folbigg said that Ms Folbigg got very frustrated with Sarah and growled at her from time to time, particularly when it came to enforcing bedtime. On the evening of 29 August 1993, he said that he saw Ms Folbigg growling at Sarah and patting her bottom hard. Ms Folbigg told Mr Folbigg to "fuck off" but a few minutes later she came in to the lounge room and "threw" Sarah at him, saying, "you fucking deal with her", before storming off.[82]

47. Sarah died on 30 August 1993 aged 10 months and 16 days.[83]

48. The autopsy report and death certificate gave the direct cause of death as SIDS.[84]

Post Sarah's death

49. Ms Folbigg said that following Sarah's death Mr Folbigg became severely and deeply depressed and she felt that she could not help him.[85]

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73 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 17; Exhibit E, ERISP of Kathleen Folbigg Q508-509.
74 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 17; 2 April 2003 T117.39-50.
75 Exhibit E, ERISP of Kathleen Folbigg Q261; 2 April 2003 T118.20-52.
76 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 18.
77 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 18.
78 Exhibit H, Forensic pathology tender bundle, Perinatal record of Sarah (undated).
79 14 April 2003 T588.16-589.20.
80 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 5; Exhibit E, ERISP of Kathleen Folbigg Q321, 692; 2 April 2003 T119.53-58, T126.47-127.14.
81 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 19; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 6.
82 2 April 2003 T126.9-127-54.
83 Exhibit H, Forensic pathology tender bundle, Death certificate of Sarah (17 January 2000).
84 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Sarah (25 November 1993).
85 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 20.

50. She decided to sell their home at Thornton as she wanted to run away from the environment where Sarah had died. Mr and Ms Folbigg bought a home at Cardiff, which was closer to Mr Folbigg's family.[86]

51. Ms Folbigg gained weight and said she felt severely insecure, unattractive and rejected by Mr Folbigg. She said this was when their relationship reached "rock bottom" and there were several periods of short separations.[87]

52. Mr and Ms Folbigg rebuilt their relationship. They initially rented a property at Singleton before purchasing a house there and deciding to try for another child. Ms Folbigg became pre-occupied with her appearance and obsessed with attending the gym.[88]

Laura Elizabeth Folbigg

53. Ms Folbigg's fourth pregnancy at age 30 was also uneventful, although she fractured her coccyx post-delivery.[89] Laura Elizabeth Folbigg was born on 7 August 1997 at full term and a healthy weight.[90]

54. At 12 days old Laura underwent full biochemical, blood and metabolic investigations, which were normal. A sleep study showed mild central apnoea and no obstructive apnoea which corrected itself by February 1998.[91]

55. Laura was monitored by a Corometrics home cardiorespiratory monitoring device, which was designed to record and download breathing and heart information during her sleep, for about 12 months without problems. Mr Folbigg was reassured by its presence, but Ms Folbigg found it created anxiety for her because there were repetitive false alarms. They decreased the use of the monitor after the first six months.[92]

56. Ms Folbigg said she was terrified of losing Laura for the first 12 months. For her first birthday the Folbiggs had a huge party, and after that Ms Folbigg began to allow herself to see a future with Laura.[93]

57. In relation to Laura's health Ms Folbigg told police:

> *there had been no other problems during that first 12 months other than when she had sort of had her first cold, during the first cold season she had come up with the sniffles a couple of times. Nothing ever serious and she always soldiered through it and it didn't last very long…* [after 12 months] *she did come down with a flu or bad cold once.*[94]

58. Mr Folbigg said that although he thought Ms Folbigg was happy being a mum, she would get frustrated and growl on a daily basis because Laura was not having dinner at the right time or going to bed when Ms Folbigg wanted her to, or because of Mr Folbigg's attitude.[95] He considered that at this point their relationship had "fairly much packed it in" and she had written him a letter in which she said she wanted to end the marriage.[96]

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86 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) pp 20-21.
87 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 21.
88 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 21.
89 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 22.
90 3 April 2004 T154.21-38; Exhibit H, Forensic pathology tender bundle, Birth certificate of Laura (4 April 2000).
91 15 April 2003 T691.45-692.19; Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Seton (23 November 1999) pp 2-3.
92 3 April 2003 T155.18-160.30; 9 April 2003 T399.47-55.
93 Exhibit E, ERISP of Kathleen Folbigg Q333.
94 Exhibit E, ERISP of Kathleen Folbigg Q333.
95 3 April 2003 T174.1-5.
96 3 April 2003 T162.24-31, T163.9-164.5.

59. On 28 February 1999 Mr Folbigg noticed that something was off between Laura and Ms Folbigg. He asked Ms Folbigg about it and said that she told him "Oh, she's got the shits with me… It's probably over what I did to her last night… I lost it with her."[97]

60. On the morning of 1 March 1999, Mr Folbigg said Ms Folbigg lost her patience with Laura and growled at her. She pinned Laura's hands down and tried to feed her cereal. She then pulled Laura out of her highchair and put her on the ground saying "go to your fucking father... I can't handle her when she's like this." By the time Mr Folbigg left for work, Laura was sitting watching television.[98]

61. Later that morning Ms Folbigg called Mr Folbigg to apologise for losing her temper and took Laura to visit him at work for morning tea. Ms Folbigg left about 11:30am and said that Laura fell asleep in the car on the way home and so she put her to bed for a nap.[99]

62. Laura died on 1 March 1999 aged 18 months and 22 days. The autopsy report gave the cause of Laura's death as "undetermined". It was reported that she had myocarditis, an inflammation of the muscular walls of the heart, but that this represented an incidental finding.[100]

The health of the children and Ms Folbigg

63. In 1991 Dr Alison Colley worked at the Newcastle and Northern NSW Genetics Service (now Hunter Genetics) as a Staff Specialist Clinical Geneticist and in that capacity met Mr and Ms Folbigg following the death of their first two children.[101] She said that both parents agreed that Caleb and Patrick were normal healthy children prior to their sudden unexpected event, which was lethal for Caleb and resulted in severe subsequent neurological damage for Patrick.[102]

64. As part of her dealings with Mr and Ms Folbigg, Dr Colley met with Mr Folbigg's sister, Carol Newitt, who she described as "normal".[103]

65. For the purposes of genetic testing being conducted during the Inquiry, Dr Colley reviewed the children's medical history and formed the view that prior to the children's deaths (and in Patrick's case, prior to his ALTE), each of the children were healthy, well-grown, normally developing children who were normal in appearance.[104]

66. In particular, there was no evidence of pregnancy-related complications, congenital malformations, or dysmorphic features, and none of the children had a surgical operation, were admitted to hospital with a significant medical problem or were on continuous medication.[105] The tests conducted on the children, including for MCAD mutation testing and inborn errors of metabolism, were normal. The children's development was also normal. [106] All were thriving at the time of their unexpected event.[107]

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97 3 April 2003 T171.21-24.
98 3 April 2003 T171.52-173.34.
99 3 April 2003 T174.25-177.19.
100 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) pp 2, 4.
101 Exhibit AA, Report of Dr Alison Colley (26 November 2018) p 1.
102 Transcript of the Inquiry, 15 April 2019 T382.17-21.
103 Transcript of the Inquiry, 15 April 2019 T379.45-380.1.
104 Transcript of the Inquiry, 17 April 2019 T553.26-35.
105 Transcript of the Inquiry, 15 April 2019 T381.30-384.10.
106 Exhibit H, Forensic pathology tender bundle, Statement of Dr Bridget Wilcken (14 January 2000) pp 2-3.
107 Transcript of the Inquiry, 15 April 2019 T382.17-21.

67. Cardiac testing was carried out on Ms Folbigg for the purpose of the Inquiry. She was also healthy, and notably she did not suffer from a cardiac condition.[108]

68. The health of the children and of Ms Folbigg was confirmed by the genetic testing completed during the Inquiry.

The police investigation

69. On the afternoon of Laura's death on 1 March 1999, Detective Senior Constable Bernard Ryan attended Singleton Hospital to commence an investigation into Laura's death, including speaking with Mr and Ms Folbigg. He also conducted an inspection of the interior of their Singleton house which had been secured to preserve the integrity of any physical scenes or exhibits.[109]

70. Following Laura's death, Mr and Ms Folbigg's relationship deteriorated. Ms Folbigg said she "could not cope with his grieving".[110] They attempted to attend counselling together but Ms Folbigg ultimately left the family home in mid-April 1999 to live in a flat.[111]

71. By mid-June Ms Folbigg had returned to the matrimonial home.[112]

72. On 23 July 1999 Detective Senior Constable Ryan attended Ms Folbigg's home and asked her to attend an interview. She agreed and was interviewed at Singleton police station from 9:26am until 5:40pm, with several breaks throughout the day.[113]

73. After the interview concluded, police executed search warrants on the matrimonial home and Ms Folbigg's flat.[114]

74. Mr and Ms Folbigg separated on a permanent basis in June 2000.[115]

75. On 19 April 2001 Detective Senior Constable Ryan arrested Ms Folbigg and charged her with four counts of murder for the deaths of Caleb on 20 February 1989, Patrick on 13 February 1991, Sarah on 30 August 1993 and Laura on 1 March 1999.[116]

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108 Exhibit Y, Report of Professor Jonathan Skinner (31 March 2019) p 8; Exhibit BJ, Further report of Professor Jonathan Skinner (24 April 2019) p 4; Exhibit BL, Report of Associate Professor Hariharan Raju (18 April 2019) p 3.

109 28 April 2003 T956.38-51.

110 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 10.

111 2 April 2003 T136.5-137.2.

112 3 April 2003 T183.7.

113 28 April 2003 T963.8-16.

114 28 April 2003 T964.30-965.36.

115 3 April 2003 T184.4-12.

116 1 May 2003 T1087.19-33.

Report of the Inquiry into the convictions of Kathleen Megan Folbigg

July 2019

Volume 1: Chapter 3

Chapter 3: The Trial and Appeal Proceedings

Introduction

1. In the first part of this chapter I set out a summary of Ms Folbigg's judicial proceedings, including the:
   a. pre-trial proceedings;
   b. evidentiary and procedural rulings during trial;
   c. trial;
   d. post-trial proceedings, including sentence and appeals.
2. I then consider the submissions made by Ms Folbigg about the use of coincidence evidence and the conduct of the Crown Prosecutor at trial.

Pre-trial proceedings

Charge of Ms Folbigg

3. On 24 May 2002, Ms Folbigg was committed to the Supreme Court of New South Wales at Sydney for trial.[1] An indictment containing the four counts of murder was presented in the Supreme Court. At arraignment Ms Folbigg entered pleas of not guilty to each count.
4. On 25 October 2002, the Crown presented an ex-officio indictment laying an additional charge of one count of maliciously inflicting grievously bodily harm with intent to do grievous bodily harm, in respect of Patrick's ALTE on 18 October 1990. Ms Folbigg was re-arraigned and entered pleas of not guilty to each count.[2]

The Crown's tendency and coincidence evidence notices

5. On 25 October 2002, the Crown served tendency and coincidence notices pursuant to Part 3.6 of the *Evidence Act 1995* (NSW). By those notices, the Crown indicated its intention to rely on evidence relating to the deaths of each child, and the ALTE concerning Patrick, as being cross-admissible in relation to each count.[3]

1 24 May 2002 T7.14-16.
2 25 October 2002 T1.30-2.5.
3 25 October 2002 T2.7-17.

6. The coincidence particularised by the Crown was as follows:

   i. that each of the accused's children died/had an ALTE (Apparent Life Threatening Event) in a similar way

   ii. that each of the accused's children died/had an ALTE from the same cause

   iii. that the accused killed/caused an ALTE to each of the four children by asphyxiating them with the intent to kill or do GBH [grievous bodily harm] to them

   iv. that the accused's four children did not die from Sudden Infant Death Syndrome or any other illness, disease or syndrome.[4]

7. The matters relied upon by the Crown in the coincidence notice as coincidence evidence related to similarities in the circumstances concerning the death or ALTE of each child, namely that:

   i. each child was under 2 years of age at the time of death or ALTE (and it may be noted, additionally, that three such deaths and one ALTE occurred in the first year of life);

   ii. each death occurred at a time which is unusual for a SIDS event;

   iii. each death occurred in the child's own cot or bed;

   iv. each death or ALTE occurred during a sleep period;

   v. each child was last seen alive by the accused;

   vi. each child was found not breathing by the accused, and in relation to those who died in the night, she claimed to have observed from a distance, and in the dark, that they had stopped breathing;

   vii. only the accused was awake or present at the time when each child was found dead or not breathing;

   viii. there was, in each case, a short interval between the time when the child was last claimed to have been seen alive by the accused, and the time when he or she was found lifeless or not breathing properly;

   ix. in relation to the children who died in their cots or had an ALTE in the night, the accused had got up to go to the toilet, and in some cases had returned to bed, before getting up again and sounding the alarm;

   x. the accused had failed to pick up or attempt to resuscitate any of the children after the discovery of his or her death or cessation of breathing (subject to her claim to have done so in relation to Laura);

   xi. when each child was found he or she was warm to the touch;

   xii. there were no signs of any injury found on any child;

   xiii. no major illness preceded the death or the ALTE in any of the cases;

   xiv. each of Caleb, Sarah and Laura gave every appearance of being normal and healthy before his or her death, as had Patrick before his ALTE;

[4] Crown Notice of Coincidence Evidence (24 October 2002) p 1.

> *xv. the sleep studies for each child were normal (save for Caleb, who by reason of being the first born was not the subject of any such study);*
>
> *xvi. the tests for any inherited and/or biochemical disorder or metabolic abnormality were negative in each case;*
>
> *xvii. the death or ALTE in each case, arose from an hypoxic event; the sleep monitors, which had been provided following the earlier deaths and ALTE, were not in use at the time of death in the case of Sarah and Laura; and*
>
> *xviii. the accused had shown acute irritation in relation to each child, or appeared to have been in a condition of stress, before the death or ALTE.*[5]

8. The Crown particularised that Ms Folbigg had a tendency to:

> *"become stressed and lose her temper and control with each of her four children, and then to asphyxiate them."*[6]

Ms Folbigg's separate trials application

9. Prior to the commencement of the trial Ms Folbigg applied to the Supreme Court to separate the counts on the indictment into separate trials ("the separate trials application"). She sought that the counts of murder relating to Caleb, Sarah and Laura each be heard individually, and separately from the counts relating to Patrick. Ms Folbigg did not oppose the counts relating to Patrick being tried together.

10. On 22 November 2002, the application was heard by Justice Wood, Chief Judge at Common Law. The Crown brief was tendered on the application, which included expert reports by Professor Berry, Dr Cala, Dr Ophoven, Professor Herdson, Professor Ouvrier and Dr Beal. The defence response was also tendered, including reports by Professors Byard and Busutill. By agreement, no oral evidence was given.[7]

11. Ms Folbigg contended that the matters asserted by the Crown to be coincidence evidence should not be admitted at the trial because they did not satisfy the requirements of ss 98 and 101(2), or that they should be excluded pursuant to ss 135 or 137, of the *Evidence Act*. Those provisions required assessment and weighing of the probative value of the evidence relied upon by the Crown, and the prejudicial effect of the evidence on Ms Folbigg if admitted before the jury.

12. It was submitted by reference to those sections of the *Evidence Act* that the evidence could or should not be admitted because its probative value was outweighed by its prejudicial effect on Ms Folbigg, and the counts should be severed and the matters tried separately.[8]

13. Ms Folbigg also submitted there should be limits to the Crown's tendency argument, however both the prosecution and defence agreed that the jury should be directed that they could only use tendency reasoning if they had already come to the conclusion that they were satisfied beyond a reasonable doubt that one of the children was killed by deliberate suffocation by the accused.[9]

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5 *R v Folbigg* [2002] NSWSC 1127, [62].
6 Crown Notice of Tendency Evidence (24 October 2002) p 1.
7 2 November 2002 T1.16-3.56.
8 *R v Folbigg* [2002] NSWSC 1127, [52], [71].
9 22 November 2002 T25.25-51.

Ruling on application

14. On 29 November 2002, Wood CJ at CL dismissed the separate trials application.[10]

15. In the reasons for judgment, his Honour first set out the provisions of ss 98, 101(2), 135 and 137 of the *Evidence Act*.

16. According to the statutory test provided for by the *Evidence Act*:

    a. the evidence of two or more substantially and relevantly similar events which occur in substantially similar circumstances is not admissible to prove that, because of the improbability of the events occurring coincidentally, a person did a particular act unless the court thinks that the evidence, either by itself or having regard to the other evidence adduced by the party (here the Crown) seeking to establish coincidence, would have "significant probative value"; [11] and

    b. to be admissible when adduced in the Crown case, the probative value of the evidence must also substantially outweigh any prejudicial effect it may have on the accused person.[12]

17. Next, his Honour set out the case law relevant to these provisions.[13] Later in the reasons explaining the ruling that the evidence was admissible as coincidence evidence, his Honour stated that "the test in *WRC, Joiner*, and *Pfennig* is satisfied."[14]

18. According to the cases referred to, the evidence would only be admissible if, in combination with the other evidence in the Crown case, it was such that no reasonable view would remain open that would be consistent with the innocence of the accused.[15]

19. His Honour distinguished Ms Folbigg's case from that in *R v Phillips* on the basis that in Ms Folbigg's case:[16]

    a. there were many more matters relied upon by the Crown as showing a substantial and relevant similarity;

    b. there was independent evidence in the form of the diaries, and Mr Folbigg's evidence; and

    c. there was some expert evidence in respect of each death which would:

        i. exclude SIDS as a cause;

        ii. identify the improbability of the various incidental medical conditions observed post-mortem as the cause of death/ALTE; and

        iii. identify asphyxia as a possible or probable cause of death.[17]

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10 *R v Folbigg [2002] NSWSC 1127, [141].*

11 *Evidence Act 1995* (NSW) s 98(1)(b).

12 *Evidence Act 1995* (NSW) s 101(2).

13 *R v Folbigg [2002] NSWSC 1127, [79]-[81], citing Pfennig v The Queen* (1995) 182 CLR 461; *R v WRC* [2002] NSWCCA 210; *R v Joiner* [2002] NSWCCA 354.

14 *R v Folbigg* [2002] NSWSC 1127, [109].

15 *R v Folbigg* [2002] NSWSC 1127, [79]-[81], [109].

16 [1999] NSWSC 1175.

17 *R v Folbigg* [2002] NSWSC 1127, [94]-[97].

20. His Honour considered the Crown case against Ms Folbigg to be similar to that of *R v Clark*.[18] The similarities between the two cases his Honour identified were:

    a. the effect of the medical evidence as a whole (taking the Crown case at its highest) was that neither baby had been the subject of a SIDS death and there was a consensus, as the lowest common denominator, that each death was unexplained, but was consistent with an unnatural death; and

    b. the medical evidence did not stand alone. A large number of other pieces of circumstantial evidence including evidence relevant to the credibility of the accused could properly be relied on as tending to prove the accused's guilt.[19]

21. His Honour identified the following other circumstantial evidence which he considered to be relevant in the assessment of the probative value of the medical evidence:

    a. the infrequent incidence of SIDS;

    b. the rarity of repeat incidents of SIDS and of unexplained infant deaths or ALTE's within one family;

    c. the absence of any metabolic abnormality in any of the children, let alone a common abnormality;

    d. the fact that each was a healthy child and that such physical or medical conditions, as were observed post mortem, were unlikely causes of death;

    e. the absence of any sleeping abnormality in the three children who were tested and/or monitored;

    f. the fact that monitoring was provided but then ceased in relation to Sarah and Laura – a matter of some importance in view of the diary entry of 25 August 1997;

    g. the fact that two of the children were found by the accused within the very brief window between a child being found moribund and dead;

    h. the fact that all children were found by the accused while they were still warm, even though in four of the five relevant instances this occurred at night;

    i. the unexplained absence of Sarah and the accused at about 1 am, shortly before she was found dead;

    j. the unusual behaviour of the accused in getting up from bed, leaving the room, returning, and then getting up again only to discover, in the case of some of the children, that they were moribund or lifeless;

    k. the fact that she claimed to have observed, in the dark and from some distance away, that some of them were not breathing;

    l. the stress and anger which the accused had expressed toward the children;

    m. the fact that the accused would not nurse or endeavour to resuscitate the children when they were found; and

[18] [2000] EWCA Crim 54.

[19] *R v Folbigg* [2002] NSWSC 1127, [101]-[104].

> n. *the diary entries including, in particular, the sections which I have emphasised in the extracts set out earlier in these reasons, so far as they may reveal an absence of love for, or a bond with, the children, an acceptance by the accused of her hand in their deaths, her black moods and stress, her fears as to the way she behaved when stressed, and any resentment which she may have held in relation to the curtailment of her outside activities by reason of the need to care for Laura.*[20]

22. His Honour referred also to what appeared to him, prima facie, to be significant admissions by the accused in the diaries concerning the deaths of some of the children as well as the evidence concerning her moods and irritation proximate to their deaths.[21]

23. In ruling the evidence admissible as coincidence evidence and dismissing Ms Folbigg's separate trials application, Wood CJ at CL concluded:

    a. the evidence relied upon by the Crown as coincidence evidence carried "considerable probative force" in relation to all counts, when considered in combination with the other circumstantial evidence (for the purposes of ss 98(1)(b) and 101(2) *Evidence Act)*;[22]

    b. that suitable directions to the jury could be framed so as to ensure the jury did not use the evidence in some illogical way or give to it a weight which it did not deserve (for the purpose of s 101(2) *Evidence Act*), meaning the Crown should be allowed to call the evidence concerning each death or ALTE as evidence admissible in respect of each and every count;[23] and

    c. the probative value of the evidence was not outweighed by any danger of unfair prejudice to the accused for the purposes of ss 135 or 137 of the *Evidence Act*.[24]

24. Notwithstanding the expression of opinion by some of the medical experts in their reports regarding the ultimate issue as to causes of death having regard to the circumstance of the other children's deaths/ALTE, his Honour held:

    a. any reasoning based only upon an exercise of statistical probability would be potentially misleading;

    b. the medical experts were not prevented from giving evidence to the effect that SIDS is a relatively infrequent event and that multiple SIDS deaths and/or multiple unexplained deaths or ALTEs involving infants within any one family are even more infrequent; and

    c. the medical experts could not take the next step, which was properly one of fact for the jury and not one dependent upon medical or scientific expertise, to offer a view that it was Ms Folbigg who induced each event or that the deaths were in fact caused by induced asphyxia.[25]

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20 *R v Folbigg* [2002] NSWSC 1127, [107].
21 *R v Folbigg* [2002] NSWSC 1127, [109].
22 *R v Folbigg* [2002] NSWSC 1127, [106]-[112].
23 *R v Folbigg* [2002] NSWSC 1127, [113]-[122].
24 *R v Folbigg* [2002] NSWSC 1127, [123]-[129].
25 *R v Folbigg* [2002] NSWSC 1127, [88]-[91], [142].

Application for leave to appeal to the Court of Criminal Appeal

25. Ms Folbigg applied for leave to appeal against Wood CJ at CL's decision of 29 November 2002 to refuse the separate trials application. On 6 February 2003, the Court of Criminal Appeal heard the application. On 13 February 2003, the Court dismissed the application for leave.[26]

Decision

26. As to the applicable test, Hodgson JA acknowledged that the Court was required to apply the *Evidence Act*, not the common law. His Honour nonetheless concluded that the Court was required to follow the *Pfennig* test when assessing whether the probative value of the evidence outweighed its prejudicial effect for the purpose of s 101(2) of the *Evidence Act*, such that this would only be established if there was no rational view of the evidence consistent with the innocence of the accused.[27]

27. Hodgson JA set out parts of Wood CJ at CL's decision dealing with the medical evidence and summarised the effect of that evidence as follows:

    a. in each case medical considerations alone left a possibility that the cause of death was asphyxiation, this being a reasonable possibility and not a possibility which was merely remote or fanciful; and

    b. in each case there was no other cause of death which could be considered as something more than a reasonable possibility.[28]

28. As to the absence of direct proof of death by induced asphyxiation precluding the evidence from having the requisite probative effect in *Pfennig*, Hodgson JA considered this submission to misconceive the principles concerning circumstantial evidence.[29]

29. Hodgson JA stated his opinion that to determine whether the test for admissibility of the coincidence evidence was met, it was necessary first to consider the evidence relating to each individual count in the absence of evidence relating to the other counts. Secondly the test required considering whether any deficiency in proof of Ms Folbigg's responsibility for the death or ALTE in question would be overcome by the evidence relating to the other counts (so that the latter evidence would leave no rational view consistent with innocence in relation to the particular count being considered).[30] His Honour observed that while Wood CJ at CL did not explicitly undertake that course, that was the substance and effect of what his Honour did.

30. Hodgson JA considered that following that process in relation to each count, there would be a deficiency of proof of guilt in relation to each count without evidence concerning the other children, but that the additional evidence concerning the others would leave no rational view consistent with innocence in relation to the particular count being considered.[31] His Honour cited the same reasons as Wood CJ at CL for this view, being

    a. the extreme improbability of four such deaths and one ALTE occurring to children in the immediate care of their mother;

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26 *R v Folbigg* [2003] NSWCCA 17 (Hodgson JA at [1]-[35], Sully and Buddin JJ agreeing at [36]-[37]).
27 *R v Folbigg* [2003] NSWCCA 17, [27].
28 *R v Folbigg* [2003] NSWCCA 17, [29].
29 *R v Folbigg* [2003] NSWCCA 17, [30].
30 *R v Folbigg* [2003] NSWCCA 17, [31]-[32].
31 *R v Folbigg* [2003] NSWCCA 17, [32].

b. asphyxiation being a substantial possibility; and

c. no other cause of death being anything more than a substantial possibility, without the mother having contributed to any of the deaths.[32] This was particularly so in light of the diary entries.[33]

31. As to the defence submission about prejudice arising from confusion due to the way the matter would be left to the jury, Hodgson JA accepted there was a possibility of such confusion which would be prejudice relevant to ss 101, 135 and 137 of the *Evidence Act*.[34] However, his Honour found that the probative value of the evidence was such that it substantially outweighed any prejudicial effect.

32. In this regard, Hodgson JA considered that the possibility of confusion could be addressed by the jury being asked to consider first whether there was any reasonable possibility that all deaths and the ALTE occurred by natural causes.[35] If so, a verdict of not guilty should be returned on all of the counts. If not, the jury would be told that this did not mean there was a contribution from Ms Folbigg in each and every case, and it would be necessary to consider the evidence in each individual case.[36]

33. His Honour concluded that in relation to each count, the evidence concerning the other counts and other children was admissible as coincidence evidence.[37] In those circumstances there was a sufficient basis to justify refusing to separate the trials, and that it was accordingly unnecessary to consider whether the evidence was also admissible as tendency evidence.

Application for special leave to the High Court

34. At the request of Ms Folbigg, the Court of Criminal Appeal ordered that the trial, which had been listed to commence on 10 February 2003, be temporarily stayed until 24 February 2003 to enable her to make an application for special leave to appeal to the High Court against the Court of Criminal Appeal's decision.[38]

35. Ms Folbigg filed an application in the High Court for special leave to appeal. Ms Folbigg also filed a summons in the High Court seeking an order that the trial be stayed pending hearing of the application. An oral hearing in respect of the stay application was held before McHugh J on 19 February 2003.

Determination

36. At the conclusion of the hearing, McHugh J gave *ex tempore* reasons dismissing the summons on the basis that the prospects of special leave being granted were not sufficiently high, and the circumstances of the case not exceptional such as to warrant a stay of the trial.

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32 *R v Folbigg* [2003] NSWCCA 17 [32].
33 *R v Folbigg* [2003] NSWCCA 17 [32].
34 *R v Folbigg* [2003] NSWCCA 17 [33].
35 *R v Folbigg* [2003] NSWCCA 17 [33].
36 *R v Folbigg* [2003] NSWCCA 17 [33].
37 *R v Folbigg* [2003] NSWCCA 17 [34].
38 Transcript of Proceedings, *Folbigg v The Queen* [2003] HCATrans 589, 402-413.

37. His Honour held that the arguments on the application were insufficient to overcome the High Court's reluctance to allow special leave to appeal from an interlocutory decision, and in particular to intervene in the criminal processes of the State before verdict.[39] The Court of Criminal Appeal had carefully considered the relevant authorities, and had accepted Ms Folbigg's contention that the *Pfennig* test applied to s 101 of the *Evidence Act*, albeit that this was, his Honour said, "a proposition that I think is highly debateable."[40] McHugh J noted that the Court of Criminal Appeal had said that even if it was only the occurrence of all four offences in similar circumstances that could prove Ms Folbigg was responsible for any one of them, the *Pfennig* test might still be satisfied.

38. Given Ms Folbigg had succeeded in persuading the primary judge and the Court of Criminal Appeal that *Pfennig* applied, his Honour found that the application did not raise any major questions of principle, as opposed to the application of established principles in particular circumstances or the application of a statutory test to the particular facts of the case.[41] His Honour decided the case was not so exceptional as to warrant granting a stay, and dismissed the summons, observing also that the Court is always in a better position to evaluate whether a miscarriage has occurred after examining all the evidence rather than at an interlocutory stage of proceedings.

Application for adjournment of the trial

39. The trial remained listed to commence on 24 February 2003. On 21 February 2003, Ms Folbigg applied to adjourn the trial. The basis for the application was the recent identification of a hypothesis as to a potential correlation between particular genetic mutations and SIDS, namely either an increased risk of SIDS or a cause of SIDS. This development had been brought to the parties' attention in the English appeal case of *R v Clark* which was heard and determined by the English Court of Appeal in January 2003.[42]

40. Wood CJ at CL granted the adjournment application and vacated the existing trial date to enable further relevant testing to occur.[43]

Evidentiary and procedural rulings during the trial

41. The trial commenced before Barr J and a jury of 12 on 1 April 2003.

42. The transcript of proceedings during both the pre-trial and trial stages reflects efforts at cooperation between the Crown and defence to attempt to narrow the issues in dispute which required rulings from the trial judge

43. A number of evidentiary and procedural matters were dealt with during the course of the trial in the absence of the jury. These matters may be grouped into the following categories:

    a. rulings on the medical opinion evidence;

    b. rulings on the evidence of Ms Folbigg's versions of events;

    c. rulings on the evidence of certain lay witnesses; and

    d. procedural rulings.

    Two of these categories warrant detailed examination.

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39 Transcript of Proceedings, *Folbigg v The Queen* [2003] HCATrans 589, 509-512.

40 Transcript of Proceedings, *Folbigg v The Queen* [2003] HCATrans 589, 546.

41 Transcript of Proceedings, *Folbigg v The Queen* [2003] HCATrans 589, 550-560.

42 [2003] EWCA Crim 1020.

43 Exhibit G, Further set of documents from 2003 trial, Judgment on application for adjournment and application for vacation of hearing date: *R v Folbigg* (Supreme Court of New South Wales, Wood CJ at CL, 21 February 2003) [22].

Key rulings on the medical opinion evidence

Expert opinions as to individual cause of death taking account of the other deaths

44. The parties sought a series of rulings about the evidence of individual medical expert witnesses concerning the admissibility of opinions expressed about the cause of death (and ALTE) in the individual cases, including opinions based on the circumstances of the death (and ALTE) of the other children.

45. The effect of the rulings was that the experts:

    a. could give evidence about the possible or probable cause of death of each child and of the ALTE based on circumstances directly relevant to the evidence in question, namely the medical history of the child, the circumstances in which the child was found, the results of the post-mortem examination and the results of subsequent tests; and

    b. could not give evidence about the possible or probable cause of death based on additionally the fact that each of the other children had died unexpectedly or that one had unexpectedly suffered an ALTE.[44]

46. His Honour held that the second opinion depended entirely on lay coincidence reasoning, and accordingly was not based on the expert's specialised knowledge based on training, study or experience as required by s 79 of the *Evidence Act* to make the opinion admissible.[45]

47. The rulings also determined that medical experts, with relevant practical and research experience, could give evidence of their knowledge of there not having been any case of three or more deaths attributed to SIDS within the same family reported in the literature, or encountered in the course of their own experience.

48. The key individual rulings are set out below.

Ruling regarding Dr Cala's evidence as to SIDS diagnoses and probable cause of death

49. Dr Cala was the forensic pathologist who carried out the autopsy on Laura, and also reviewed the autopsies of all the children for the Crown.

50. Barr J noted that Dr Cala's conclusions appeared to be based on nothing more than that the child died in unexplained circumstances while in the care of the same family.[46] His Honour held that a statement that an unexplained death is more likely to be called a SIDS death if there is no prior unexplained death in the family, but less likely to be properly called a SIDS death if there is such a prior unexplained death, was not a statement of medical opinion. His Honour noted it may nonetheless be a statement of common sense and it may be right.[47]

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44 Exhibit G, Further set of documents from 2003 trial, Judgment on Crown application for exception to earlier ruling regarding Professor Roger Byard: *R v Folbigg* (Supreme Court of New South Wales, Barr J, 7 May 2003) [1].

45 Exhibit G, Further set of documents from 2003 trial, Judgment on Crown application for exception to earlier ruling regarding Professor Roger Byard: *R v Folbigg* (Supreme Court of New South Wales, Barr J, 7 May 2003) [2].

46 Exhibit G, Further set of documents from 2003 trial, Judgment on admissibility of evidence of Dr Allan Cala: *R v Folbigg* (Supreme Court of New South Wales, Barr J, 16 April 2003) [17].

47 Exhibit G, Further set of documents from 2003 trial, Judgment on admissibility of evidence of Dr Allan Cala: *R v Folbigg* (Supreme Court of New South Wales, Barr J, 16 April 2003) [18].

Ruling regarding Professor Berry's and Professor Herdson's evidence concerning the incidence of unexplained infant deaths and probable cause of death

51. Professor Berry was a consultant paediatric pathologist who provided opinions to the Crown about the deaths of each of the children and the ALTE. The defence objected to evidence from Professor Berry:

    a. observing that he knew of no examples in the literature of four infant deaths in the same family dying due to natural causes and considered this event to be unprecedented; and

    b. expressing the view that it was probable that all children were suffocated by the person who found them lifeless, namely Ms Folbigg.[48]

52. Barr J accepted the Crown's submission that the evidence was connected with Professor Berry's report of which the defence had had notice, and permitted the evidence to be given.[49]

Rulings on Crown's applications in respect of Professor Byard's evidence concerning a global view of cause of death

53. Professor Byard was a specialist forensic pathologist with a particular expertise in sudden infant and childhood deaths. He was to be called by the defence as a witness to give opinions about the deaths of each child.

54. His Honour ruled that Professor Byard's evidence was to be limited to expressing opinions about probabilities based only upon the circumstances directly relevant to the child concerned.[50]

Ruling regarding Dr Beal's opinion concerning probable cause of death of all children

55. Dr Beal was a paediatrician with approximately 30 years' study and practice experience in SIDS. Dr Beal was to be called as a witness in the Crown case, and had provided opinions about the cause of death of the children in formal reports, interviews with the Crown, and letters to the Crown.[51]

56. Barr J ruled that the probative value of the evidence concerning assessment of cause of death using only information relevant to the individual case outweighed any risk of unfair prejudice and was therefore admissible.

Rulings on evidence of Ms Folbigg's versions of events

Ms Folbigg's diary entry recording "I am my father's daughter" (Crown application to open, and admissibility/exclusion of the words)

57. One of Ms Folbigg's diaries obtained by police contained the following entry dated 14 October 1996:

> *Obviously I'm my father's daughter. But I think losing my temper stage and being frustrated with everything has passed.*[52]

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48 Exhibit G, Further set of documents from 2003 trial, Judgment on admissibility of evidence of Professor Berry and Professor Herdson: *R v Folbigg* (Supreme Court of New South Wales, Barr J, 24 April 2003) [1].

49 1 May 2003 T1079.45-47.

50 6 May 2003 T1174.57-1175.3.

51 Exhibit G, Further set of documents from 2003 trial, Exhibit B (VD) – Report of Dr Susan Beal (8 December 1999), Exhibit C (VD) – Facsimile of Dr Susan Beal (24 April 2003); Responses to Crown model questions – Dr Susan Beal, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C; 29 April 2003 T997.46-998.38.

52 Exhibit AZ, Diaries tender bundle, pp 102-103.

58. The trial judge did not deal with the diary note but ruled against admitting the evidence of Ms Folbigg's answers to questions relating to this in the record of interview.

59. The trial judge later indicated he had excluded the interview answers having performed a balancing exercise under s 137 of the *Evidence Act,* concluding that the probative value of the evidence did not outweigh the risk of unfair prejudice to Ms Folbigg.[53]

60. The trial judge determined not to admit any evidence which would tend to show that the accused's father murdered her mother on the basis that while the probative value of the evidence as explained by the Crown was substantial, it did not outweigh the danger of unfair prejudice to Ms Folbigg before the jury.[54]

Officer in charge's evidence regarding applicant's conduct in trial suggesting consciousness of guilt

61. During the course of the trial, while the video of her interview with police had been played Ms Folbigg had become visibly upset and distressed. She then appeared to be sobbing, broke down and left the dock.

62. On the basis that there were multiple possible explanations for Ms Folbigg's conduct during the playing of the interview, the trial judge held that the evidence was inadmissible because the probative value of the evidence was not high and the risk of impermissible prejudice was high.[55]

The trial

The Crown case as put to the jury

Evidence of the circumstances of each child's death and the medical evidence

Caleb's death

63. It was the Crown case that the evidence demonstrated that Caleb had a healthy 19 days of life, except for difficulty breathing and drinking at the same time.[56] Dr Springthorpe, the consultant paediatrician whom the parents had attended upon before Caleb's death, assessed that this was a "very very mild" problem of stridor or floppy larynx.[57]

64. The Crown prosecutor noted that Dr Springthorpe considered Caleb would grow out of the problem, and that the floppy larynx had nothing to do with Caleb's death.[58] He also noted that Dr Cummings found at post-mortem there was no abnormality of the larynx and nothing that could account for an obstruction of the airways.[59]

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53 3 April 2003 T188.45-189.25.
54 3 April 2003 T195.35-47.
55 5 May 2003 T1160.28-1162.55.
56 13 May 2003 T1309.50-54.
57 13 May 2003 T1310.9-10; 7 April 2003 T266.19-51.
58 13 May 2003 T1310.1-18.
59 13 May 2003 T1310.25-28; 7 April 2003 T267.31-46.

65. The Crown prosecutor referred to Dr Springthorpe's evidence that it is possible to smother a young baby and leave no external signs at all, described the distinction between "SIDS" and "undetermined" as descriptions of causes of death, and submitted that in Caleb's case the circumstances were "reduced to the extent of saying there was no known cause of death found."[60]

66. The Crown case relied on the following circumstances of Caleb's death on 19 February 1989:

    a. he was put to sleep at about 8:00pm;

    b. he was seen at about 10:00 or 10:30pm by his parents;

    c. Mr Folbigg was then woken to Ms Folbigg screaming "My baby, there is something wrong with my baby". Mr Folbigg found Ms Folbigg standing inside Caleb's room at the end of the bassinet, crying; and

    d. Caleb was warm to touch, suggesting one ambulance officer who wrote down he was not warm was wrong given Mr Folbigg's and the other ambulance officer's accounts that he was warm.[61]

67. The Crown prosecutor submitted that Ms Folbigg's accounts of what occurred on the night of Caleb's death differed across her versions as recorded by a police officer on the day following his death, in her own statement to police, in her record of interview with police, and in her diary entry for 19 February 1989.[62]

68. The Crown prosecutor raised with the jury the question of why Ms Folbigg had not picked Caleb up out of his bassinet, suggesting she had not done so because she had just smothered him.[63] The Crown prosecutor also suggested to the jury that it did not make sense that Ms Folbigg had awoken to go to the toilet 50 minutes after Caleb had gone to sleep after two restless hours, suggesting that when he cried again she had smothered him in her stressed state, desperate for sleep.[64]

69. The Crown prosecutor also referred to a diary entry recorded on the page of Patrick's date of birth:

> *This is the day that Patrick Allen David Folbigg was born. I had mixed feelings this day whether or not I was going to cope as a mother or whether I was going to get stressed out like I did last time. I often regret Caleb and Patrick, only because your life changes so much, and maybe I'm not a person that likes change, but we will see.*[65]

70. The Crown provided the jury with a summary document of the medical evidence in the Crown case and highlighted the following medical evidence regarding Caleb:

    a. Professor Berry said the haemosiderin in Caleb's lungs indicated there may have been a previous episode of asphyxia;

    b. a number of doctors who said the findings from post-mortem examination were consistent with Caleb having been deliberately suffocated;

    c. a number of doctors explained one would not really expect to see signs of smothering; and

    d. a number of doctors said that if they had done the post-mortem their finding would have been undetermined as opposed to SIDS, referring specifically to Dr Beal's evidence.[66]

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60 13 May 2003 T1310.30-1311.6.
61 13 May 2003 T1311.8-21.
62 13 May 2003 T1313.4-1314.19.
63 13 May 2003 T1311.41-45.
64 13 May 2003 T1313.22-31.
65 13 May 2003 T1314.21-44; Exhibit AZ, Diaries tender bundle, p 40.
66 13 May 2003 T1315.39-56; Crown summary of prosecution medical evidence at trial.

71. Later in the course of the closing address, while dealing with Sarah's death, the Crown prosecutor returned to the medical evidence concerning Caleb. He referred to the effect of the evidence of Professors Berry and Byard, that in all reported medical literature over the years there had never ever been a child reported to have died from a floppy larynx or displaced uvula, as was present at Sarah's post-mortem.[67]

Medical experts' opinions as to cause of death in isolation

72. Between the parts of the address concerning Caleb and Patrick, the Crown prosecutor drew the jury's attention to the fact that each of the experts who had given evidence, in the Crown case and in the defence case, was asked to give evidence about a diagnosis for each child looked at in isolation.

73. The Crown prosecutor explained that the rules of evidence required the doctors to look only at each individual case, whereas the jurors had the task of looking at the overview and combination of events, not as experts but as ordinary members of the community looking at logic, chances and possibilities.[68]

Patrick's ALTE

74. It was the Crown case that the evidence established Patrick had no health concerns whatsoever prior to the ALTE at four months of age.

75. In outlining the circumstances of Patrick's ALTE as relied on by the Crown, the Crown provided to the jury a chronology of the events of the day of each child's death and identified:

    a. at 8:30pm Patrick was put in his cot;

    b. at about 10:30pm he was seen in his cot, on his back with a sheet and blanket over him;

    c. at about 12:00 midnight or 1:00am Ms Folbigg fed Patrick;

    d. at about 3:00am Ms Folbigg heard Patrick coughing but went back to sleep;

    e. in the early hours of the morning Mr Folbigg was awoken to screams again, with Ms Folbigg standing at the end of Patrick's cot, screaming; and

    f. Patrick was warm to the touch.[69]

76. When dealing with the medical evidence, the Crown prosecutor referred first to the evidence of paediatric neurologist Dr Ian Wilkinson, and paediatrician Dr Joseph Dezordi, who treated Patrick at the time of the ALTE.

77. The Crown case relied on the following evidence from Dr Wilkinson:

    a. when Patrick arrived at the hospital his EEG was perfectly normal;

    b. he was never able to determine what had caused the original starvation of oxygen from Patrick's brain;

    c. any form of encephalitis was excluded, following a vast array of extensive testing including lumbar puncture, blood tests, scans and EEGs;

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67 13 May 2003 T1341.5-11.

68 13 May 2003 T1316.10-22.

69 13 May 2003 T1317.8-11, T1317.37-44, T1317.48-50, T1317.58; Crown chronology of deaths and ALTE.

d. no inherited disease was found; and

e. the extensive brain damage that Patrick suffered was "absolutely consistent" with him having suffered from a catastrophic asphyxiating event from unknown causes.[70]

78. From Dr Dezordi's evidence the Crown case highlighted the following:

a. when Patrick was admitted to hospital he appeared to be a normal child, nothing was obstructing his airway and there was no sign of long-term or general or acute illness, or trauma, or any severe infection;[71] and

b. Patrick had a high level of glucose in his urine which suggested a fairly catastrophic event, such as an asphyxiating event or a prolonged seizure.[72]

79. Referring to the summary of the Crown's medical evidence, the Crown prosecutor noted that Dr Dezordi and Dr Wilkinson, as well as Professor Herdson, Dr Beal and Professor Berry, were all of the view that it was very unlikely that the ALTE was caused by an initial seizure because one would expect a history of epilepsy, which Patrick did not have.[73] They each considered that one would not expect a first epileptic fit to cause brain damage of the kind that occurred.[74]

80. The Crown prosecutor pointed to consensus among the Crown's experts that the findings were consistent with Patrick having been deliberated smothered.[75]

81. The Crown prosecutor repeatedly referred to the necessarily small timeframe in which Patrick must have stopped breathing before Ms Folbigg found him, on Ms Folbigg's version of events, given he was able to be revived by ambulance officers.[76] The Crown prosecutor submitted it was not a mere coincidence that Ms Folbigg happened to wake up and hear Patrick in the approximately two minutes between his cessation of breathing and potential death.[77]

82. As when addressing about Caleb, the Crown prosecutor again raised with the jury the question of why Ms Folbigg had not picked up the child, particularly in circumstances where he was making noises and some respiratory effort.[78]

83. It was the Crown case that when combining the apparent coincidental timing and Ms Folbigg's failure to pick Patrick up upon finding him, with the circumstance of Mr Folbigg's return to work only three days earlier after three months at home with Ms Folbigg,[79] the only conclusion which the jury could reasonably come to was that Ms Folbigg had caused the ALTE.[80]

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70 13 May 2003 T1318.37-1319.7.
71 13 May 2003 T1319.16-21.
72 13 May 2003 T1319.25-28.
73 13 May 2003 T1319.37-43.
74 13 May 2003 T1319.44-49, T1319.55-58.
75 13 May 2003 T1319.51-55.
76 13 May 2003 T1318.4-9, T1320.4-15.
77 13 May 2003 T1321.14-29.
78 13 May 2003 T1317.57-1318.3, T1320.40-43.
79 13 May 2003 T1316.39-42, T1317.21-23.
80 13 May 2003 T1321.23-29.

Patrick's death

84. It was the Crown case that for the period between Patrick's ALTE and his death, the evidence established that Ms Folbigg was in a stressed, frustrated and angry state, and that Patrick was monitored and had no health problems other than blindness and epilepsy.[81]

85. The Crown prosecutor referred to Mr Folbigg's and Carol Newitt's (Mr Folbigg's sister's) account of Ms Folbigg's thought that Patrick and Craig may be better off without her. The Crown prosecutor referred in this regard to a diary entry from years later on 4 December 1996, which he suggested displayed similar thinking. The Crown prosecutor read the extract: "I have already decided, if I get any feelings of jealousy or anger too much I will leave Craig and baby rather than answer being as before".[82] He suggested a mother would only have such a thought if she thought the child was at risk because she had killed the earlier child.[83]

86. As to the day of Patrick's death, the Crown case highlighted the following circumstances:

    a. Mr Folbigg left for work at about 7:30am;

    b. at about 10:00am Mr Folbigg received a phone call from Ms Folbigg stating "it's happened again. I need you to come home";

    c. Ms Folbigg also called Carol Newitt, who attended the home very quickly, before Mr Folbigg;

    d. the ambulance called was logged at 10:03am;

    e. when Carol Newitt attended she went in to pick Patrick up from the cot and Ms Folbigg said "no, don't";

    f. Patrick was warm to the touch when Mr Folbigg arrived home five to seven minutes after Ms Folbigg called him, and when the ambulance officers arrived at 10:10am; and

    g. when Mr Folbigg arrived he grabbed Patrick from the cot, took him to a suitable place in the house and attempted to render resuscitation assistance.[84]

87. The Crown prosecutor referred to the following medical evidence arising from Patrick's attendance at the emergency department:

    a. Director of the Emergency Department at the time of Patrick's attendance, Dr Christopher Walker's evidence that Patrick had suffered a cardiac arrest, which can be caused by asphyxiation, and that there was no definitive diagnosis for what had happened to Patrick;[85] and

    b. Dr Wilkinson's evidence that Patrick had suffered from a catastrophic asphyxiating event from an unknown cause, and that despite what was recorded on the death certificate (asphyxia due to airways obstruction) he had not in fact found any obstruction.[86]

88. As to medical evidence upon autopsy, the Crown case relied upon Dr Wilkinson's evidence that there was no sign at all, during examination at the hospital or at the autopsy, of Patrick having suffered an epileptic seizure. After viewing all hospital records, the post-mortem report and histopathological findings, Dr Wilkinson's view was that the direct cause of Patrick's death was a catastrophic asphyxiating event from unknown causes and smothering could have caused this event.[87]

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81 13 May 2003 T1322.6-21.
82 13 May 2003 T1323.20-22.
83 13 May 2003 T1322.23-1323.23.
84 13 May 2003 T1323.33-1324.12.
85 13 May 2003 T1324.21-31.
86 13 May 2003 T1324.38-43.
87 13 May 2003 T1324.53-1325.4.

89. The Crown prosecutor also relied on the evidence of Dr Singh-Khaira who conducted the post-mortem, and Dr Kan, a neuropathologist assisting Dr Singh-Khaira, who excluded causes of death by way of metabolic or genetic disorders. They found that the only damage on the brain was consistent with the timing of the ALTE, and concluded no cause of death could be found.[88]

90. Finally the Crown prosecutor referred the jury to the summary of the Crown's medical evidence and Dr Wilkinson's and Professors Berry's and Herdson's view that an epileptic fit had not caused Patrick's death because of the absence of the normal signs one would expect to see if he had an epileptic fit.[89]

91. The Crown prosecutor referred again to Ms Folbigg having made multiple telephone calls and failed to render assistance to Patrick, and submitted the only reason for this behaviour was because she knew he was dead because she had killed him.[90]

Sarah's death

92. It was the Crown case that Sarah was born a happy and healthy child. The sleep study conducted by sleep expert Dr David Cooper was noted, which identified occasional normal episodes of central apnoea, being short pauses between breaths.[91] The Crown prosecutor suggested Dr Cooper was not at all concerned about the central apnoea Sarah had displayed. He pointed to Dr Cooper's opinion that it was a common occurrence in children which usually resolved with age, and that Sarah was getting as much oxygen as she needed despite the apnoea.[92]

93. The Crown case relied on the evidence concerning the sleep monitor blanket which was provided to Ms Folbigg and Mr Folbigg. The Crown prosecutor emphasised that it was Mr Folbigg who insisted on it being used, that Ms Folbigg wanted to abandon use of it, that it was liable to giving a lot of false alarms and that it had been used up until a day or two before Sarah's death.[93]

94. As when addressing on Patrick's death, the Crown prosecutor referred the jury to Mr Folbigg's evidence of Ms Folbigg getting frustrated with Sarah a lot, and additionally organising for Sarah to be minded by other people "a huge amount of the time".[94]

95. The Crown prosecutor went on to refer to:

    a. a letter by Ms Folbigg to Mr Folbigg demonstrating that she contemplated leaving him and Sarah;[95]

    b. the same diary entry referred to earlier while addressing Patrick's death referring to leaving Mr Folbigg and the baby "rather than being as before" (dated 4 December 1996, during Laura's life);[96] and

88 13 May 2003 T1325.8-20.
89 13 May 2003 T1326.20-24.
90 13 May 2003 T1325.52-1326.9.
91 13 May 2003 T1326.42-50.
92 13 May 2003 T1326.52-1327.2.
93 13 May 2003 T1327.33-43.
94 13 May 2003 T1327.45-53.
95 13 May 2003 T1327.54-58; Exhibit E, trial Exhibit AM, Letter from Kathleen Folbigg to Craig Folbigg (undated).
96 13 May 2003 T1328.18-31.

c. the following diary entry also written during Laura's life (dated 28 January 1998) but submitted as relating to Sarah's death:

> *I feel like the worst mother on this earth, scared that she will leave me now, like Sarah did. I knew I was short tempered and cruel sometimes to her and she left, with a bit of help. I don't want that to ever happen again. I actually seem to have a bond with Laura. It can't happen again. I'm ashamed of myself. I can't tell Craig about it because he'll worry about leaving her with me.*[97]

96. Arriving at the circumstances immediately preceding Sarah's death, the Crown prosecutor pointed to Mr Folbigg's evidence of Ms Folbigg's significant frustration towards Sarah the night before, including throwing her onto Mr Folbigg.[98] The Crown prosecutor sought to illustrate similarities between this event and a subsequent incident with Laura, recorded by Ms Folbigg in her diary for 28 January 1998:

> *Very depressed with myself. Angry and upset. I've done it. I lost it with her. I yelled at her so angrily that it scared her. She hasn't stopped crying. Got so bad I nearly purposely dropped her on the floor and left her. I restrained enough to put her on the floor and walk away, went to my room and left her to cry. Was gone probably only five minutes but it seemed like a lifetime.*[99]

97. The Crown prosecutor also emphasised the significance to the Crown case that the use of the sleep monitor was ceased no more than three nights prior to Sarah's death.[100] He referred in this regard to a subsequent diary entry when Laura was two and a half weeks old on 25 August 1997, in which Ms Folbigg wrote:

> *Monitor is good idea. Nothing can happen without the monitor going and since I am not game enough to not plug it in because they would want to know why I hadn't, everything will be fine this time.*[101]

The Crown prosecutor submitted the monitor was "keeping her honest".[102]

98. Having identified at the outset of the closing address the difference between Mr Folbigg's and Ms Folbigg's accounts of what occurred in the 20 minutes or so before Mr Folbigg heard Ms Folbigg scream at approximately 1:30am on the night of Sarah's death, the Crown prosecutor addressed the jury at length on the evidence on this issue.[103]

99. Mr Folbigg's account was to the effect that he had woken up at 1:10am and noticed both Sarah and Ms Folbigg were not in the bedroom where he, Ms Folbigg and Sarah slept, and also that a light was on. Mr Folbigg admitted to having lied to police about this issue during the course of giving his statements.[104]

100. Ms Folbigg's account was to the effect that Sarah had not left her bed and she had only noticed Sarah was not breathing when she returned from going to the toilet at 1:30am. She denied she was feeding Sarah or had left the room prior to going to the toilet.[105]

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97 13 May 2003 T1329.26-1330.3.
98 13 May 2003 T1329.45-51.
99 13 May 2003 T1330.12-20.
100 13 May 2003 T1330.29-50.
101 13 May 2003 T1330.52-1331.3.
102 13 May 2003 T1331.12-13.
103 13 May 2003 T1331.24-1339.51.
104 13 May 2003 T1332.4-19.
105 6 April 2003 T771.55-772.6; Exhibit E, ERISP of Kathleen Folbigg Q546, 552.

101. Ultimately, it was the Crown case that Mr Folbigg's lies to police could be readily understood in the context of the events and his relationship, and that there was other evidence which was "confirmatory" of his account at trial.[106]

102. The evidence referred to in this regard was firstly a diary entry for 16 May 1997 when Ms Folbigg was pregnant with Laura, submitted as relating to Sarah's death, in which Ms Folbigg wrote:

> *Night-time and early morning such as these will be the worst for me. That's when wishing someone else was awake with me will happen. Purely because of what happened before. Craig says he will stress and worry but he still seems to sleep okay every night and did with Sarah. I really needed him to wake that morning and takeover from me. This time I've decided if I ever feel that way again I'm going to wake him up.*[107]

103. Secondly referred to was the evidence of Lea Bown, Ms Folbigg's foster sister. Ms Bown gave evidence that the within hours of Sarah's death Ms Folbigg told her she had gotten up during the night, gone to the toilet and put on the hall light or toilet light and seen from the toilet that there was something wrong with Sarah.[108] This was said to be inconsistent with Ms Folbigg's version to police in which she said there was no light on until after she realised Sarah was not breathing.[109]

104. Thirdly referred to was a calendar from August 1993 in which the words "Sarah left us 1:00am" were recorded.[110]

105. Next referred to were two further diary entries submitted as relating to Sarah's death:

> ***9 November 1997:***
>
> *With Sarah, all I wanted was her to shut up and one day she did.*[111]
>
> ***28 January 1998:***
>
> *Scared that she will leave me like Sarah did. I knew that I was short tempered and cruel sometimes to her, and she left with a bit of help.*[112]

106. Finally the Crown prosecutor referred to an answer given by Ms Folbigg during her interview with police, submitted as a "Freudian slip" by a person who knew that, having smothered her daughter, she put her back into her bed and then went to find and uncover her, supposedly having discovered that she was deceased:

> *Q546. Do you understand the significance of that statement that Craig made to me at your family home in May this year?*
>
> *A. Yep but that's not how it was. She never left the bed. She was in the bed and I did shut the door. Yes, but I didn't turn any lights on.*[113]

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106 3 May 2003 T1334.22-1335.22.
107 13 May 2003 T1332.28-37; Exhibit AZ, Diaries tender bundle, p 162.
108 13 May 2003 T1334.50-58.
109 13 May 2003 T1335.12-17; Exhibit AZ, Diaries tender bundle, p 62.
110 13 May 2003 T1338.54-57; Exhibit E, trial Exhibit H, Calendar page of August 1993 in relation to Sarah Folbigg.
111 13 May 2003 T1339.3-4; Exhibit AZ, Diaries tender bundle, p 231.
112 13 May 2003 T1339.16-19; Exhibit AZ, Diaries tender bundle, p 258.
113 13 May 2003 T1339.22-35.

107. Dealing with the medical evidence, the Crown prosecutor referred the jury first to his cross-examination of Professor Hilton. He submitted that it was not appropriate for Professor Hilton to find that Sarah had died from SIDS because this excluded a possible view that she may have died from unnatural causes. It was the Crown case that a prudent pathologist ought to have found a cause of death from undetermined causes rather than from SIDS.[114] It relied in this regard on Dr Cala's evidence that he did not consider Professor Hilton had excluded deliberate or accidental trauma and that it was inappropriate to diagnose SIDS.[115]

108. The Crown case also relied on Professor Berry's opinion that if he was looking at Sarah's death in isolation he probably would have said SIDS but he would have had misgivings. The Crown prosecutor pointed out that Professor Hilton was not looking at Sarah's case in isolation as he "knew perfectly well" what had happened to the previous children.[116]

109. As to the displaced uvula located by Professor Hilton at post-mortem, the Crown prosecutor noted:

    a. Dr Cala, Professor Berry and Dr Beal were of the view this was not the cause of death;[117] and

    b. Professor Byard for the defence and Professor Berry for the Crown both said they had never seen a child who had died of a displaced uvula, nor had their colleagues, and nor had a case of death from a displaced uvula been identified in all the medical literature.[118]

Ms Folbigg's decision to have another child

110. In between the parts of the address concerning Sarah's and Laura's death, the Crown prosecutor raised with the jury Ms Folbigg's decision to have more children after the deaths of earlier children. He referred in this regard to the circumstances of her being stressed by the children, resenting the impositions they made on her life, her resentment of Mr Folbigg and the children themselves, and her threat to leave Mr Folbigg unless he cooperated by having another child.[119]

111. By reference to Ms Folbigg's diaries, it was the Crown case that the reason she chose to have another child was that she wanted to prove to herself that she was capable of having and bringing up a baby. The Crown contended that each time she believed that she was better able to manage and keep control, and to see the warning signs and to ask for help to avoid losing control again and killing a child again.[120] The following specific entries after Sarah's death were relied on:

> ***3 February 1997***
>
> *I wonder whether having this one wasn't just a determination on my behalf to get it right and not be defeated by my total inadequate feelings about myself. What sort of a mother am I, have I been? A terrible one. That is what it boils down to. That is how I feel and that is what I think I'm trying to conquer with this baby: to prove that there is nothing wrong with me. If other women can do it, so can I.*[121]

114 13 May 2003 T1340.17-27.
115 13 May 2003 T1340.32.36.
116 13 May 2003 T1340.38-42.
117 13 May 2003 T1340.44-52.
118 13 May 2003 T1340.50-56.
119 13 May 2003 T1345.29-1346.34.
120 13 May 2003 T1346.28-50.
121 13 May 2003 T1345.34-47; Exhibit AZ, Diaries tender bundle, p 126.

17 February 1997

This time I'm prepared and know what signals to watch out for in myself, changes in mood… I will get help if need be.[122]

14 October 1996

I suppose I would like to make all my mistakes and terrible thinking to be corrected and mean something now, plus I'm ready to continue my family time now. I think losing my temper and being frustrated with everything has passed. I now just let things happen and go with the flow and attitude I should've had with all my children. If given the chance I'll have it with the next one.[123]

28 April 1997

I think this baby deserves everything I can give her, considering I really gave nothing to the others. I think even my feelings towards this one are already deeper. Shame, but that is the way it is. I think it is because I'm 30 now, and time to settle and bring up a child. Obviously I wasn't ready before all that.[124]

112. On this issue the Crown prosecutor referred also to Mr Folbigg's evidence of:

    a. a conversation in which Ms Folbigg said to him that it would not happen again as they were more mature, more patient people, older and wiser;[125] and

    b. him, not Ms Folbigg, wanting to pursue genetic advice.[126]

Laura's death

113. In outlining the circumstances around Laura's death which the Crown relied upon, the Crown prosecutor first referred to the evidence of extensive testing done after her birth at Westmead Children's Hospital, followed by the use of a sleeping corometrics monitor which downloaded information to the Children's Hospital. It was the Crown case there was no abnormality found during the testing, and no instance of genuine respiratory distress recorded by the monitor.[127]

114. The Crown prosecutor pointed to Margaret Tanner's evidence that she had told the parents to use the monitor with all sleeps for 12 months, and both Ms Tanner's and Mr Folbigg's evidence that Ms Folbigg had stopped using the monitor during daytime sleeps from about two months after Laura's birth.[128]

115. He also pointed to evidence from Ms Bown, who was present at the home one day when she heard the monitor alarm go off twice. Ms Bown's evidence was that she told Ms Folbigg it had gone off, Ms Folbigg did not check on the child and replied "Oh don't worry about it. It's just a false alarm".[129]

122 13 May 2003 T1345.52-56; Exhibit AZ, Diaries tender bundle, p 127.
123 13 May 2003 T1346.2-10; Exhibit AZ, Diaries tender bundle, p 178.
124 13 May 2003 T1346.14-26; Exhibit AZ, Diaries tender bundle, p 156.
125 13 May 2003 T1346.53-58.
126 13 May 2003 T1347.5-12.
127 13 May 2003 T1347.17-19, T1348.27-28.
128 13 May 2003 T1347.30-35, T1349.22-35.
129 13 May 2003 T1348.45-48.

116. By reference to this evidence, as well as Ms Folbigg's own diary entries, it was the Crown case that Ms Folbigg knew that all the alarms the monitor made at various times were false, because she knew she had caused the death of her three earlier children.[130] The entries relied upon in this regard were as follows:

> ***20 September 1997***
>
> *Sleep. Who needs it? Yes, I am getting a little irritable now. This is my punishment for the others, to be continually woken up, because this time we know that we have a child with a sleeping disorder, even though I'm sure they're all false alarms. The thought is still scary.*[131]
>
> ***9 November 1997***
>
> *There is a problem with his security level with me, and he has a morbid fear about Laura. He, well, I know there is nothing wrong with her, nothing out of the ordinary anyway, because it was me. Not them.*[132]

117. Moving then to the circumstances more proximate to Laura's death, it was the Crown case that Ms Folbigg's relationships with Laura and with Mr Folbigg started to deteriorate when Laura was about 11 months of age. Reference was made to Mr Folbigg's evidence that Ms Folbigg growled every day, and on one occasion pulled Laura by one arm out of her high chair in frustration while feeding her.[133]

118. The Crown prosecutor referred to Ms Folbigg's answers in her interview with police, suggesting that by February 1999, about two weeks before Laura's death, Ms Folbigg was again considering leaving Mr Folbigg and her child because it would be better for them if she was not around.[134]

119. Arriving then at the days before Laura's death, the Crown case relied on Mr Folbigg's evidence of Ms Folbigg's account to him on the Sunday morning (before Laura died on the Monday), that she had "lost it" with Laura on the Saturday evening and hit and knocked her over.[135] The Crown prosecutor suggested that defence counsel's cross-examination of Mr Folbigg suggested there was no challenge to the suggestion that Laura had been knocked over, whether inadvertently or not.[136]

120. As to the morning of Laura's death, the Crown case relied on Mr Folbigg's account of events, the essential parts of which he submitted were independently confirmed by Ms Folbigg's police interview questions and the instructions she provided to her solicitors after she had done the interview.[137] Those features were as follows:

    a. Laura woke at about 6:20am and was clingy, subdued and whingey. She became very upset when she realised Mr Folbigg was going to work;[138]

    b. Ms Folbigg awoke at 6:45am. She lost patience with Laura and growled at her;[139]

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130 13 May 2003 T1347.55-1348.2.
131 13 May 2003 T1347.46-51; Exhibit AZ, Diaries tender bundle, p 220.
132 13 May 2003 T1349.6-10; Exhibit AZ, Diaries tender bundle, p 231.
133 13 May 2003 T1349.45-1350.2.
134 13 May 2003 T1350.16-34.
135 13 May 2003 T1351.5-26.
136 13 May 2003 T1351.42-1352.9.
137 Exhibit E, trial Exhibit O, Statement of Kathleen Folbigg (1 March 1999).
138 13 May 2003 T1352.11-15.
139 13 May 2003 T1352.12-18.

c. while feeding Laura breakfast, Ms Folbigg had both Laura's hands pinned down under her own hand and was trying to force-feed her. When Mr Folbigg confronted her she pulled the child out of the chair, growled, swore and screamed "I can't handle her when she is like this". Laura was hysterical, shaking, sobbing;[140]

d. before Mr Folbigg left for work Ms Folbigg said to him "Look she's fine now" and Laura was watching television;[141]

e. at about 8:30am Ms Folbigg telephoned Mr Folbigg stating she wanted to apologise for losing her temper and that they needed to talk about their life. Mr Folbigg asked Ms Folbigg to bring Laura to see him at work after they attended the gym that morning;[142] and

f. at about 10:30am Ms Folbigg and Laura attended Mr Folbigg's workplace. Laura was happy, active and giggly, and did not want to go with Ms Folbigg when they left at about 11:30am.[143]

121. In describing the circumstances of Laura's death, the Crown prosecutor referred to Ms Folbigg's accounts to various people, as Mr Folbigg was not home on this occasion.[144]

122. He noted first her account to the ambulance officer. Ms Folbigg said she had heard Laura coughing in the bedroom and checked her five minutes later and found her not breathing. He noted her report to the officer that Laura had been suffering from a runny nose and coughing for a couple of days.[145] Next he noted Ms Folbigg's account to police five months after Laura's death, which omitted any reference to coughing or having had a cold.[146]

123. The Crown prosecutor suggested that had the coughing in fact occurred, it was not a matter Ms Folbigg would have forgotten. It was the Crown case that she invented the story about Laura coughing because she needed an excuse for having gone into Laura's room to check up on her.[147]

124. The Crown prosecutor referred also to Ms Folbigg's account that she had carried Laura out of the car and into the house through the hallway to Laura's bedroom. He noted the location of Laura's shoes and bottle in the loungeroom, and suggested by reference to the plans of the house that getting to the bedroom did not require passing through the loungeroom.[148]

125. In dealing with the medical evidence, the Crown prosecutor stated at the outset it was a bit more complicated in Laura's case by reason of the finding of mild myocarditis.[149] On this issue, it was the Crown case that the most accurate expert evidence was from Dr Cala, who said:

a. the post-mortem findings were consistent with asphyxiation; and

b. the inflammatory infiltrate of her heart caused by myocarditis was consistent with the after effects of a cold or flu.[150]

140 13 May 2003 T1352.18-29.
141 13 May 2003 T1352.32-34.
142 13 May 2003 T1352.36-43.
143 13 May 2003 T1352.45-48.
144 13 May 2003 T1353.18-20.
145 13 May 2003 T1353.25-29.
146 13 May 2003 T1354.58-1355.22.
147 13 May 2003 T1354.26-38.
148 13 May 2003 T1356.35-49.
149 13 May 2003 T1356.51-57.
150 13 May 2003 T1356.57-1357.6.

126. The Crown prosecutor submitted that nobody could ever say, looking just at the medical evidence on its own, that Laura did not die from myocarditis.[151]

127. He referred to Dr Cala's opinion that the myocarditis was an incidental finding, and that it was not a reasonable possibility that Laura had died from myocarditis, based on:

    a. the absence of any evidence of heart failure;

    b. that her heart was normal; and

    c. the inflammation was patchy, mild and of a low amount.[152]

128. The Crown case relied also on the following other opinions:

    a. Professor Herdson's opinion that myocarditis was an incidental finding and not the cause of death, which it was submitted was supportive of Dr Cala's view;[153]

    b. Professor Hilton's view that myocarditis could have led to her death but his agreement that the finding of myocarditis did not exclude deliberate suffocation;[154] and

    c. Cardiologist Dr Bailey's opinion that the agonal or dying heartbeat found in Laura by the paramedics after her breathing stopped made it most likely that her breathing stopped before her heart stopped, which is not what would be expected from myocarditis as a cause of death, and that it was therefore unlikely that myocarditis caused her death. Also that the agonal rhythm was more consistent with smothering than with myocarditis.[155]

129. The Crown prosecutor went on to submit that the best evidence about myocarditis came not just from the Crown's experts but the defence expert paediatric cardiologist, Dr Owen Jones. He noted Dr Jones' evidence that:

    a. most people who have myocarditis recover from it;

    b. something like five or 10 per cent of people who get viral illnesses may have myocarditis;

    c. of those who present to cardiologists with myocarditis from viral illnesses, about a quarter of them die, and three quarters of them live;

    d. most of those who die do not die suddenly; and

    e. of those who die, most have symptoms that are able to be ascertained by a doctor.[156]

130. The Crown prosecutor noted that Laura had no symptoms and was playing perfectly happily in the pool the day before her death, and at the *crèche* of the gym and at her father's workplace on the day of her death.[157]

131. The Crown prosecutor submitted that if the jury looked only at the medical evidence alone in isolation they could not say it was impossible that Laura died from myocarditis. The Crown prosecutor suggested this was unrealistic, and not the way the jury should look at the evidence. He noted the jury does not look at the evidence in isolation, and brings into the courtroom common sense and knowledge of the world.[158]

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151 13 May 2003 T1357.8-17.
152 13 May 2003 T1357.17-29.
153 13 May 2003 T1357.29-31.
154 13 May 2003 T1357.33-36.
155 13 May 2003 T1357.36-49.
156 13 May 2003 T1357.56-1358.25.
157 13 May 2003 T1358.17-20.
158 13 May 2003 T1358.52-1359.1.

132. It was the Crown case that there was strong medical evidence that myocarditis was very likely to have been an incidental finding, and that the jury should then look at what happened on the morning, days and weeks before Laura's death, and also that Ms Folbigg had previously killed three of her children.[159]

133. It was the Crown case that looking at all the evidence, Laura was murdered by Ms Folbigg by suffocation.[160]

Coincidence and tendency evidence

134. The Crown prosecutor described the term "coincidence" to the jury as similarity evidence to disprove coincidence. The Crown alleged that the deaths and Patrick's ALTE had 10 features in common which disproved the events were merely coincidental. Those features were listed in a document provided to the jury at the commencement of this part of the Crown prosecutor's closing address.[161]

135. Those features, as described during the closing address, were:

    a. all five events occurred suddenly: the events were over in a matter of minutes;[162]

    b. all five events occurred unexpectedly: no child had any health problem that preceded the sudden deaths or ALTE, or gave any sort of warning sign or previous symptom;[163]

    c. all five events occurred at home, in circumstances where the children spent a proportion of their time away from the home;[164]

    d. all five events occurred during the child's sleep period, rather than whilst playing at home, watching television, in the bath, or in the garden for example;[165]

    e. all five events occurred when the child was in a bed, cot or a bassinet, rather than whilst asleep on the floor, or sitting, standing, running, jumping, skipping, eating or watching television;[166]

    f. all five events occurred when the only person effectively at home or awake was Ms Folbigg, noting that Mr Folbigg was a deep sleeper, which gave her the opportunity to have done the children harm;[167]

    g. each child was discovered dead or moribund by Ms Folbigg;[168]

    h. each child was discovered by Ms Folbigg during what she claimed was a normal check on their well-being during the course of their sleep period, including on three occasions when she said she was on her way to the toilet;[169]

    i. each child was discovered dead or moribund at around or shortly after death when they were still warm to the touch, and two of them still had a heartbeat, so they were found literally minutes after the cessation of breathing;[170] and

    j. in relation to four of the five events, Ms Folbigg failed to render any assistance at all to the children after discovering them dead or moribund, to the extent that she did not even lift them up out of their beds.[171]

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159 13 May 2003 T1359.11-21.
160 13 May 2003 T1359.24-27.
161 13 May 2003 T1362.23-45; Notice of Crown coincidence evidence.
162 13 May 2003 T1362.47-50.
163 13 May 2003 T1362.53-57.
164 13 May 2003 T1363.7-12.
165 13 May 2003 T1363.14-18.
166 13 May 2003 T1363.22-27.
167 13 May 2003 T1363.29-34.
168 13 May 2003 T1363.37-38.
169 13 May 2003 T1363.40-43.
170 13 May 2003 T1363.53-57.
171 13 May 2003 T1364.16-21.

136. It was the Crown case that these features were incapable of being explained except by the common feature of Ms Folbigg, because she was responsible for all the events.[172]

137. The Crown case relied in this regard on evidence from doctors that:

    a. there had never been recorded a family such as this where four children died of natural causes, whether from the same or different causes; and

    b. there had never been recorded three or more deaths in one family from SIDS.[173]

138. The Crown prosecutor explained that these matters did not mean such things had never ever happened, or could not happen, but rather were an expression of their rarity. By reference to Professor Berry's description of the four deaths as like four sudden lightning bolts, he submitted the only reasonable conclusion was that Ms Folbigg had killed them.[174]

139. The Crown prosecutor then sought to explain the difference between coincidence and tendency evidence, which the Crown case also relied on. He explained that coincidence evidence requires the jury to look at all of the cases at the one time to compare the similarities with a view to assisting them to come to a conclusion as to what caused all the events.[175]

140. He described tendency as the jury coming to a conclusion, based on other evidence, that one fact had been proven. He pointed out this required a conclusion on one charge first, then looking at the other charges, in contrast to the coincidence approach which turned on consideration of all the evidence together.[176]

141. The Crown prosecutor suggested the jury would only use the tendency approach if they did not decide all counts by using the coincidence approach. He hypothesised they may be satisfied Ms Folbigg had killed Caleb, Patrick and Sarah by smothering them, but have concern about Laura's myocarditis. He submitted that in that instance the jury could use the fact they had already decided about the other three children, to help come to the conclusion that the myocarditis was an incidental finding and that Ms Folbigg had killed Laura also.[177]

142. In concluding the Crown's submissions about coincidence and tendency, the Crown prosecutor referred to the following additional factors as common to some but not all of the five events:

    a. Patrick's ALTE was three days after Mr Folbigg had returned to work, and Sarah's death was one or two or three days after the sleep monitor was taken off;[178]

    b. Ms Folbigg had shown acute irritation at both Sarah and Laura very shortly before their deaths;[179] and

    c. Ms Folbigg had thought of leaving home in the period shortly before the deaths of Patrick, Sarah and Laura.[180]

172 13 May 2003 T1364.23-28.
173 13 May 2003 T1364.30-35.
174 13 May 2003 T1364.37-58.
175 13 May 2003 T1365.10-14.
176 13 May 2003 T1365.16-18, T1365.52-54.
177 13 May 2003 T1365.34-49.
178 13 May 2003 T1366.1-4.
179 13 May 2003 T1366.6-8.
180 13 May 2003 T1366.8-10.

Ms Folbigg's diaries

143. It was the Crown case regarding the contents of Ms Folbigg's diaries that:

    a. she never thought anybody would ever read them, the entries were only for herself;[181]

    b. they did not have one entry of the kind one would expect from a person who had cruelly lost three children to natural causes;[182] and

    c. they contained repeated ramblings about her tiredness, and her frustrations with the restrictions placed on her by having children.[183]

144. The Crown prosecutor read a number of extracts to the jury.

145. It was the Crown case that the explanations given by Ms Folbigg to police during her interview were unbelievable and unsatisfactory.[184] The Crown prosecutor submitted the diaries were the strongest evidence the jury could possibly have for Ms Folbigg having murdered her four children.[185]

146. It was the Crown case that Ms Folbigg had shown an unusual grief reaction following the children's deaths, consistent with ambivalence on her part. It was suggested her reaction was one of grief, coupled with guilt for the children's deaths.[186]

Anticipated submissions on behalf of Ms Folbigg

147. After having addressed the jury on the evidence in the Crown case, the Crown prosecutor made submissions to the jury about what submissions he anticipated the jury would hear from defence counsel.

148. Firstly the Crown prosecutor identified the submission that the Crown must prove that the children did not die from natural causes, and that the Crown could not prove that they did not die from four incidental findings.[187]

149. In response, the Crown prosecutor submitted that he could not disprove that Caleb may have died from a floppy larynx or SIDS, that Patrick's ALTE was a first epileptic attack or encephalitis, that Patrick's death may have been caused by an epileptic attack or seizure, that Sarah may have had a displaced uvula or SIDS and that Laura may have died of myocarditis.[188] He submitted further:

> *I can't disprove any of that, but one day some piglets might be born from a sow, and the piglets might come out of the sow with wings on their back, and the next morning Farmer Joe might look out the kitchen window and see these piglets flying out of his farm. I can't disprove that either. I can't disprove that one day some piglets might be born with wings and that they might fly. Is that a reasonable doubt? No. Is the hypothesis that the defence advances a reasonable doubt? No. Why not? Because if you look at what they are suggesting, not in isolation, but in totality: There has never ever been before in the history of medicine that our experts have been able to find any case like this. It is preposterous. It is not a reasonable doubt. It is a fantasy, and of course the Crown does not have to disprove a fanciful idea.*[189]

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181 13 May 2003 T1366.30-34.
182 13 May 2003 T1366.49-55.
183 13 May 2003 T1367.12-14.
184 13 May 2003 T1369.9-10, T1370.9-13.
185 13 May 2003 T1372.54-56.
186 13 May 2003 T1373.26-44.
187 13 May 2003 T1375.10-13.
188 13 May 2003 T1375.17-22.
189 13 May 2003 T1375.22-37.

150. Secondly, the Crown prosecutor identified the submission that there were no signs of any physical abuse to the children during their lifetimes, or at the time of their death. In response the Crown prosecutor submitted that the evidence showed that suffocation is easy to do on a young child without leaving signs, and that the accused snapped on the occasions of the events.[190]

151. The Crown prosecutor then anticipated reference to the following:

    a. the evidence in the defence case, from other women at the gym, that the accused was a good mother. The Crown prosecutor suggested the jury would give very little weight to this evidence given the women had no idea about the thoughts expressed in the diaries;[191]

    b. selections from the diary entries where Ms Folbigg expressed joy at having her children, which the Crown prosecutor acknowledged and said her flashes of anger, resentment, bitterness and hatred were not matters she thought all of the time;[192]

    c. the suggestion there was no logical reason that had been advanced in the Crown case as to why Ms Folbigg would kill her children, which the Crown prosecutor acknowledged but suggested did not mean she did not do it;[193] and

    d. the suggestion that the diaries were the ramblings of a grief stricken parent who had lost her three children and should not be read literally, in response to which the Crown prosecutor noted his earlier submissions on the diaries.[194]

152. Neither defence counsel nor the trial judge raised any issues about the Crown prosecutor's closing address.

The defence case as put to the jury

153. Defence counsel commenced the closing address on behalf of Ms Folbigg by referring to the matters he had set out in his opening address. These matters concerned the case as a whole. The address then dealt with the non-medical and medical evidence in relation to each child, before dealing finally with the diaries generally. It was repeatedly noted that the submissions on behalf of Ms Folbigg in the closing would be the same as during the opening address, and that there had been no change in the accused's case.[195]

154. In the first part of the opening address, defence counsel submitted to the jury that the case could not be determined on the mere fact that such circumstances had not yet been recorded in medical history or known to medical practitioners. Nor could the jury reason that Ms Folbigg must be guilty because of the number of charges. It was submitted that to reason by the mere fact of numbers would be a flawed process.[196]

155. Defence counsel described a "tension" between the manners in which the Crown and the defence suggested the jury should look at the evidence.[197] Counsel suggested the Crown said to look at the evidence in overview, whereas the defence said to look at the evidence with precision, which would give rise to disquiet about the evidence.[198]

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190 3 May 2003 T1375.46-52.
191 13 May 2003 T1376.3-13.
192 3 May 2003 T1376.15-23.
193 3 May 2003 T1376.25-37.
194 13 May 2003 T1376.39-42.
195 14 May 2003 T1390.17-20, T1392.11-16, T1393.26-28.
196 14 May 2003 T1383.6-14.
197 14 May 2003 T1383.22-24.
198 14 May 2003 T1383.26-32

156. Counsel accepted that looking at the counts in combination was "part of the process", but suggested that if there was disquiet about the evidence in the individual cases then the jury would not be satisfied beyond reasonable doubt that Ms Folbigg in fact murdered the children.[199] It was suggested there was a danger in using the global view in order to convict in the individual case, shown by going to the detail of the individual case because a lot of what the Crown had put "just does not fit in the individual case".[200]

157. Referring to the Crown prosecutor's closing address, defence counsel submitted that the case was not assisted by statements that pigs might fly or that lightning doesn't strike four times in one place.[201]

158. Defence counsel emphasised it was not a question of weighing up both cases to see which the jury preferred, but rather the Crown bore the onus of proof and that onus was the touchstone in resolving the tension between the Crown case and the defence case.[202]

159. It was noted that the Crown did not have to prove motive in order to establish the offences, but nonetheless a motive had been proffered. Counsel submitted that when looking at the alleged motive critically it was "very, very shaky", and in the additional absence of any history of abuse of the children, the Crown case became "immeasurably weaker".[203]

160. Defence counsel suggested there had been a weakness demonstrated in the Crown case, as the emphasis about a motive for murder in order to go to the gym, socialise and go dancing which was emphasised in the opening address was not emphasised in the closing address.[204]

161. A significant part of the defence case was that Mr Folbigg had tried to paint a picture of Ms Folbigg which contrasted with the detail of the other evidence.[205] This plank of the defence case was expanded upon when discussing the evidence in relation to each child.

162. Defence counsel emphasised it was not a matter for Ms Folbigg to prove that the children had died of natural causes such as floppy larynx, epilepsy, encephalitis or myocarditis. Rather it was for the Crown to prove that she had murdered her children. In doing so the Crown had to exclude other reasonable hypotheses consistent with innocence which were considered by medical experts.[206]

163. Counsel referred to his submission at the start of the trial about the absence of the typical signs of suffocation at autopsy and said this had been borne out by the evidence. None of the experts said there was positive proof of suffocation. He cautioned that the phrase used by the experts "consistent with suffocation", was not consistent with positive proof of suffocation but rather consistent with suffocation because it is possible to have suffocation where there are no indicated symptoms.[207]

164. As to the diaries, defence counsel asked the jury to consider them in detail, and in context.[208] It was the defence case that the diaries exhibited fairly normal reactions not only of grief, but also of shame, guilt and responsibility, though not in the sense contended for by the Crown.[209]

165. The defence case relied on answers during Ms Folbigg's interview with police as giving the jury some insight into

199 14 May 2003 T1383.40-47.
200 14 May 2003 T1384.12-17.
201 14 May 2003 T1383.34-36.
202 14 May 2003 T1383.57-1384.3.
203 14 May 2003 T1386.44-54.
204 14 May 2003 T1387.34-38.
205 14 May 2003 T1388.5-11.
206 14 May 2003 T1388.18-53.
207 14 May 2003 T1389.1-22, T1389.43-50.
208 14 May 2003 T1390.22-25.
209 14 May 2003 T1390.35-44.

her state of mind in relation to those excerpts.[210] This aspect of the defence case was expanded upon towards the end of the closing address.

166. Defence counsel said the Crown case was that the diaries manifested not only a state of mind but also a real propensity for Ms Folbigg to lose her cool, lose control and vent a high level of anger, frustration and stress.[211] The defence case raised for the jury's consideration that there was no corresponding pattern of behaviour, no manifested course of conduct or history of abuse to represent that underlying state of mind. Instead, there was evidence portraying her in a positive light.[212]

167. Mr Folbigg's evidence that the things he read in the diaries about her attitude to the children were not things she had expressed to him before during 16 years of marriage, and not things he had seen, experienced or witnessed before in her prior to reading the entries, was referenced.[213]

168. Before moving to the evidence in relation to each child, defence counsel referred again to the "tension" between the Crown and defence cases, and submitted the jury would have disquiet about the evidence when looking at it in detail.[214] In particular it was said that the absence of motive and the absence of corresponding behaviour raised disquiet, as well as the lack of evidence to support the picture of Ms Folbigg which Mr Folbigg attempted to place before them.[215]

Caleb

169. In respect of Caleb it was the defence case that the motive alleged by the Crown did not fit the evidence. Mr Folbigg's evidence was referred to in this regard. From the time of becoming pregnant with Caleb, Ms Folbigg had ceased going out to night clubs and the couple just mixed within their own family.[216] After Caleb's death, Mr Folbigg said that they "really hunkered down" and just spent time with their family.[217]

170. Defence counsel suggested there was a danger in just looking at the overall picture as urged by the Crown, noting that Ms Folbigg was not attending the gym prior to or during Caleb's life.[218] Further, Mr Folbigg's evidence was that she was happy and ecstatic to be pregnant,[219] and during his 19 days of life Ms Folbigg had been happy to have a child and diligently fussed over him.[220]

171. It was submitted that no evidence was proffered to suggest there was anything about Ms Folbigg's behaviour during Caleb's life that was consistent with the high level of anger and resentment, and capacity and propensity to lose her cool, as was alleged by the Crown.[221]

172. The diaries were referred to in support of the defence case. The entries of "changed nappy", "fed Caleb", "slight spew", and "brought Caleb home!!" were said to be "really quite far removed" from a person who had a supposed low tolerance, frustration and battle of wills. It was submitted that Ms Folbigg had embraced the life of this child, and that Mr Folbigg agreed with this.[222]

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210 4 May 2003 T1390.53-58.
211 14 May 2003 T1391.12-21.
212 14 May 2003 T1391.37-44, T1392.46-1393.4.
213 14 May 2003 T1392.11-44.
214 14 May 2003 T1393.30-33.
215 14 May 2003 T1393.48-58.
216 14 May 2003 T1394.38-42.
217 14 May 2003 T1394.54-58.
218 14 May 2003 T1395.4-13.
219 14 May 2003 T1395.24-29.
220 14 May 2003 T1397.34-42.
221 14 May 2003 T1396.20-25.
222 14 May 2003 T1398.15-23, T1398.33-40.

173. As to the significance which the Crown ascribed to the entry on the night of Caleb's death, "2am finally asleep", defence counsel noted there were in fact a number of "finally asleep" entries throughout the diary.[223] Counsel also noted Mr Folbigg's statement to police that Caleb had been a "very quiet baby" and slept well, contrasted with his reticence in evidence to accept the "very" description.[224] It was the defence case this was an example of the "flavour" of Mr Folbigg's evidence, being pedantic and not wanting to say anything that was favourable to the accused.[225]

174. Counsel again noted nothing in the evidence was consistent with a manifestation of the lack of control and Ms Folbigg losing her cool which the Crown alleged, and submitted in fact there was no evidence of anything out of the ordinary on the night of Caleb's death.[226]

175. Certain aspects of the Crown's coincidence notice were then dealt with. In particular, it was the defence case that the particulars of coincidence needed to be considered in the context of Ms Folbigg's role as the primary carer, and Mr Folbigg's status as a heavy sleeper.[227]

176. As to the specific aspect of a lack of any resuscitation attempt by Ms Folbigg, defence counsel submitted to the jury they could fall into error in applying a test of "what would I do", or "what would the average person do". He submitted they ought to look at a range of human emotions and acknowledge that different people react differently in different situations.[228]

177. Ms Folbigg's explanation to police that she was not experienced in CPR and the ambulance officers' observations that she was hysterical and overcome with grief were noted in this regard. It was submitted that her demeanour was consistent with the panic of realising the child had stopped breathing or was having trouble breathing and would not wake properly.[229]

Mr Folbigg's evidence

178. At this point in the closing address defence counsel dealt at some length with the evidence of Mr Folbigg. It was the defence case that in the jury's assessment of his evidence, they would have disquiet and determine to give it little weight.[230]

179. Defence counsel submitted that excerpts from the listening devices that Mr Folbigg was taken to in cross-examination showed he was motivated by revenge at certain times when he was his making his statements to police.[231] Counsel referred to his recorded statements such as the below, and his denial in evidence that this was his state of mind when he went to the police:

> *I said I went there because I was so full of hate and spite and anxiety and grief and anguish over the fact that not only had I lost my daughter, I'd lost my wife, you know… I was so frustrated, I was hurting, so I thought I'll fucking fix this.*[232]

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223 14 May 2003 T1399.7-9.
224 14 May 2003 T1400.53-1401.7.
225 4 May 2003 T1401.21-25.
226 14 May 2003 T1401.27-52.
227 14 May 2003 T1399.36-49.
228 14 May 2003 T1402.15-1403.42, T14047-36.
229 14 May 2003 T1404.7-19, T1404.38-1405.33.
230 14 May 2003 T1405.35-1408.43.
231 14 May 2003 T1408.45-48.
232 14 May 2003 T1408.53-58.

> *I'll fuck your life. You fucked mine. I'll fuck yours. I will go and tell some fucking horrible thing about you that the police think you did it anyway.*[233]
>
> * * *
>
> *I don't want him going after you for something you didn't do. That you and I know. I knew you didn't do it like you know I didn't do it. But get this sick pack of pricks that, you know, maybe what we're doing now is what he wants us to do, sit there and have factional fights instead of joint cohesive support. I really wanted to pose all that to you to show you're not a wicked person either, but I can be a wicked person, I can be the deviate person, how I sell motor cars. I use whatever I can to my advantage to sell a motor car, whether it's my children's lives or my children's deaths.*[234]

180. Defence counsel pointed to the difference between Mr Folbigg's account in his evidence in chief of Ms Folbigg's grief regarding Caleb, and his account in cross-examination. During evidence in chief his evidence was that he had fallen to pieces whereas she appeared to be coping much better than him. During cross-examination he acknowledged she was a private person with her emotions who never let anything out, and the difference between them was understandable in light of the differences in their personality.[235]

181. Counsel also referred to the cross-examination of Mr Folbigg in which the picture the defence said he had attempted to create of Ms Folbigg going out dancing or discoing after Caleb's death became limited to an "infrequent" occurrence of "no more than two or three occasions".[236]

182. Counsel also referred to Ms Folbigg's own account to police, that if she appeared to be handling her grief after Caleb's death better it was probably because she "blocked and blocked rather than actually just dealing with the situation".[237]

183. It was the defence case that Detective Senior Constable Ryan's evidence could be taken into account in assessing Mr Folbigg's credibility.[238] Whereas Mr Folbigg had said in cross-examination he lied to police in his statement on 23 May 1999 and added that financial reasons were the cause for Ms Folbigg returning to work after Sarah's birth,[239] Detective Senior Constable Ryan's evidence was that on 23 May nothing had been changed in the statement typed on 19 May 1999.[240]

Medical evidence

184. It was the defence case that when looking at the detail of the medical evidence it was clear there was no positive evidence of suffocation, and that the Crown could not exclude certain natural conditions as the cause of death.[241]

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233 14 May 2003 T1409.5-8.
234 4 April 2003 T245.51-246.6.
235 14 May 2003 T1410.13-49.
236 14 May 2003 T1410.51-1411.35.
237 14 May 2003 T1410.37-46.
238 14 May 2003 T1412.6-7.
239 14 May 2003 T1411.23-57.
240 14 May 2003 T1090.9-12; 14 May 2003 T1412.1-4.
241 14 May 2003 T1412.9-14.

185. Defence counsel referred to Professor Herdson's opinion that there was a "quite reasonable conclusion of SIDS", which was "slightly worrying" because of Caleb's young age. Professor Herdson agreed that age did not prevent making a diagnosis of SIDS but was a reason for having his "antennae up a little bit".[242]

186. Next, counsel referred to Professor Berry's opinions that he would "never say never", but it was not his view that Caleb had died of floppy larynx, and that SIDS had primarily been excluded on the finding of haemosiderin which suggested previous bleeding to the lungs within 36-48 hours before death.[243]

187. On the haemosiderin issue, defence counsel pointed to the evidence that Ms Folbigg had taken the child to see Dr Springthorpe two days prior to Caleb's death with concerns about his stridor. It was the defence case that this information raised disquiet about the medical evidence insofar as it provided potential support for a possible asphyxial episode prior to death.[244]

188. Next, defence counsel noted the absence of any indications of physical struggle or any other signs of suffocation or manual asphyxiation, not just in the case of Caleb but in any of the other children. It was noted that the punctate abrasions in respect of Sarah could have been consequent upon resuscitation, which Mr Folbigg had performed. The absence of any pattern of injury was said to be incongruous with the patterns and coincidences alleged by the Crown.[245]

189. Dr Beal's opinion that floppy larynx was excluded as a cause of death, and in isolation the cause of death was "SIDS with the proviso the child was under 3 weeks of age" was referred to. The doctor's acceptance that the child's age and position was not conclusive or determinative of a SIDS diagnosis was noted.[246]

190. Professor Byard's opinions were referred to next. His opinion as to cause of death was "undetermined", because there was no death scene examination, the brain was not examined and the issue of the floppy larynx could not be excluded. His evidence of a study of six babies who presented with respiratory arrest that were diagnosed as having been caused by floppy larynx was noted, as well as his identification of clinical findings (classic stridor) which fit with an upper airway problem. The professor could not agree with the Crown proposition that it was highly unlikely that the floppy larynx had no significance at all in Caleb's death.[247]

191. Defence counsel submitted there was accordingly for the jury the question of whether the Crown could exclude Caleb having died of floppy larynx as a reasonable possibility. This was in addition to the question of what proof there was of suffocation, which it was submitted the medical evidence did not, in isolation, permit.[248]

192. Defence counsel concluded the address regarding Caleb by referring again to the coincidence evidence, submitting it was consistent with Ms Folbigg's role as primary caregiver and noting the particular of children being "warm to the touch" lacked precision.[249] Counsel also noted the absence of anything consistent with the Crown allegation about Ms Folbigg's attitude to Caleb in the diaries, the lack of any injury or pattern of injury, and the contrasting evidence that Ms Folbigg was enjoying motherhood.[250]

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242 14 May 2003 T1412.16-36.
243 14 May 2003 T1412.46-51, T1413.10-19, T1413.41-48.
244 14 May 2003 T1414.2-17.
245 14 May 2003 T1414.34-1415.10.
246 14 May 2003 T1415.12-26.
247 14 May 2003 T1415.50-.55.
248 14 May 2003 T1417.5-11.
249 14 May 2003 T1417.21-38.
250 14 May 2003 T1417.41-55.

193. Finally it was submitted that having made an entry at 2:00am in her diary about getting the child to sleep was incongruous with having murdered the child soon after.[251] It was also submitted that the observation of an ambulance officer upon arrival that the child was being held by Ms Folbigg was inconsistent with the Crown case that she had abandoned him.[252]

Patrick

194. The defence case in respect of Patrick was similar to that in respect of Caleb: that there was no manifestation in the accused's behaviour towards Patrick of the thoughts in her diary.[253]

195. Counsel noted by reference to Mr Folbigg's evidence that it was a happy time for them both when Ms Folbigg fell pregnant, and that effecting renovations to the house based on the SIDS organisation's advice had been a joint effort. They had also both been overcome with happiness about the birth of Patrick.[254]

196. Mr Folbigg's way of answering questions in his evidence on this topic was again referred to, suggesting an attempt by him to minimise any positive indications in relation to Ms Folbigg compared with what he had said in earlier statements.[255]

197. Counsel submitted the evidence was that during Patrick's first three months everything was normal, Ms Folbigg was happy to be a mother, and this was the case when Mr Folbigg returned to work. The night of the ALTE, Mr Folbigg said, seemed like a normal night.[256] It was the defence case that these circumstances did not fit the scenario of using the diaries to demonstrate that Ms Folbigg was a person who had frustrations, and lost her cool and control.[257]

198. Mr Folbigg's evidence that the attitudes in the diary were nothing he had before seen, experienced or witnesses in 16 years of marriage with Ms Folbigg was referred to.[258]

199. In relation to the Crown's coincidence point about the unlikelihood of Ms Folbigg finding the child within a couple of minutes between the time of being unconscious and the time of being resuscitated,[259] it was the defence case that this presupposed that Patrick had stopped breathing whereas there was no evidence that he had in fact stopped breathing.[260]

200. In relation to Mr Folbigg's evidence in chief that after the ALTE Ms Folbigg did not cope very well and lost her temper and became frustrated, defence counsel pointed to his answers in evidence in chief that she was "doing the best she could"[261] and that he could not recall any incident that impacted negatively on her treatment of Patrick.[262]

201. Turning to the day of Patrick's death, defence counsel noted there was nothing in Mr Folbigg's earlier or more recent statements consistent with a manifestation of Ms Folbigg losing her cool or control.[263]

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251 14 May 2003 T1421.23-31.
252 14 May 2003 T1421.33-48.
253 14 May 2003 T1421.50-1422.2, T1425.7-12.
254 14 May 2003 T1422.4-44.
255 14 May 2003 T1422.46-1423.52.
256 14 May 2003 T1424.48-1425.4.
257 14 May 2003 T1424.48-1425.46.
258 14 May 2003 T1425.16-21, T1425.30-36.
259 14 May 2003 T1426.30-50.
260 14 May 2003 T1427.5-23.
261 14 May 2003 T1429.7-33.
262 14 May 2003 T1430.4-23.
263 14 May 2003 T1430.52-1432.7.

202. Defence counsel highlighted Mr Folbigg's acceptance of a difference in grief reactions, and his ultimate evidence that to the extent Ms Folbigg socialised and went to nightclubs and dancing after Patrick's death, this was together with Mr Folbigg. Ms Folbigg did not socialise anywhere on her own between Patrick's death and Sarah's birth.[264]

203. It was the defence case that there was other evidence which pointed towards Ms Folbigg's conduct being incongruent with the state of mind alleged by the Crown by reference to the diaries. In particular, defence counsel referred to the treating general practitioner Dr Christopher Marley's evidence that Ms Folbigg was diligent in attending upon specialists and upon him, that he had expertise in screening for signs of child abuse and nothing made him anxious about her care of the children, and that he had no record or memory of her finding things difficult or that the children were not bonding properly.[265]

204. On this point, defence counsel referred also to the evidence of Ms Folbigg's concern for Sarah being unwell and suggesting a further sleep study be performed.[266] It was submitted that the frequency of her attendances upon various doctors indicated a caring role which was inconsistent with the state of mind and demeanour the Crown invited the jury to conclude from the diaries.[267]

205. As to Ms Folbigg's state of mind about leaving Patrick and Mr Folbigg after Patrick's ALTE, defence counsel submitted consideration of this evidence was not a matter of applying the test of "what would I do" or "what would the average person do".[268] Counsel referred to Mr Folbigg's sister's evidence that Ms Folbigg had expressed concerns to her about not being good enough, which appeared to pass with the assistance of the sister.[269] It was submitted that it was readily understandable in context that a parent looking after a child with particular difficulties would have feelings of inadequacy.[270]

206. In relation to Ms Newitt's evidence that Ms Folbigg had said "don't pick him up" when she attended shortly after Ms Folbigg called her on the day of Patrick's death, counsel referred to the absence of any such statement in her statements to police and her response in cross-examination that Ms Folbigg had in fact just said "no." It was submitted there was no foundation for the Crown submission that Ms Folbigg had attempted to restrict her from picking up the child.[271]

Medical evidence – ALTE

207. It was the defence case that there was division among the experts on the cause of the ALTE, and it was possible that the ALTE was the manifestation of a seizure disorder, or the first manifestation of epilepsy.

208. Professor Berry's and Dr Beal's evidence that it was unlikely to be epilepsy but they would defer to neurologists was noted first.[272] Next it was noted that while Dr Dezordi and Dr Wilkinson appeared to be strong in their evidence about features they said led them to conclude that encephalitis and epilepsy could be ruled out, it was also apparent that there were times when these matters were very clearly considered as explanations.[273]

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264 14 May 2003 T1433.1-22.
265 14 May 2003 T1433.46-1434.-47.
266 14 May 2003 T1436.6-13.
267 14 May 2003 T1436.9-17, T1437.12-24.
268 14 May 2003 T1439.21-57.
269 14 May 2003 T1441.42-56.
270 14 May 2003 T1442.1-9.
271 14 May 2003 T1443.44-55.
272 14 May 2003 T1444.8-1445.35.
273 14 May 2003 T1445.43-1446.4.

209. In this regard, defence counsel referred to the ultimate discharge summary prepared by Dr Dezordi in October 1990 and his opinion that the cause of the ALTE was "probably viral encephalitis".[274]

210. Counsel also referred to a letter from Dr Wilkinson to Mr and Ms Folbigg in September 1991, in which he noted there had been at the time of the ALTE changes of a type that could occur after seizure and encephalitis, or interference with oxygen supply.[275] It was suggested that given Dr Wilkinson could not recall during cross-examination what caused him to now depart from that position despite having all the same material, the jury would have a question as to the degree of confidence with which they could rely on Dr Wilkinson's opinion at trial as the Crown invited them to do.[276]

211. The absence of evidence of any injuries at the time of Patrick's ALTE and his death, and the absence of any physical findings consistent with suffocation, were again referred to as matters casting doubt on whether the Crown could prove murder with no history of abuse and a disconnect of the evidence with the proposed interpretation of the diaries.[277]

212. Finally defence counsel noted the evidence of neuropathologist Dr Kan. Dr Kan initially said there was a relatively remote possibility that the infiltrate identified could have related to encephalitis and then in cross-examination said that encephalitis and seizure are both possible causes of infiltrate.[278]

213. When subsequently dealing with the medical evidence concerning Patrick's death, defence counsel also noted Professor Byard's opinion that he could not exclude the possibility that the ALTE was epilepsy resulting in an asphyxial event.[279] He considered it was possible to observe clinically as a result of a first epileptic fit the amount of brain damage Patrick presented with.[280]

Medical evidence – death

214. It was the defence case that Patrick's death could have been caused by a seizure or an epileptic fit, against the background of seizures that occurred between the ALTE and death. Professor Berry's and Herdson's evidence was referred to for this position.[281]

215. Defence counsel observed there was a difference of opinion between the experts about whether one could have expected in the case of death by seizure the factors Professor Herdson indicated would necessarily be present, such as bite marks on the tongue or lips. Professor Byard's opinion that it was not necessary to find such physical findings was referred to.[282]

216. Dr Beal's opinion that the cause of death was probably asphyxia, being a lack of oxygen, but she could not be certain and could not rule out an epileptic fit, was also referred to.[283]

217. Defence counsel noted Dr Kan's evidence that there was nothing in the neuropathology to exclude the possibility that the ALTE was an epileptic seizure which led to a subsequent seizure disorder causing death. His evidence that such a possibility is very hard to diagnose because often there are not findings on autopsy was highlighted.[284]

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274 14 May 2003 T1446.6-49.
275 14 May 2003 T1447.23-40.
276 14 May 2003 T1447.42-1448.54; 15 May 2003 T1455.53-1456.4.
277 14 May 2003 T1448.56-1449-56.
278 14 May 2003 T1450.4-48.
279 15 May 2003 T1457.14-19.
280 15 May 2003 T1458.8-17.
281 15 May 2003 T1456.19-38.
282 15 May 2003 T1457.4-19.
283 15 May 2003 T1457.27-41.
284 15 May 2003 T1458.8-47.

218. Similarly highlighted was Professor Byard's evidence that in four epileptic patients he had conducted post-mortems on in the past year, each was found to have neuropathology.[285]

219. Defence counsel referred additionally to Professor Byard's query of how Dr Wilkinson excluded epilepsy as a possible cause.[286] It was noted that Professor Byard considered Patrick's death was consistent with the background of the ALTE resulting from stopping breathing, with a subsequent seizure disorder resulting from the initial asphyxiating event. He also considered the death was consistent with a background of encephalitis, and while he deferred to the clinicians he noted that on the pathology he saw he did not think he could exclude encephalitis.[287]

220. It was reiterated that it was for the Crown to both exclude the mechanisms of epilepsy or encephalitis, and prove its own mechanism of murder. Defence counsel submitted that looking at the medical evidence the jury would conclude there was no medical proof that could be used to conclude beyond reasonable doubt that Patrick's death was in fact caused by suffocation.[288] The absence of any signs of struggle or abuse was pointed to.[289] It was said this was a significant barrier to finding Ms Folbigg guilty.[290]

221. Finally it was noted that Professor Byard was cross-examined by the Crown prosecutor at length, but nonetheless maintained that he could not exclude epilepsy as a cause of death because he could not ignore the fact that there had been a history of epilepsy and neuropathological findings to support a damaged brain. Counsel rhetorically asked the jury that given Professor Byard's expertise and unchallenged standing, whether could they exclude his opinion out of hand.[291]

Sarah

222. In respect of Sarah, defence counsel invited the jury to consider where there was anything that manifested in the relationship between the child and Ms Folbigg which might support the conclusions the Crown invited the jury to draw from the diaries.[292]

223. It was the defence case that there were significant alternative views about why Ms Folbigg wanted to have more children after Patrick's death. It was submitted that this decision was inconsistent with the desire the Crown pointed to, namely to lead a socialising, gym-attending lifestyle. Her statements to Mr Folbigg about there being no point in being married unless they had a family were said to be incongruent with her wanting to get rid of her children.[293]

224. Mr Folbigg's observations in his statements to police about Ms Folbigg being happy while pregnant with Sarah were submitted as being incongruent with his attempts to minimise anything positive about her in his evidence, and with the slant the Crown put on the diaries.[294]

225. Defence counsel referred to the sleep apnoea blanket and corometrics monitor used with Sarah. It was the defence case that having regard to the false alarms, it was reasonable for Ms Folbigg to have started to question the utility and reliability of the monitor, and that there was a legitimate reason as to why the blanket was no longer used.[295]

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285 15 May 2003 T1458.49-1459.5.
286 15 May 2003 T1459.31-41.
287 15 May 2003 T1457.43-1458.23, T1460.30-58.
288 15 May 2003 T1460.1-28, T1461.3-17.
289 15 May 2003 T1462.2-22.
290 15 May 2003 T1461.50-54.
291 15 May 2003 T1462.30-43.
292 15 May 2003 T1463.10-16.
293 15 May 2003 T1463.18-1467.4.
294 15 May 2003 T1464.18-46.
295 15 May 2003 T1464.48-1465.25.

226. It was noted that both were prone to false alarms, on a regular basis according to Mr Folbigg's evidence.[296] Defence counsel also noted Mr Folbigg's acceptance that the blanket had caused Ms Folbigg stress,[297] and said the diary entries reflected that the alarm woke the child.[298] Nonetheless, the monitor was used for a period of nine months and stopped only when the child was moved into a single bed which would not feed information down to the blanket. Counsel submitted that weight needed to be given to the evidence that Ms Folbigg persisted with the monitor for nine months.[299]

227. As to Mr Folbigg's observations of Ms Folbigg becoming frustrated and growling, defence counsel submitted that the weight the jury would give to that evidence depended on how they viewed his evidence and its reliability as a whole. Counsel pointed to the evidence of Dr Marley about Ms Folbigg's concern to conduct a further sleep study of Sarah as evidence casting doubt on Mr Folbigg's evidence.[300]

228. It was the defence case that Ms Folbigg's frustrations concerned her belief in the need to regiment Sarah's sleep patterns, and the jury could use its common sense to conclude this was not an unusual parental belief. Counsel suggested there was a tension of views in the family about this issue, as Mr Folbigg's view was that the child would fall asleep when she was tired.[301] Counsel queried whether the jury could elevate the evidence of Ms Folbigg's frustration about Sarah's sleeping problems to proof of murder, and suggested it would be a significant step to use the evidence in that way.[302]

229. Dealing with the night of Sarah's death, defence counsel suggested it was clear there was some tension between Ms and Mr Folbigg that evening. He invited the jury to consider whether this tension could be elevated to some proof of murder, or whether this night was in fact no different from what might otherwise be an understandable tension in putting the child to sleep.[303]

230. It was the defence case that the jury could not make a finding of fact other than that this evening was no different to the usual tension that existed in the rearing of Sarah.[304]

231. Counsel referred to Mr Folbigg's evidence that the family had had an enjoyable day out, that Sarah was wound up from the day as Mr Folbigg had a tendency to wind her up, and that Ms Folbigg had taken the child to put her to sleep.[305]

232. It was noted that during the course of his evidence in chief about what happened next, Mr Folbigg became very emotional and said it was a very dramatic incident. He described hearing Ms Folbigg trying to comfort Sarah who was crying, and then hearing Ms Folbigg growl. He said he went into the bedroom and saw Ms Folbigg had Sarah pinned down in a one-armed bear hug, slapping her bottom.[306]

233. Defence counsel submitted it was significant that this description of events in the bedroom had not been included in Mr Folbigg's initial statement to police and did not manifest until much later.[307]

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296 15 May 2003 T1464.51-1465-3.
297 15 May 2003 T1465.23-33.
298 15 May 2003 T1465.35-53.
299 15 May 2003 T1466.20-29, T1466.53-57.
300 15 May 2003 T1467.18-28.
301 15 May 2003 T1467.45-56.
302 15 May 2003 T1467.45-1468.18, T1469.1-9.
303 15 May 2003 T1471.7-42.
304 15 May 2003 T1471.44-57.
305 15 May 2003 T1472.8-17.
306 15 May 2003 T1472.23-35.
307 15 May 2003 T1472.42-1473.41.

234. In relation to Mr Folbigg's evidence that Ms Folbigg had then thrown Sarah at him, defence counsel pointed to his earlier having told a solicitor at the Office of the DPP that he had over-exaggerated this detail and that it was not true that Ms Folbigg had thrown the child.[308]

235. It was the defence case that a conversation between Mr and Ms Folbigg captured on the listening device at their home showed this was not what occurred. In the context of Ms Folbigg returning home from making her record of interview with police, it was submitted that Ms Folbigg was indignant about what Mr Folbigg had told the police suggesting that she had thrown the child. It was the defence case that contrary to Mr Folbigg's evidence that she was telling him what to say, the listening device demonstrated that she was in fact telling him to "go back and tell the truth".[309]

236. Counsel submitted that Mr Folbigg's explanation for these statements that he was "covering his arse" would cause the jury to have difficulty accepting his account of Ms Folbigg throwing the child and elevating it in the way the Crown suggested.[310]

237. A listening device conversation between Mr Folbigg and a friend in which Mr Folbigg said "he [Detective Senior Constable Ryan] come and planted some bullshit in my head when I was at me lowest point there when Kath had left me" was also referred to.[311]

238. It was the defence case that Mr Folbigg's statements to the effect of "why would I want to get back together with Kathy if I thought she had done this" were inconsistent with Mr Folbigg's observations of Ms Folbigg as given at trial.[312]

239. On the issue of Mr Folbigg's evidence about whether or not Ms Folbigg and Sarah were out of the bedroom at 1:00am, defence counsel submitted that there were so many versions of this particular time sequence from Mr Folbigg, and that there was in fact no foundation at all to conclude as the Crown invited that Ms Folbigg was out of the room at that time.[313] Mr Folbigg's accounts that he could not be sure, and that he assumed this was the case, were referred to.[314]

240. Defence counsel also noted that the first time he told anyone Sarah was out of the room at 1:00am was in May 1999.[315] It was submitted that it would have been logical at the time for Mr Folbigg to have said to those attending the scene at the time of her death, "my wife was out of bed with her just 20 minutes ago, ask her if she was okay then", if that had been the case.[316]

241. In relation to the lack of resuscitation attempt by Ms Folbigg raised as a coincidence point by the Crown, defence counsel noted the evidence from a number of sources, including attending ambulance officers, that Ms Folbigg was extremely upset.[317]

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308 15 May 2003 T1473.43-45, T1474.12-25.
309 15 May 2003 T1474.53-1476.1.
310 15 May 2003 T1475.32-51-8.
311 15 May 2003 T1476.10-17.
312 15 May 2003 T1478.28-34.
313 15 May 2003 T1478.36-54.
314 15 May 2003 T1479.48-54.
315 15 May 2003 T1482.30-32.
316 15 May 2003 T1482.46-54.
317 15 May 2003 T1485.6-1486.6.

242. Regarding Ms Folbigg's grief after Sarah's death, counsel referred to Mr Folbigg's statements to police which confirmed they were both devastated.[318] Counsel also referred to Mr Folbigg's acknowledgment in cross-examination that any reference to Ms Folbigg "going out" only came after their separation about five or six months after Sarah's death, and that the first time she went to the gym was after she had been to Jenny Craig with Ms Newitt.[319] Counsel also noted Mr Folbigg's evidence that he also went out and socialised, and that he and Ms Folbigg had maintained contact during their separation.[320]

243. The defence submitted that there was no evidence other than that of Mr Folbigg about Ms Folbigg growling and being unable to cope. Dr Marley's evidence of a good relationship between Ms Folbigg and the children was again referred to. It was the defence case that there was nothing upon which the jury could safely conclude that there was a manifestation of behaviour, during the life of Sarah, of the thoughts that the Crown said should be interpreted when the jury looked at the diaries.[321]

Medical evidence

244. Defence counsel first referred to the evidence of Professor Hilton. Professor Hilton's expertise was referred to at length, including that he had been a pathologist for over 30 years, had conducted more than 2,000 autopsies on babies, and was at one stage the chairperson of a SIDS international pathology committee.[322]

245. It was submitted it was important for the jury not to dismiss Professor Hilton's opinions, given these were soundly based on his experience, that he was aware of the previous deaths and that he continued to give evidence saying his examination was thorough.[323]

246. In relation to the punctate abrasions on Sarah not being photographed, it was the defence case that this was a distraction issue. Defence counsel submitted this issue was not a basis on which to dismiss Professor Hilton's opinion out of hand.[324] It was further submitted that it was not for Ms Folbigg to exclude the possibility that the abrasions may have been the result of suffocation because photographs were not taken.[325]

247. Defence counsel warned the jury it would be dangerous to speculate as to what the unphotographed injuries might have been. Counsel noted there was no other opinion to challenge Professor's Hilton's view that the abrasions were not relevant, noting again his 30 years of pathology experience.[326] Counsel submitted it would be wrong to start to speculate otherwise, and noted there was nothing to suggest the marks might have been other than marks left during the course of attempted resuscitation.[327]

248. It was the defence case that the evidence must remain that there was no injury, in which case there was nothing that was determined at autopsy which amounted to any medical proof of suffocation.[328] The absence of any injuries typical of suffocation on any of the four post-mortems was again referred to.[329]

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318 15 May 2003 T1486.42-48.
319 15 May 2003 T1487.14-43.
320 15 May 2003 T1487.44-58.
321 15 May 2003 T1488.12-38.
322 15 May 2003 T1490.40-1491.20.
323 15 May 2003 T1491.22-26.
324 15 May 2003 T1491.28-47.
325 15 May 2003 T1492.9-12.
326 15 May 2003 T1492.17-25, T1492.49-52.
327 15 May 2003 T1492.32-36.
328 15 May 2003 T1493.23-31.
329 15 May 2003 T1494.4-6.

249. Defence counsel noted Professor Hilton's view that the uvula was somewhat of an incidental finding. Counsel submitted that what was important from Professor Hilton's evidence was that there was no positive medical evidence of suffocation and the death was consistent with SIDS,[330] because there were no negative findings.[331]

250. The opinions of other experts were also referred to as follows:

    a. Professor Herdson's view that the pathology most closely resembled that of SIDS, that the punctate abrasions "ring alarm bells", and that he was "lost because I don't really know what the nature of trauma was";[332]

    b. Professor Berry's acknowledgment that abrasions on children who have died suddenly and unexpectedly are often caused by a parent in panic or in resuscitation efforts, and his ultimate opinion that in isolation the cause of death was SIDS with slight misgivings about age;[333]

    c. Dr Beal's opinion of a non-specific diagnosis, either undetermined or SIDS;[334] and

    d. Professor Byard's view that the question of the uvula remained undetermined, and that he could not exclude the possibility of SIDS.[335]

251. Finally, defence counsel again reiterated there were no medical symptoms in this individual case which amounted to suffocation.[336]

Laura

252. The defence case in respect of Laura again was that the evidence did not fit the pattern of Ms Folbigg being a person who lost control and had a high level of anger as the Crown said the jury would glean from the diaries.[337]

253. Defence counsel pointed to Mr Folbigg's evidence about the circumstances leading up to Laura's birth. Counsel referred to Mr Folbigg's account that she was good at pushing his buttons in respect of his recollection of the fun of babies and having babies around, and that being a father was the best time of his life. It was submitted this was inconsistent with the picture he painted in his evidence of her not coping as a parent and growling every day, which suggested there were no positive buttons to push insofar as his memory of their time with children.[338]

254. It was the defence case that on Mr Folbigg's evidence, Ms Folbigg was happy at the time of her pregnancy with Laura, that she stopped going to the gym approximately three months into the pregnancy and that it was a happy time when Laura was born.[339]

255. As to the use of the corometrics monitor with Laura, defence counsel noted this was also prone to false alarms and submitted that it was reasonable for Ms Folbigg to question the utility of the device and note in her diary that she knew the alarms were false.[340]

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330 15 May 2003 T1494.8-20.
331 15 May 2003 T1494.31-35.
332 15 May 2003 T1495.13-17.
333 15 May 2003 T1495.44-51, T1496.20-23.
334 15 May 2003 T1496.28-39.
335 15 May 2003 T1496.44-53.
336 15 May 2003 T1496.55-57-1497.5.
337 15 May 2003 T1497.7-14.
338 15 May 2003 T1497.40-55, T1498.19-34.
339 15 May 2003 T1498.58-1499.11.
340 15 May 2003 T1499.22-36.

256. Defence counsel noted in regards to the Crown's coincidence point that Ms Folbigg was the one who found each child, that during Laura's life Mr Folbigg was working five and a half days a week and Ms Folbigg was doing everything at home.[341]

257. Counsel noted a contrast between Mr Folbigg's statement to police that Ms Folbigg "appeared to get stressed occasionally looking after Laura" and his evidence which evolved to an account that Ms Folbigg was cranky every day. Counsel submitted the jury would question whether it could elevate his evidence to a strand of proof that Ms Folbigg in fact murdered Laura.[342]

258. It was the defence case that the letters indicating Ms Folbigg's desire to leave Mr Folbigg and take Laura with her did not make sense in the Crown story of Ms Folbigg wanting to rid herself of the obligations of a family and child.[343] It was further submitted that the couple's marriage was on the rocks, according to the letters, not because Ms Folbigg wanted to leave Laura but because of poor communication between her and Mr Folbigg.[344]

259. Counsel also noted the evidence that Ms Folbigg's return to the gym in March 1999 was precipitated by a girlfriend encouraging her, in contrast to the Crown's picture of her having murdered Laura because she wanted to go to the gym and go out.[345]

260. Defence counsel dealt at length with Mr Folbigg's evidence of the three days leading up to Laura's death. It was the defence case that having regard to the circumstance that his statement outlining these three days was only made for the first time four months before the trial, Mr Folbigg was attempting to portray normal domestic situations in a sinister light. Counsel noted there was no other evidence for what the Crown relied on to set the picture or foundation for an alleged murder.[346]

261. The following matters were highlighted as differences in Mr Folbigg's accounts which bore on his credibility and displayed an evolution in his versions which sought to make events more sinister:[347]

    a. his account in evidence at the trial of the circumstances in which Ms Folbigg attended his workplace on the morning of Laura's death (that he said to her "if you say she's fine and you're fine come and have morning tea with me", compared with his earlier account which did not suggest any such direction but rather was "do you want to come down here for the morning", to which she replied "yeah no worries");[348]

    b. his account in his statement four months before the trial that Ms Folbigg told him "I lost it with her", which was not in the original statement he made to police in May 1999 or in the conference he had with the Crown in October 2002;[349]

    c. his account in his statement four months before the trial that Ms Folbigg had pinned Laura down and was force-feeding her, which was not in the original statement, and his account that the statement was four months prior to trial was the first time he was given an opportunity to talk in terms of the child being pinned down;[350] and

341 15 May 2003 T1500.34-46.
342 15 May 2003 T1500.53-1501.41.
343 15 May 2003 T1501.51-1502.6.
344 15 May 2003 T1502.18-34.
345 15 May 2003 T1505.31-43.
346 15 May 2003 T1502.54-1503.27.
347 15 May 2003 T1505.13-20.
348 15 May 2003 T1503.29-46.
349 15 May 2003 T1504.1-13.
350 15 May 2003 T1504.18-39.

d. similar submissions were made in relation to the evidence of Ms Bown. It was submitted that Ms Bown's descriptions of Ms Folbigg's behaviour towards Laura had also become exaggerated. Her account that Ms Folbigg was "a bit angry" and "short tempered" was contrasted with her trial account which described "yanking out of the chair" and "over the top".[351] Changes in Ms Bown's account of Ms Folbigg's reaction in the car after Laura's funeral were also referred to.[352] Counsel submitted the jury would be cautious about assessing what weight to give to Ms Bown's account of events at trial.[353]

262. Defence counsel referred to the evidence of Kerry Anderson, gym *crèche* worker, and other witnesses in the defence case and submitted this evidence of what occurred at the gym on the morning of Laura's death suggested there was no tension or frustration, or any venting of a high level of anger or frustration on the part of Ms Folbigg at that time.[354]

263. It was noted that the Crown had called witnesses about Ms Folbigg leaving Laura with other persons, but then made no real issue of the evidence in the Crown's closing address. It was submitted this was a matter about which the jury would not reach any adverse conclusion, as the arrangements were always made appropriately and there was no suggestion Ms Folbigg had left the child haphazardly in the care of others.[355]

264. Finally before dealing with the medical evidence, defence counsel invited the jury to listen to the emergency 000 call and consider whether it was the sound of a person who had just murdered her child. Counsel submitted this was a very graphic piece of evidence which suggested completely otherwise, and also addressed the issues of coincidence evidence.[356]

Medical evidence

265. In dealing with the medical evidence, defence counsel noted first there was no positive evidence of suffocation. Counsel submitted this was particularly interesting because Laura was a child who, given her age, might clearly be expected to be able to struggle.[357] Counsel noted that Dr Cala had recognised the significance of this and in fact performed a facial dissection to investigate further.[358]

266. In relation to the myocarditis found on autopsy, counsel noted Dr Cala had concluded this was not the cause of death based on macroscopic and microscope views of Laura's heart.[359]

267. Defence counsel submitted there were quite significant findings of myocarditis. He noted myocarditis was present in all samples taken by Dr Cala, and necrosis was observed on the slides, and there were aggregates of lymphocytes in certain areas inside and on the surface of the heart.[360]

268. Counsel noted it was not a matter for Ms Folbigg to prove death due to myocarditis, but rather it was a question of whether the Crown could exclude death by reason of myocarditis and if so then whether there was positive evidence of suffocation. Counsel submitted there was no positive evidence of this.[361]

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351 15 May 2003 T1507.4-25.
352 15 May 2003 T1507.38-44.
353 15 May 2003 T1507.24-36.
354 15 May 2003 T1506.13-22.
355 15 May 2003 T1507.56-1508.25.
356 15 May 2003 T1508.40-1509.4.
357 15 May 2003 T1509.6-21.
358 15 May 2003 T1509.21-52.
359 15 May 2003 T1510.2-13.
360 15 May 2003 T1510.29-1511.15.
361 15 May 2003 T1511.17-34.

269. The opinions of other experts were referred to as follows:

    a. Professor Berry's opinion that most pathologists would probably describe Laura's death as due to myocarditis in isolation, and that it was not a question of the quantity of myocarditis. It was submitted that even smaller amounts of myocarditis could lead to death;[362]

    b. Dr Beal's evidence that the significance of myocarditis was a matter for pathologists such as Professor Byard and others;[363]

    c. Professor Hilton's opinion that myocarditis could have been the cause of death;[364]

    d. Professor Byard's view, having regard to the slides, that the myocarditis was quite well established and quite well spread such that he could not exclude death being the result of myocarditis. His disagreement also with Dr Cala that there should necessarily be macroscopic findings on autopsy if death is the result of myocarditis, and that myocarditis was common in autopsies. It was submitted that the jury could not dismiss his views in light of his expertise, particularly given Dr Beal had said she would defer to him and specifically noted his experience in looking at slides;[365] and

    e. Dr Jones' view that the agonal rhythm was a very difficult foundation upon which to conclude there was a loss of breathing before cardiac arrest and it could not be used to support a finding that that was the sequence of what had occurred.[366] Also his evidence that there are cases where it does not necessarily follow that you would expect to find macroscopic findings if one died suddenly with myocarditis.[367]

Ms Folbigg's diaries

270. It was the defence case that Ms Folbigg's diary entries were expressions of feelings she had of blame, because of what might be recognised as a range of human emotions.[368]

271. Defence counsel invited the jury to reflect on certain parts of Ms Folbigg's answers in her interview with police when she was asked questions about the diaries. Counsel submitted these answers indicated expressions of guilt or blame, but not actual guilt or blame for having murdered the children.[369]

272. In this regard, the following excerpts from the interview were some of those referred to during the defence closing address: [370]

> *In relation to Sarah, that is the day I will probably recriminate for the rest of my life that I wasn't sort of awake when I got out of, when I got out of bed.*[371]

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362 15 May 2003 T1511.39-55.
363 15 May 2003 T1512.4-8.
364 15 May 2003 T1512.24-31.
365 15 May 2003 T1512.48-54, T1513.14-20.
366 15 May 2003 T1513.25-37.
367 15 May 2003 T1513.39-52.
368 15 May 2003 T1513.54-1514.24.
369 15 May 2003 T1514.26-31.
370 15 May 2003 T1514.42-1515.32.
371 Exhibit E, ERISP of Kathleen Folbigg Q308.

> *We were really enjoying, she [Laura] was getting to that next sort of child stage, not sort of, she was getting out the baby bit that was so frightening to us and into more of the toddler child sort of thing… so we relaxed, which is again something that Craig and I have to live with… we started to relax and think everything was okay.*[372]
>
> * * *
>
> *I felt I should have tried hardest this time because I felt the last few times that I had the opinion I hadn't tried hard enough, something I should have done or hadn't done or should have done. I felt I needed just to try and pay more attention and not miss anything.*[373]
>
> * * *
>
> *You can't have something like this happen without one lookin' at the other and saying is there a blame of some kind to be held? I was sort of thinking to myself I've got the look of blame every time, or to me I thought I had the look of blame every time.*[374]
>
> * * *
>
> *As in have an angry thought here or there. When Sarah wouldn't go to sleep sure the battle of wills would kick in, the frustration would kick in and yes I would have an angry thought but it was never to harm her it was always why wasn't Craig there to help me. So I sort of decided that stress must have been the trigger for all that and we sort of knew for all the stress that come with Laura, so it was a case of being pre prepared for all that and hopefully I wouldn't, you know, get the odd angry thought or be frustrated and go from there.*[375]

273. It was the defence case that Ms Folbigg's answers showed insight into the root cause of her feelings of blame, namely her feeling of being a failure, becoming too relaxed and complacent, and questioning whether she could have done more to avoid the children's deaths.[376]

274. Defence counsel asked the jury to look at the video of the interview for Ms Folbigg's demeanour, particularly around questions 717-726 where, it was submitted, she expressed real emotion and sentiments of thinking she was a terrible mother because it was not possible to have something like these events happen without thinking it had something to do with you. Ms Folbigg's statement that when Laura arrived she barely stayed in the house because she was afraid of the terrible things which kept happening while she was by herself was specifically referred to.[377]

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372 Exhibit E, ERISP of Kathleen Folbigg Q333.
373 Exhibit E, ERISP of Kathleen Folbigg Q604.
374 Exhibit E, ERISP of Kathleen Folbigg Q677.
375 Exhibit E, ERISP of Kathleen Folbigg Q702.
376 15 May 2003 T1515.16-24.
377 15 May 2003 T1516.15-32.

275. It was again submitted that the jury should consider the detail of the evidence. Defence counsel suggested it would be very difficult to conclude that the deaths of the children could be understood on the basis of the diaries alone.[378] Counsel also submitted it was not just a matter of concluding that it was not possible for four children to die of natural causes so Ms Folbigg must have murdered them.[379]

276. Defence counsel reiterated finally that the onus of proof was on the Crown and not Ms Folbigg. He submitted that when the jury paid attention to the detail of the evidence, they would have misgivings and disquiet, and could not return verdicts of guilty on any of the counts.[380]

277. Neither the Crown prosecutor nor the judge raised any issues about the defence closing address.

The judge's summing-up to the jury

278. At the commencement of the summing-up the judge noted he would not be making any detailed reference to the evidence. His Honour observed that the Crown prosecutor in his closing address did not refer to the detail of the evidence, and that defence counsel had taken a different approach and read much of the detail of the evidence from the transcript.[381]

279. The trial judge explained to the jury once again their role compared with his role.[382] He directed the jury about the need to lay their emotions aside so that those feelings would not play any part in their decision. His Honour reiterated it was the jury's duty to apply themselves and return verdicts only on the evidence presented in the course of the trial, and the submissions of counsel based on that evidence.[383]

280. The trial judge then gave the jury directions about:

    a. the assessment of reliability and credibility of evidence and of witnesses;[384]

    b. drawing inferences from direct evidence, emphasising the importance of not rushing to inference too quickly and not speculating;[385]

    c. the nature of circumstantial evidence, emphasising that the Crown relied entirely on circumstantial evidence;[386]

    d. the onus of proof;[387] and

    e. an accused's right to silence.[388]

378 15 May 2003 T1516.45-48.
379 15 May 2003 T1516.56-58.
380 15 May 2003 T1517.21-24.
381 19 May 2003 T1.
382 19 May 2003 T1-2.
383 19 May 2003 T3-5.
384 19 May 2003 T9-11.
385 19 May 2003 T11-13.
386 19 May 2003 T13-14.
387 19 May 2003 T15.
388 19 May 2003 T15.

281. In summing-up, the trial judge said that the fundamental issue was whether each event happened naturally or by human intervention. His Honour said human intervention could only have been by Ms Folbigg, and the evidence permitted only one conclusion or the other.[389]

282. His Honour noted that apart from the circumstances immediately surrounding the events giving rise to any charge the jury was considering, the jury was entitled when deciding whether the Crown had proved its case on that charge to take into account the events giving rise to the other charges as well. His Honour noted it was the Crown case that there was a remarkable degree of similarity in the five events.[390]

283. The judge directed the jury that the law was that sometimes there may be such a striking similarity between different events that a jury may safely conclude that they did not all happen by coincidence.[391] His Honour said that if, having considered the submissions of the Crown and the defence, the jury came to the view that the five events or any number of them were so strikingly similar that they could not all have happened naturally, the jury were entitled to take that conclusion into account in considering whether the Crown had proved its case on the charge under consideration.[392]

284. His Honour specifically warned the jury that they must not say that simply because Ms Folbigg killed a particular child or caused Patrick's ALTE that she must have killed all the children and caused Patrick's ALTE. His Honour said this was an unfair way of approaching the matter and directed the jury not to use it.[393]

285. In relation to the expert witnesses, the trial judge directed the jury to treat expert witnesses as they would any other evidence in the case, weighing their evidence against all the other relevant evidence, in light of submissions from each counsel.[394]

286. The trial judge noted that the experts were not permitted to say what his or her opinion might have been about the probable cause of death of any child, or about Patrick's ALTE, after taking into account also the fact that the other children died unexpectedly. His Honour noted the jury were not confined in the same way, and were entitled to take into account the unexpected deaths of the other three children and Patrick's ALTE, and all the circumstances surrounding those deaths and the ALTE. [395]

287. His Honour also directed the jury that they were entitled to take into account all the other evidence in the case, particularly the entries made by Ms Folbigg in her diaries from time to time, and any meaning they attributed to those entries.[396]

288. The trial judge then directed the jury as to the separate counts, dealing in each case with the circumstances of the death/ALTE, the medical evidence, the other evidence and each counsel's submissions about the evidence.[397]

289. The trial judge said that there were four possible causes of death of any of the children: identified natural causes, unidentified natural causes, accidental suffocation and deliberate suffocation, although there was no evidence of accidental suffocation.[398]

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389 19 May 2003 T15-16.
390 19 May 2003 T16.
391 19 May 2003 T16.
392 19 May 2003 T16-17.
393 19 May 2003 T17.
394 19 May 2003 T17.
395 19 May 2003 T18-19.
396 19 May 2003 T19.
397 19 May 2003 T27-52 (Caleb), T53-68 (Patrick ALTE), T68-77 (Patrick death), T77-89, T95-98 (Sarah), T98-107 (Laura).
398 19 May 2003 T22.

290. The trial judge directed the jury that there were three possible explanations for Caleb's death: floppy larynx, unidentified natural causes and deliberate suffocation by Ms Folbigg.[399]

291. In relation to SIDS the trial judge said:

> *SIDS is not a cause of death. It is not a disease. The fourth letter of the acronym is the initial letter of the word "syndrome" but it appears that SIDS is not a syndrome either. It is a diagnosis by way of a description which is applied where a child of appropriate age has died suddenly and unexpectedly, where unnatural causes have been excluded or are not suspected and where the cause of death cannot be identified.*
>
> *The various expert witnesses have given their own definitions of what is meant by the expression, but I think that that fairly summarises the way the expression has been used in this trial. SIDS has been called a diagnosis of exclusion. That means that the diagnostician excludes unnatural causes and known natural causes of death and concludes that the child died naturally in a way that cannot be explained.*
>
> *The evidence is that there is no established relationship between apnoea and SIDS and no established relationship between ALTE and SIDS.*[400]

292. The trial judge went on to discuss the expert evidence about the rarity of multiple SIDS deaths in the same family, saying:

> *You may have wondered whether the fact that all the children had the same biological father and mother might explain or go some way towards explaining why they all died suddenly and unexpectedly. The evidence of Dr Cooper was that until ideas changed after 1990 it was believed that SIDS ran in families and that if a family suffered a SIDS death the chances of any subsequent child of that family dying suddenly and unexpectedly of natural causes was greater than in the community generally. The evidence of Dr Cooper is that that is no longer the belief of experts in the field. He says that the opinion now is that there is probably no increased risk at all…*
>
> *SIDS deaths are rare in the community. There is no authenticated record of three or more such deaths in a single family. This does not mean, of course, that such events are impossible. It is an illustration of the rarity of deaths diagnosed as SIDS.*[401]

293. The trial judge then discussed the use of the terms "undetermined" and "unascertained", used to mean that the deaths could not be explained by reference to natural causes and that unnatural causes were suspected or could not be excluded. His Honour said:

> *The difference between the allocation of the term "SIDS" and the term "undetermined" is that with SIDS those making the diagnosis have no reason to believe that the cause of death might be unnatural.*[402]

399 19 May 2003 T22-23.
400 19 May 2003 T23-24.
401 19 May 2003 T24-25.
402 19 May 2003 T25.

294. The trial judge noted the general medical opinion about which there seemed no dispute was that except where there are obvious (physical) signs of deliberate or accidental suffocation, "it is virtually impossible to distinguish between a death resulting from asphyxiation and a death resulting from natural but unidentified causes."[403]

295. The trial judge went on to clarify the use of the terminology "consistent with", stating:

> *When a particular condition is established on a post-mortem examination to exist, the pathologist will be able to say whether that condition could have come about from a particular cause. There that pathologist is speaking of a mere possibility. That seems to be the same as saying that the sign or condition found is consistent with having been caused in the manner postulated. The expression often used, that the condition is not specific for that cause, simply means that the proper medical conclusion to draw is that the postulated condition could have been the cause of the condition, but not that it must have been, or very likely or probably was, so that an opinion that a condition is consistent with a particular cause implies that it might also be consistent with another cause.*[404]

296. The trial judge noted there was no evidence that any of the environmental factors mentioned in relation to SIDS might have had any bearing upon the events resulting in the death of Caleb or any of the other children, or Patrick's ALTE.[405]

297. His Honour also referred the jury to the evidence of Dr Wilcken, that a large number of possible natural causes of death had been excluded so that many or all of the likely candidates as a cause of death by way of infection or metabolic or genetic causes were excluded.[406]

298. In explaining why he was not engaging in a detailed review of the evidence, his Honour referred to the Crown's summary of the medical evidence. He noted this summarised the medical evidence concerning the children, was produced by the Crown, and dealt only with the Crown witnesses.[407]

299. The trial judge then dealt with each counsel's submissions in relation to Caleb's death.[408] His Honour said that if the jury were satisfied that Ms Folbigg smothered Caleb, thereby killing him, then:

> *that is a matter which you can take into account in considering whether the accused realised that if she went on with the act of smothering Patrick he would probably die... her realisation might be informed by what happened before.*[409]

300. The trial judge went on to discuss the elements of the offence of murder and the alternative verdict of manslaughter available for consideration if the jury were not satisfied on the element of intention for the charge of murder.[410] The judge gave to the jury a set of written directions regarding the elements of the offences and a list of "yes/no" questions referrable to the elements to assist consideration of its verdicts.[411]

301. In relation to Patrick's ALTE, the trial judge said there was a dispute about whether the damage to Patrick's brain was caused by an act of Ms Folbigg or whether what happened was a spontaneous epileptic seizure.[412]

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403 19 May 2003 T25-26.
404 19 May 2003 T26-27.
405 19 May 2003 T27.
406 19 May 2003 T28.
407 19 May 2003 T28.
408 19 May 2003 T35-43.
409 19 May 2003 T77.
410 19 May 2003 T43-49.
411 19 May 2003 T49.
412 19 May 2003 T53, T56-57, T62-66.

302. In relation to Patrick's death, his Honour directed the jury that it seemed possible that they may take the view that his epileptic condition may have played a part in his death or rendered his death easier or swifter at the hands of Ms Folbigg, and that if they did, they may convict her of the murder or manslaughter of Patrick.[413]

303. The jury were told that to avoid the double counting of any action by Ms Folbigg in relation to Patrick's ALTE also causing his death, to find her responsible for Patrick's death they must be satisfied that she smothered him on the day of his death.[414]

304. In the course of summing-up in relation to Sarah's death, the trial judge set out at length Mr Folbigg's evidence and versions to police, as well as the evidence about the statement-taking process from the police officers who interviewed Mr Folbigg.[415] His Honour set out the defence case that Mr Folbigg's evidence was exaggerated and unreliable,[416] and the Crown case that the differences in his accounts were understandable in light of the difficulties in the marriage at the time.[417]

305. Upon the request of defence counsel,[418] his Honour subsequently gave the jury a warning in relation to Mr Folbigg's evidence. He said that they should scrutinise his evidence very carefully before deciding whether to act on it and if so, what weight to put on it.[419]

306. In relation to Laura the trial judge directed the jury there were only two possible causes of death: smothering or myocarditis.[420] His Honour observed that none of the experts were prepared to say that myocarditis could be absolutely excluded, and in isolation, an expert might well ascribe the death to myocarditis.[421]

307. The trial judge then recapped the respective approaches taken by counsel. His Honour referred to the Crown's submissions in relation to the diaries. The jury were reminded of the diary entries specifically referred to by the Crown prosecutor, and the interview questions which were specifically referred to by defence counsel.[422]

308. The trial judge told the jury they should look very carefully at the detail of the circumstances attending each of the five events as defence counsel submitted, and also look at the picture overall as shown by the other events and explained by the diaries (if the jury thought the diaries provided explanation) as the Crown prosecutor submitted.[423]

309. In relation to tendency, his Honour directed the jury that if they were satisfied beyond reasonable doubt that on any of the five occasions Ms Folbigg smothered her child, they could take her conduct into account when they considered whether she was guilty on any other count. He directed the jury on the circumstances in which they could employ such reasoning.[424]

413 19 May 2003 T70.
414 19 May 2003 T71-72.
415 19 May 2003 T79-83.
416 19 May 2003 T83-84.
417 19 May 2003 T84-86.
418 19 May 2003 T89-90, T92-95.
419 19 May 2003 T96-98.
420 19 May 2003 T98-99.
421 19 May 2003 T99.
422 19 May 2003 T109-112.
423 19 May 2003 T113.
424 19 May 2003 T113-114.

310. At the request of the Crown and over the objection of defence counsel,[425] in relation to the Crown's submissions that parts of Ms Folbigg's interview with police were lies exposing consciousness of guilt, the trial judge directed the jury as to what they must be satisfied of in order to so find. He directed the jury further that if they were satisfied she had deliberately lied they could use that finding in aid of other evidence in the Crown case.[426]

311. At the request of defence counsel,[427] the trial judge also told the jury they should take into account the condition in which Ms Folbigg might have been when she gave the relevant answers, noting the interview went for nine hours altogether and that these answers were much closer to the end of the interview than the beginning.[428]

312. At the request of defence counsel,[429] the trial judge also directed the jury that they must exclude any rational hypothesis consistent with innocence in order to find Ms Folbigg guilty.[430]

Post-trial proceedings

Sentence proceedings and appeal

313. A sentence hearing was held on 29 August 2003. On 24 October 2003, Barr J sentenced Ms Folbigg to terms of imprisonment as follows:

    a. Count 1: manslaughter of Caleb Gibson Folbigg – 10 years' imprisonment with no non-parole period;

    b. Count 2: intentional infliction of grievous bodily harm upon Patrick Allen Folbigg – 14 years' imprisonment with no non-parole period;

    c. Count 3: murder of Patrick Allen Folbigg – 19 years' imprisonment with no non-parole period;

    d. Count 4: murder of Sarah Kathleen Folbigg – 20 years' imprisonment with no non-parole period; and

    e. Count 5: murder of Laura Elizabeth Folbigg – 22 years' imprisonment with a non-parole period of 12 years to commence on 22 April 2012 and expire on 21 April 2033.[431]

314. The sentences were partially accumulated, resulting in an effective head sentence of 40 years' imprisonment with a non-parole period of 30 years to commence on 22 April 2003.[432]

Appeal to the Court of Criminal Appeal

315. Ms Folbigg appealed against her convictions and sentence to the Court of Criminal Appeal. The appeal was heard on 26 November 2004. A judgment was delivered on 17 February 2005 by Sully J, with whom Dunford and Hidden JJ agreed.

316. The appeal against convictions was dismissed. The sentence appeal was upheld, and the Court resentenced Ms Folbigg to a reduced overall term of imprisonment of 30 years with a non-parole period of 25 years.[433]

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425 20 May 2003 T116-118.
426 19 May 2003 T124-128.
427 20 May 2003 T122.
428 20 May 2003 T127-128.
429 20 May 2003 T118-119.
430 20 May 2003 T123-124.
431 *R v Folbigg* [2003] NSWSC 895, [102]-[106].
432 *R v Folbigg* [2003] NSWSC 895, [100].
433 *R v Folbigg* [2005] NSWCCA 23 (Sully J at [191], Dunford and Hidden JJ agreeing at [192]-[193]).

317. The grounds of the conviction appeal were:

a. Ground 1: the trial miscarried as a result of the five charges being heard jointly;

b. Ground 2: the verdicts of guilty were unreasonable and could not be supported having regard to the evidence;

c. Ground 3: the trial miscarried as a result of evidence being led from prosecution experts to the effect that they were unaware of any previous case in medical history where three or more infants in one family died suddenly as a result of disease processes; and

d. Ground 4: the trial judge erred in his directions as to the use the jury could make of coincidence and tendency evidence.[434]

Ground One

318. The Court of Criminal Appeal rejected the argument that there was any error in permitting the Crown to rely upon coincidence and tendency reasoning and the counts being heard by way of a joint trial as a result.[435]

319. Sully J began by considering the applicable test in s 101 of the *Evidence Act*, which had been the subject of consideration during the course of the separate trials application before the trial commenced. Sully J noted that the correct construction and application of s 101(2) of the *Evidence Act*, concerning the requirement that the probative value of coincidence evidence must "substantially outweigh" any prejudicial effect on the defendant, had been the subject of consideration in *R v Ellis* (*"Ellis"*).[436]

320. In that decision the Court determined that the words "substantially outweigh" in a statute could not be construed to have the meaning which the majority in *Pfennig v The Queen* determined for the common law balancing exercise of the competing considerations.[437] Spigelman CJ (Sully, O'Keefe, Hidden and Buddin JJ agreeing) stated that:

> *94. … The "no rational explanation" test may result in a trial judge failing to give adequate consideration to the actual prejudice in the specific case which the hprobative value of the evidence must specifically outweigh.*
> *95. Section 101(2) calls for a balancing exercise which can only be conducted on the facts of each case. It requires the court to make a judgment, rather than to exercise a discretion… The "no rational explanation" test focuses on only one of the two matters to be balanced – by requiring a high test of probative value – thereby averting any balancing process. I am unable to construe s 101(2) to that effect.*
>
> *96. My conclusion in relation to the construction of s 101(2) should not be understood to suggest that the stringency of the approach, culminating in the Pfennig test, is never appropriate when the judgment for which the section calls has to be made. There may well be cases where, on the facts, it would not be open to conclude that the probative value of particular evidence substantially outweighs its prejudicial effect, unless the "no rational explanation" test was satisfied.*[438]

321. Sully J noted further that the statements of principle in that case had been subsequently approved by the High Court in *Ellis v The Queen*.[439]

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434 *R v Folbigg* [2005] NSWCCA 23, [49], [93], [115], [145].
435 *R v Folbigg* [2005] NSWCCA 23, [145]-[160].
436 *R v Folbigg* [2005] NSWCCA 23, [147]; *R v Ellis* (2003) 58 NSWLR 700.
437 *R v Ellis* (2003) 58 NSWLR 700, 94-99, cited in *R v Folbigg* [2005] NSWCCA 23, [147]; *Pfennig v The Queen* (1995) 182 CLR 461.
438 *R v Ellis* (2005) 58 NSWLR 700, 94-96.
439 *R v Folbigg* [2005] NSWCCA 23, [128], citing Transcript of Proceedings, *Ellis v The Queen* [2004] HCA Trans 488.

322. Ms Folbigg submitted that in her case, it would not be open to conclude that the probative value of particular evidence substantially outweighed its prejudicial effect, unless the "no rational explanation" test was satisfied.[440] She submitted that the evidence did not even satisfy the tests in ss 97 and 98 of the *Evidence Act*, because the evidence did not have significant probative value.[441] This was because the five events, on the Crown case, were at their highest undetermined in their origins and, since no event was proven, the evidence had an element of circularity about it.[442]

323. Sully J considered that the four deaths and the ALTE satisfied every relevant part of s 98 of the *Evidence Act*, because the five events were substantially and relevantly similar, and the circumstances in which they occurred were substantially similar.[443] His Honour noted further that:

> *Had any one of the five counts charged been severed and tried separately, there must have been a Crown application to lead as coincidence evidence that the event central to the severed count was not, in truth, an isolated event at all but was, rather, but one in a chain of events that were "related events" in the section 98 sense; that whole chain of events having occurred in such an overall context, of which the diaries were a most cogent feature, as to negate any reasonable possibility of mere, albeit somewhat astonishing, coincidence.*[444]

Ground Two

324. Ground two was rejected on the basis that the medical evidence established that it was:

> *Amply open to the jury, which saw and heard the witnesses, to reject the defence hypothesis that each of the five relevant events could be explained away as having derived from identified natural causes; and so to be satisfied beyond reasonable doubt that the Crown had demonstrated that the five events could not be so explained away.*[445]

325. In concluding that the five incidents had been "anything but extraordinary coincidences unrelated to acts done by the appellant", Sully J referred to the "overwhelming weight of the medical evidence", including concessions by the leading defence expert Professor Byard as to cause of death, and Ms Folbigg's diary entries relied upon by the Crown.[446]

326. His Honour described that the diary entries made "chilling reading" and had "damning" probative value.[447]

327. Ms Folbigg's case on ground two relied significantly on the judgment of the English Court of Appeal in *R v Cannings* ("*Cannings*") which was decided between Ms Folbigg's trial and appeal.[448] In that case, the defendant was the mother of four children, three of whom died in infancy. She was charged with the murder of two children and a third charge of murder did not proceed.[449]

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440 *R v Folbigg* [2005] NSWCCA 23, [149].
441 *R v Folbigg* [2005] NSWCCA 23, [149].
442 *R v Folbigg* [2005] NSWCCA 23, [149].
443 *R v Folbigg* [2005] NSWCCA 23, [151].
444 *R v Folbigg* [2005] NSWCCA 23, [153].
445 *R v Folbigg* [2005] NSWCCA 23, [132].
446 *R v Folbigg* [2005] NSWCCA 23, [130]-[132].
447 *R v Folbigg* [2005] NSWCCA 23, [132].
448 [2004] EWCA Crim 1.
449 *R v Folbigg* [2005] NSWCCA 23, [134], citing the headnote of the *Cannings* case report.

328. Significant fresh evidence was put before the Court on appeal as to the rarity of three natural and unexplained infant deaths in the same family, the interval between the infant's death or near death, and the last time when the infant appeared to be well and the possible significance of an ALTE preceding death. That evidence presented a picture more favourable to the defendant than that which was before the jury.[450]

329. Sully J considered that the English Court of Appeal "sounded warnings" which were as appropriate to Ms Folbigg's case as they were to Ms Cannings' case.[451] The English Court of Appeal set out to "encapsulate different possible approaches" to cases where three infant deaths occur in the same family, where each is apparently unexplained and of which there is no evidence extraneous to the expert evidence that harm was or must have been inflicted.[452]

330. The Court concluded that much depends on the starting point for reasoning. One approach is to examine each death to try to identify any known natural cause and if this cannot be done, the rarity of such incidents in the same family raises a very powerful inference that the deaths must have resulted from deliberate harm. On this approach, the route to guilt is wide open. Almost any other piece of evidence can reasonably be interpreted to fit this conclusion.[453]

331. The alternative approach is to proceed on the basis that if there is nothing to explain the deaths, they remain unexplained and still possible natural deaths. The Court stated that in such a case, it had no doubt that the second approach is correct. The exclusion of currently known natural causes does not establish that the deaths resulted from deliberate infliction of harm.[454]

332. Sully J cited the following passage from *Cannings*:

> *We recognise that the occurrence of three sudden and unexpected infant deaths in the same family is very rare, or very rare indeed, and therefore demands an investigation into their causes. Nevertheless the fact that such deaths have occurred does not identify, let alone prescribe, the deliberate infliction of harm as the cause of death. Throughout the process great care must be taken not to allow the rarity of these sad events, standing on their own, to be subsumed into an assumption or virtual assumption that the dead infants were deliberately killed, or consciously or unconsciously to regard the inability of the defendant to produce some convincing explanation for these deaths as providing a measure of support for the prosecution's case. If on examination of all the evidence every possible known cause has been excluded, the cause remains unknown.*[455]

333. Sully J concluded that *Cannings* was case-specific and had features quite different from Ms Folbigg's case.[456] For example, in the appeal in *Cannings:*

    a. it had been demonstrated, after one of the principal Crown experts gave evidence in another trial, that his evidence in the *Cannings* trial had been seriously flawed. The Court concluded this would likely have impacted on the verdict in *Cannings* if defence counsel had been able to cross-examine the witness and undermine the weight the jury would invariably attach to his evidence;[457]

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450 *R v Folbigg* [2005] NSWCCA 23, [134].
451 *R v Folbigg* [2005] NSWCCA 23, [135].
452 *R v Cannings* [2004] EWCA Crim 1, [10], cited in *R v Folbigg* [2005] NSWCCA 23, [135].
453 *R v Cannings* [2004] EWCA Crim 1, [10]-[13], cited in *R v Folbigg* [2005] NSWCCA 23, [135].
454 *R v Cannings* [2004] EWCA Crim 1, [10]-[13], cited in *R v Folbigg* [2005] NSWCCA 23, [135].
455 *R v Cannings* [2004] EWCA Crim 1, [177], cited in *R v Folbigg* [2005] NSWCCA 23, [136].
456 *R v Folbigg* [2005] NSWCCA 23, [137].
457 *R v Folbigg* [2005] NSWCCA 23, [138].

b. the Court received a substantial body of scientific research evidence that was not before the jury;[458]

c. the Court considered both trial evidence and post-trial fresh evidence about Ms Cannings' family tree, and concluded that there may well be a genetic cause, as yet unidentified, for the deaths of the Cannings' children;[459] and

d. the Court emphasised that "there is no suggestion of ill-temper, inappropriate behaviour, ill treatment let alone violence, at any time, with any one of the four children", and there was also no parallel in *Cannings* for the diaries in Ms Folbigg's case.[460]

334. Referring to the differences between Ms Folbigg's and Ms Cannings' cases, Sully J decided that it did not follow that the reasoning which led to the quashing of the convictions in *Cannings* must lead, as a matter of course, to the quashing of Ms Folbigg's convictions.[461] His Honour considered there was ample evidence at trial to justify the convictions because:

a. none of the events was caused by an identified natural cause;

b. the possibility that each of the events had been caused by an unidentified natural cause was "only in the sense of a debating point possibility". Evidence of Ms Folbigg's temper and ill-treatment, and the diaries, and the striking similarities of the four deaths was "overwhelmingly to the contrary of any reasonable possibility of unidentified natural causes";

c. only the conclusion that somebody had killed the children, by smothering, remained reasonably open; and

d. the evidence pointed to nobody other than Ms Folbigg.[462]

Ground Three

335. In relation to ground three, Sully J rejected the argument which challenged Barr J's ruling permitting the Crown to lead the evidence from experts as to their knowledge of three or more deaths in the one family.[463]

336. Sully J held that the evidence was relevant. The Crown case was circumstantial which raised questions as to whether it was a reasonable possibility that any of the children's deaths had been caused by natural causes, and whether absent a natural cause in any one of four successive infant deaths in a single family, the only inference rationally available was that the deaths had been caused in some unnatural way.[464]

337. Sully J held that the evidence relevant to this ground tended to prove there was no natural cause, that the deaths had been caused in some unnatural way, and that the only rational inference was suffocation.[465]

338. Sully J also considered there was no basis for the trial judge to be apprehensive that the probative value of the evidence was outweighed by the danger of unfair prejudice by reversing the onus of proof, provided that the trial judge made clear to the jury it was the Crown's burden to prove its case. Sully J considered his Honour did so.[466]

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458 *R v Folbigg* [2005] NSWCCA 23, [139], citing *R v Cannings* [2004] EWCA Crim 1, [138].
459 *R v Folbigg* [2005] NSWCCA 23, [140], citing *R v Cannings* [2004] EWCA Crim 1, [31]-[35].
460 *R v Folbigg* [2005] NSWCCA 23, [141], citing *R v Cannings* [2004] EWCA Crim 1, [160].
461 *R v Folbigg* [2005] NSWCCA 23, [142].
462 *R v Folbigg* [2005] NSWCCA 23, [143].
463 *R v Folbigg* [2005] NSWCCA 23, [76].
464 *R v Folbigg* [2005] NSWCCA 23, [78]-[81].
465 *R v Folbigg* [2005] NSWCCA 23, [81].
466 *R v Folbigg* [2005] NSWCCA 23, [83].

339. Sully J concluded that the evidence ought not to have been excluded on the basis that its probative value was outweighed by the danger of unfair prejudice to Ms Folbigg, in that the jury would misuse the evidence in some other way.[467]

340. It was submitted on behalf of Ms Folbigg that:

> *the proposition that a combination of events is entirely without precedent in medical history is not far removed from the expression of the odds of such a combination of events occurring innocently in terms of a statistic.*[468]

341. Sully J rejected this submission on the basis that the relevant evidence from the prosecution experts (Professors Herdson and Berry, and Dr Beal) did no more than establish that reputable and apparently reliable expert opinion could not identify another known case where four infants in one family had died successively from unknown natural causes.[469]

342. Sully J emphasised that it was for the jury to decide whether or not to accept this evidence, and that it was no more than a piece of circumstantial evidence which the Crown argued that, when added to all the other known facts and circumstances concerning the four deaths, there was left open no other reasonable hypothesis than that the four deaths were unnatural.[470]

Ground Four

343. In relation to ground four, Sully J rejected Ms Folbigg's complaints as to the directions to the jury on tendency and coincidence evidence.[471] Having referred to the directions given by the trial judge, Sully J found that:

a. the jury was correctly instructed that in considering each individual count of murder there were effectively three possibilities open on the evidence: identified natural causes, unidentified natural causes, and deliberate suffocation;

b. the jury was correctly instructed that if, in any particular case, it remained open as a reasonable possibility that the death had been caused by some natural cause that could be identified, there must be an acquittal on that count; and

c. the trial judge explained to the jury correctly that if, in any particular case, the jury did not regard it as remaining open as a reasonable possibility that death had been caused by an identified natural cause, then, in considering whether it remained open as a reasonable possibility that the cause of death had been some unidentified natural cause, it was permissible to have regard to the whole of the context of the death. This included, where appropriate, that part of the context was some other death or deaths similarly unexplained but so strikingly similar as to cause the jury to infer that it was not open as a reasonable possibility that the particular death had been caused by some unidentified natural cause.[472]

344. Sully J concluded that while the trial judge's tendency directions did not tell the jury in terms that the legitimate use of tendency evidence, if the jury found tendency in fact, was "to help show what happened was not an accident" or "to help to show what was the intention with which the accused did the act which she is proved to have done", that had nonetheless been made "quite clear" to the jury by the time the summing-up concluded.[473]

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467 *R v Folbigg* [2005] NSWCCA 23, [84]-[85].
468 *R v Folbigg* [2005] NSWCCA 23, [86], citing written submissions on behalf of Ms Folbigg, [110].
469 *R v Folbigg* [2005] NSWCCA 23, [91].
470 *R v Folbigg* [2005] NSWCCA 23, [91].
471 *R v Folbigg* [2005] NSWCCA 23, [93]-[114].
472 *R v Folbigg* [2005] NSWCCA 23, [103].
473 *R v Folbigg* [2005] NSWCCA 23, [111]-[112].

Application for special leave to the High Court

345. Following the Court of Criminal Appeal decision, Ms Folbigg filed an application for special leave in the High Court. That application was heard and refused on 2 September 2005 by McHugh ACJ, Kirby and Heydon JJ.[474]

346. Ms Folbigg raised two grounds:

   a. that tendency and coincidence reasoning was not permissible; and

   b. that it was not properly available to the Crown to lead evidence that three or more infant deaths in the one family from natural causes is without precedent, on the basis that such evidence reverses the onus of proof.[475]

347. On the first ground, Ms Folbigg's counsel referred expressly to ss 97, 98 and 101 of the *Evidence Act*, following which McHugh ACJ asked whether, given the words in s 98(1)(b), "or having regard to other evidence adduced", the difficulty in the argument was that "the diary entries lend very cogent weight to what inferences can be drawn from the unexplained deaths". His Honour queried why, "when the coincidence evidence is read in light of those diary entries, was it not open to a court to think that the evidence was of significant probative value."[476]

348. Kirby J observed "there must be a point at which it has to be left, in our system, to the jury to evaluate these things so long as the jury is properly instructed."[477]

349. McHugh ACJ and Kirby J considered Ms Folbigg's case to be distinguishable from the *Cannings* case.[478] Kirby J noted:

> *It is the combination of the coincidences which are collected by the prosecution submissions and the diary entries which seem to me to be very powerful in combination, in this case, and lift the case above the Cannings case.*[479]

350. In relation to the second ground, Kirby J observed that "the standard directions on onus of proof and burden of proof were given by the trial judge."[480] McHugh ACJ noted the trial judge's statement in the summing-up that:

> *SIDS deaths are rare in the community. There is no authenticated record of three or more such deaths in a single family. This does not mean, of course, that such events are impossible. It is an illustration of the rarity of deaths diagnosed as SIDS.*[481]

474 Transcript of Proceedings, *Folbigg v R* [2005] HCATrans 657.
475 Transcript of Proceedings, *Folbigg v R* [2005] HCATrans 657, 13-17.
476 Transcript of Proceedings, *Folbigg v R* [2005] HCATrans 657, 65-80.
477 Transcript of Proceedings, *Folbigg v R* [2005] HCATrans 657, 130-135.
478 Transcript of Proceedings, *Folbigg v R* [2005] HCATrans 657, 220.
479 Transcript of Proceedings, *Folbigg v R* [2005] HCATrans 657, 230-235.
480 Transcript of Proceedings, *Folbigg v R* [2005] HCATrans 657, 287-289.
481 Transcript of Proceedings, *Folbigg v R* [2005] HCATrans 657, 293-300.

351. In refusing to grant special leave to appeal, McHugh ACJ said:

> *We are not convinced that error has been shown in the conclusion or the reasoning of the Court of Criminal Appeal of New South Wales such that it would warrant the grant of special leave against the applicant in this unusual case. But apart from the coincidence evidence, there was other strong evidence, especially the diary entries made by the applicant, that was available to support the inferences that could be drawn from the tendency or coincidence evidence. In addition, we can detect no relevant misdirection of the jury by the learned trial judge. Nor are we convinced that there has been any miscarriage of justice in this case. Accordingly, special leave to appeal must be refused.*[482]

Further appeal to the Court of Criminal Appeal

352. On 27 November 2007 the Court of Criminal Appeal heard a further appeal against conviction, after granting an application for leave to reopen the appeal against conviction.[483]

353. The grounds of appeal were that the trial miscarried because:

   a. a juror or jurors obtained information from the internet which revealed that Ms Folbigg's father had killed her mother; and

   b. a juror or jurors informed themselves away from the trial as to the length of time an infant's body is likely to remain warm to the touch after death.[484]

354. The appeal was dismissed. McClellan CJ at CL (Simpson and Bell JJ agreeing) was satisfied that the irregularities were not material and did not give rise to a miscarriage of justice.[485] McClellan CJ at CL observed:

> *I have reviewed the whole of the evidence. I am satisfied this was an overwhelming Crown case. I am entirely satisfied that notwithstanding the irregularities, no substantial miscarriage of justice has occurred.*[486]

Ms Folbigg's submissions to the Inquiry – coincidence evidence at trial

355. Ms Folbigg made extensive submissions in the Inquiry concerning the use of coincidence evidence at trial, and as to whether and how it ought to be employed in the Inquiry.[487]

356. Ms Folbigg submitted, in effect, that the use of coincidence evidence at trial should be reconsidered now, and gives rise to a reasonable doubt as to her guilt. In this regard, Ms Folbigg made comprehensive submissions in the Inquiry on the coincidence notice served by the prosecution in advance of the trial, the decision by Wood CJ at CL on the separate trials application, the interlocutory appeal to the Court of Criminal Appeal from his Honour's decision dismissing that application, and the effect of evidence received in the Inquiry upon all of these.

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482 Transcript of Proceedings, *Folbigg v R* [2005] HCATrans 657, 311-322.
483 *Folbigg v R* [2007] NSWCCA 371.
484 *Folbigg v R* [2007] NSWCCA 371, [4].
485 *Folbigg v R* [2007] NSWCCA 371, [60]-[62].
486 *Folbigg v R* [2007] NSWCCA 371, [64].
487 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E.

357. Ms Folbigg also submitted that new and fresh evidence affects whether coincidence reasoning can now legitimately be applied in the discharge of my functions under the *CAR Act.*[488]

General principles – separate trials

358. Ms Folbigg referred to authority highlighting the requirement that coincidence (and tendency) evidence be admissible before trials can be joined.[489] She noted difficulties when evidence considered before trial is significantly different to that presented at trial, and when subsequent fresh evidence is inconsistent with an earlier determination that evidence presented before trial was admissible.[490]

359. Ms Folbigg submitted that evidence of smothering had to be firmly established to the exclusion of a reasonably available natural cause before coincidence evidence could be introduced (i.e., impermissible speculation could not be used to support the Crown case).[491] Further, select diary entries referred to in the separate trials application (without evidence of context) were necessary for admissibility of the coincidence evidence, because "most of the points of coincidence were without striking similarity when considered in the context of a family environment".[492]

360. Ms Folbigg's submissions set out the list of similarities in the Crown's notice, contained in Wood CJ at CL's judgment (see above at [5]). That was a list of 19 matters. The same list was considered in the Court of Criminal Appeal in the appeal against Wood CJ at CL's decision. Significantly, the list contained all 10 matters relied upon by the Crown at the time of the trial and it was that list of 10 matters considered by the Court of Criminal Appeal in the appeal against conviction. The question posed by the submissions is whether the 10 matters are properly to be considered as coincidence and tendency evidence.

Section 101 (substantial probative value) – the threshold test

361. In relation to s 101 of the *Evidence Act*, Ms Folbigg referred to *Ellis* (see above at [317]).[493] The court is to consider actual prejudice, however, *Ellis* does not remove consideration of "no other rational explanation" if it is a factor in the particular case.[494] *Hoch v The Queen* provides guidance in determining whether evidence has significant probative value – striking similarities, unusual features, underlying unity, system or pattern.[495] Ms Folbigg submitted that there was nothing striking about her actions and no unusual patterns – she led a suburban life "with all its travails, the search for more should have led nowhere".[496]

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488 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [1], [104]-[106].
489 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [9].
490 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [9].
491 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [11] n 8.
492 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [12].
493 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [61]-[62], citing Spigelman CJ in *R v Ellis* (2003) 58 NSWLR 700, 74, 83-84.
494 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [63].
495 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [65], citing Simpson J in *Hoch v The Queen* 165 CLR 292, 294-295.
496 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [66].

Findings – coincidence evidence at trial

Principles in a circumstantial case

362. In some inquiries directed under Part 7 of the *CAR Act* it may be appropriate to concentrate on a single aspect of the evidence or trial process in order to report on the results of the inquiry and to form an opinion for the purposes of s 82(1)(a) of the *CAR Act*.[497]

363. In this Inquiry a broader approach was warranted, because the Crown case at trial was wholly circumstantial.

364. In a circumstantial case, a finding of guilt of an accused should not only be a rational conclusion but should be the ***only*** rational conclusion that can be drawn from the circumstances.[498] The accused must be found not guilty if there is an inference consistent with innocence reasonably open on the evidence.[499] Similarly, if there was an inference consistent with innocence reasonably open on the evidence available to me in this Inquiry, this would ground an opinion that there exists a reasonable doubt for the purposes of s 82(1)(a) of the *CAR Act*.

365. To be reasonably open on the evidence, an inference must rest upon something more than mere conjecture. The bare possibility of innocence does not prevent a finding of guilt, if the inference of guilt is the only reasonable inference open upon a consideration of all the facts in evidence.[500] There is a very real distinction between drawing an inference from proven facts and engaging in speculation.[501] Reasonable hypotheses – possessing some degree of acceptability or credibility – consistent with innocence must be excluded.[502]

366. *All* of the circumstances established by the evidence are to be considered and weighed in deciding whether there is an inference consistent with innocence reasonably open on the evidence.[503] This involves focussing not on each individual fact but rather their combined force.[504]

Coincidence rules

367. Coincidence evidence is evidence of a set of circumstances which has probative force (or proof value) due to the degree of improbability or unlikelihood that the circumstances happened coincidentally. If coincidence evidence is admitted in a criminal trial, the jury may take into account the similarities between relevant acts or events in deciding whether they are satisfied that the person who did one act must have done the other/s, employing reasoning that it is highly improbable that the events occurred simply by chance (or coincidence).

368. Because of the risk of prejudice to an accused by this kind of evidence, ss 98 and 101 of the *Evidence Act* prescribe rules for admissibility of coincidence evidence in criminal trials.

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497 *Anderson Inquiry*, p 61.

498 *Shepherd v The Queen* (1990) 170 CLR 573, 579 (Dawson J); *Knight v The Queen* (1992) 175 CLR 495, 502 (Mason CJ, Dawson and Toohey JJ); *The Queen v Baden-Clay* [2016] HCA 35, referring to *Barca v The Queen* (1975) 133 CLR 82 at 104 (Gibbs, Stephen and Mason JJ).

499 *Shepherd v The Queen* (1990) 170 CLR 573, 579 (Dawson J); *Knight v The Queen* (1992) 175 CLR 495, 502 (Mason CJ, Dawson and Toohey JJ).

500 *The Queen v Baden-Clay* [2016] HCA 35, referring to *Barca v The Queen* (1975) 133 CLR 82 at [104] (Gibbs, Stephen and Mason JJ).

501 *Lane v R* (2013) 241 A Crim R 321, [69].

502 *East v Repatriation Commission* (1987) 16 FCR 517, 532, approved in *Bushell v Repatriation Commission* (1992) 175 CLR 408, 414 (Mason CJ, Dean and McHugh JJ); see also *Burke v The Queen* (1997) 96 A Crim R 334, 353 (Walsh J, approving other remarks of Malcolm CJ).

503 *The Queen v Hillier* [2017] HCA 13, [48]-[49] (Gummow, Hayne and Crennan JJ).

504 *Lane v R* (2013) 241 A Crim R 321, [70] (citations omitted).

369. Section 98(1) of the *Evidence Act* provides:

> *(1) Evidence that 2 or more events occurred is not admissible to prove that a person did a particular act or had a particular state of mind on the basis that, having regard to any similarities in the events or the circumstances in which they occurred, or any similarities in both the events and the circumstances in which they occurred, it is improbable that the events occurred coincidentally unless:*
>
> > *(a) the party seeking to adduce the evidence gave reasonable notice in writing to each other party of the party's intention to adduce the evidence, and*
> >
> > *(b) the court thinks that the evidence will, either by itself or having regard to other evidence adduced or to be adduced by the party seeking to adduce the evidence, have significant probative value.*

370. Any similarities in the events or circumstances will inform whether it is improbable that two or more events occurred coincidentally, and whether the evidence is admissible to disprove that the events occurred coincidentally.[505] "Significant" probative value means important or of consequence; the evidence must be influential in its context.[506]

371. Section 101 of the *Evidence Act* prescribes an additional safeguard on the use of coincidence evidence in criminal trials. Such evidence cannot be used unless its probative value substantially outweighs any prejudicial effect it may have on the defendant.

372. A significant aspect of the submissions made on behalf of Ms Folbigg to the Inquiry relates to the decision to have a joint trial and the use of coincidence and tendency evidence.

Use of coincidence evidence at trial

373. The submissions of Ms Folbigg focus on the decision made by Wood CJ at CL and the reasons given by his Honour, and also by the Court of Criminal Appeal in the appeal against his Honour's decision. A point that is properly made is that at that early stage before the trial had commenced, evidence relied upon by the Crown in opposing the separate trial application included statements of evidence from Professor Ouvrier and Dr Ophoven. Neither of those experts gave evidence at the trial itself.

374. As described earlier in this Chapter, the trial proceeded with other experts being called, and after conviction there was a further appeal to the Court of Criminal Appeal. The first ground of appeal was that "[t]he trials of the appellant miscarried as a result of the five charges in the indictment being heard jointly." The conviction appeal was also dismissed and there was then an application for special leave made to the High Court which was heard on 2 September 2005. That application was dismissed.

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505 Jill Anderson, *The New Law of Evidence: Annotation and Commentary on the Uniform Evidence Acts* (LexisNexis Butterworths, 2nd ed, 2009) 386.

506 *IMM v The Queen* (2016) 257 CLR 300, 302, 314 (French CJ, Kiefel, Bell and Keane JJ); ***R v Lockyer*** (1996) 89 A Crim R 457 (Hunt CJ at CL).

375. In dealing with the first ground in the conviction appeal, the Court of Criminal Appeal (Sully J, Dunford and Hidden JJ agreeing) said at [151] of the judgment:

> *It seems to me that the four deaths and Patrick's ALTE satisfy every relevant part of section 98 of the Evidence Act, the section dealing generally with coincidence evidence. The five events were substantially and relevantly similar. The circumstances in which they occurred were, plainly I should have thought, substantially similar. The five events were, therefore, 'related events' in the statutory sense established by section 98.*

376. The Court went on to say at [153]:

> *Had any one of the five counts charged in the present case been severed and tried separately, there must have been a Crown application to lead as coincidence evidence, evidence that the event central to the severed count was not, in truth, an isolated event at all; but was, rather, but one in a chain of events that were "related events" in the section 98 sense; that whole chain of events having occurred in such an overall context, of which the diary entries were a most cogent feature, as to negate any reasonable possibility of more, albeit somewhat astonishing, coincidence.*

377. The Court of Criminal Appeal in that decision was not in any way considering the reports of Professor Ouvrier and Dr Ophoven. The Court was simply considering the evidence at the trial. The evidence at the trial consisted of ten points of coincidence which were given to the jury.[507] These are set out above at [135].

378. I agree with the judgment of the Court of Criminal Appeal in Ms Folbigg's appeal against conviction for the reasons given by the Court. To the extent that Ms Folbigg's submissions challenged the use of coincidence and tendency evidence at trial, on the basis of evidence then available, I do not accept those submissions.

379. The real question that arises in accordance with my task under s 82 of the *CAR Act*, is whether, in light of the fresh and new evidence in the Inquiry, there has been any relevant change to the evidence adduced at the trial that in my opinion gives rise to a reasonable doubt as to Ms Folbigg's guilt. If there were, that could have the effect of causing a reconsideration of the separate trial issue. That issue, including the application of coincidence reasoning to the performance of my functions, is considered in Chapter 9.

Criticisms of Crown prosecutor's conduct

Alleged reversal of onus of proof

Ms Folbigg's submissions

380. Ms Folbigg submitted that in many instances during the course of the trial, the Crown prosecutor reversed the onus of proof. The only example given by Ms Folbigg was the Crown prosecutor's "pigs might fly" submission in his closing address.[508] That submission was as follows:

> *I would like you to briefly consider what I anticipate will be submissions made by my learned friend, Mr Zahra. As I said, I don't know exactly what he is going to say, but I anticipate it a little bit, and I have to anticipate.*

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507 Exhibit AR, Notice of Crown coincidence evidence to disprove mere coincidence; 13 May 2004 T1362.34- 1364.21; 19 May 2003 T41.

508 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part F, [4].

> *I can't disprove that one day some piglets might be born with wings and that they might fly. Is that a reasonable doubt? No. Is the hypothesis that the defence advances a reasonable doubt? No. Why not? Because if you look at what they are suggesting, not in isolation, but in totality.*

381. This portion of the Crown prosecutor's closing address was said to contravene the decision of the Court of Criminal Appeal in *Wood v The Queen*,[509] in which the Court held that the Crown prosecutor's conduct in inviting the jury to consider a list of fifty questions, which the Crown prosecutor informed the jury were the salient questions in order to decide the outcome of the case, was an impermissible course for him to follow as it reversed the onus of proof.

382. Ms Folbigg submitted that the decision in *Wood* constituted a "change or clarification in the law" and that, had her appeal been heard after *Wood*, she would have had an appeal point which would have been likely to have been accepted.[510]

Findings

383. The conduct of the Crown prosecutor in making his "pigs might fly" submission, and in his closing address more generally, does not cause me to form a reasonable doubt as to the guilt of Ms Folbigg due to a procedural irregularity or an error in the trial process.

384. First, I do not consider that the Crown prosecutor reversed the onus of proof in making his "pigs might fly" submission. In my view, the Crown prosecutor was highlighting what was, on the Crown case, the implausibility of a defence case consistent with innocence when regard was had to the totality of the evidence that had been presented to the jury.[511] In other words, he was making the point that, when the evidence was viewed as a whole, the defence submissions were fanciful.

385. Secondly, I consider that, following the trial judge's clear and unambiguous directions, the jury could have been in no doubt that the onus of proving Ms Folbigg's guilt lay squarely on the Crown. [512]

386. In summing-up to the jury, Barr J repeatedly referred to the fact that the onus was on the Crown to prove its case,[513] and that the Crown was required to prove the offences beyond reasonable doubt.[514] Moreover, Barr J specifically directed the jury that the Crown must disprove any reasonable possibility of death by natural causes and that, given the Crown case was a circumstantial one, the Crown must exclude all possible explanations of the evidence which were consistent with the innocence of the accused.[515]

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509 (2012) 84 NSWLR 581.

510 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part F, [6], [10].

511 The Court of Criminal Appeal came to a similar conclusion in ***DL v R*** [2017] NSWCCA 57, [94] (Leeming JA, Rothman and Wilson JJ).

512 In *Ibrahim v R* [2014] NSWCCA 160, [42]-[43], [75]-[76] and [85], Simpson J (Hidden J agreeing) and Hamill J came to a similar conclusion, despite the fact that comments made by the Crown prosecutor in that case were apt to encourage the jury to reason that there was an onus on the accused to fill a "very big hole" in the defence case. The Court found that the clear and unambiguous directions of the trial judge removed any possibility that the jury was left in doubt as to the onus of proof.

513 19 May 2003 T15-16, T44-45, T47, T50-51, T54, T59, T67-71.

514 19 May 2003 T14, T17, T21, T35, T43, T47, T49-50, T67, T69, T77-78, T113, T123-124, T128.

515 19 May 2003 T70, T123-124.

387. Thirdly, in her appeal to the Court of Criminal Appeal in 2004, Ms Folbigg argued that her trial miscarried as a result of the admission of expert evidence that there were no documented cases of three or more infants in the one family who had died of natural causes, which she claimed had the effect of reversing the onus of proof.[516] She also argued that the directions by the trial judge effectively cast an onus on Ms Folbigg to demonstrate an innocent explanation for each of the deaths.[517]

388. The Court concluded that the jury could not sensibly have understood from anything said by Barr J that Ms Folbigg bore any onus of proof upon any of the elements of the crimes charged.[518] Ms Folbigg's application for special leave to appeal to the High Court which again submitted there had been a reversal of the onus was also refused. Kirby J noted that the standard directions on onus and burden of proof were given by the trial judge.[519]

389. Fourthly, I consider there to be no basis for Ms Folbigg's submission that *Wood* constituted a change or clarification in the law. The conclusion in *Wood* that the Crown prosecutor had reversed the onus of proof was based on the 2001 decision of *R v Rugari*,[520] in which the Court of Criminal Appeal had held that rhetorical questions put to the jury by the Crown prosecutor reversed the onus of proof.[521] That case was decided two years before Ms Folbigg's trial.

390. The Crown prosecutor's conduct in making the "pigs might fly" submission bears no similarity to the conduct considered in *Wood* or in *Rugari.* Although the Crown prosecutor asked rhetorically whether the defence hypothesis raised a reasonable doubt, he answered that question by reference to the expert evidence that there was no prior record of a case of that nature and asked the jury to look at all of the evidence, rather than at the medical evidence in isolation.

391. I am satisfied that the conduct of the Crown prosecutor in making his "pigs might fly" submission did not constitute a procedural irregularity or an error in the trial process and did not invite the jury to reverse the onus of proof.

Matters of fact

392. The Crown was criticised by Ms Folbigg for suggesting that SIDS is a diagnosis and referring to children "dying of SIDS".[522]

393. I do not find that the reference at trial to children "dying of SIDS" could have misled or confused the jury. Two forensic pathologists and a paediatrician at trial adopted this terminology,[523] and the Crown qualified the phrase in their opening address, stating that

> *SIDS… is a mysterious illness or combination of illnesses which causes the sudden and unexpected death of an otherwise healthy infant during sleep… The cause of SIDS and mechanism of SIDS is unknown.*[524]

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516 *R v Folbigg* [2005] NSWCCA 23, [49], [71].
517 *R v Folbigg* [2005] NSWCCA 23, [107].
518 *R v Folbigg* [2003] NSWCCA 23, [113].
519 Transcript of Proceedings, *Folbigg v The Queen* [2005] HCA Trans 657, 289-290.
520 (2001) 122 A Crim R 1.
521 The "fundamental submission" in *Wood* was that the Crown prosecutor had committed the error discussed in *R v Rugari* (2001) 122 A Crim R 1: *Wood v The Queen* (2012) 84 NSWLR 581, 609. The Court in *Wood* cited *Rugari* at 609 and 610 and in various paragraphs that were omitted from the reported version of the case. See *Wood v R* [2012] NSWCCA 21, [580], [631] and the extract from *Livermore v The Queen* (2006) 67 NSWLR 659 set out at [632].
522 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [51], [53].
523 7 May 2003 T1210.13 (Professor Byard); 28 April 2003 T981.14 (Dr Beal); 15 April 2003 T710.15 (Dr Cala).
524 1 April 2003 T30.

Alleged misleading of the jury

394. Ms Folbigg submitted that the Crown drifted between referring to three or more deaths due to SIDS and three or more deaths due to unknown causes, potentially causing confusion.[525] I note that the Crown asked Professor Berry and Dr Beal in both forms and referred to both forms in its closing address. By referring to both alternatives in the Crown closing address, I do not find that the Crown applied the definition laxly. Further, I do not believe that a reasonably-minded jury would have been confused by the two concepts as evidence was elicited from two witnesses as to the incidence of each event.

395. The Crown was also criticised for its tenth point of coincidence that Ms Folbigg failed to render assistance to the children upon finding them.[526] Ms Folbigg submitted that she rendered assistance by calling an ambulance, calling Mr Folbigg and by providing CPR to Laura.[527]

396. Whilst I accept this submission, I find the commonality in failing to pick up Caleb, Patrick (including following his ALTE) and Sarah from their beds in an attempt to rouse them to be a relevant point of coincidence.

Alleged failure to provide proper context

397. Ms Folbigg criticised the Crown for failing to provide the full context of the diary entries relied upon at trial, leading to interpretations which were absurd, neglected the distinction between omission/commission, and demonstrated a predetermination of the interpretation to be placed on them.[528]

398. I have considered the interpretation of Ms Folbigg's diaries at length in Chapter 8. On the basis of the discussion in that Chapter, I am satisfied that it was reasonably open to the jury to interpret Ms Folbigg's diary entries as they did.

525 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [54].
526 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [18(x)].
527 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [19(g)].
528 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [21], [145], [156], [175].

Report of the Inquiry into the convictions of Kathleen Megan Folbigg

July 2019

Volume 1: Chapter 4

Chapter 4: Sudden Infant Death Syndrome and Recurrence

Introduction

1. In the first part of this chapter I consider the advances in our understanding of Sudden Infant Death Syndrome, or SIDS, since Ms Folbigg's trial.

2. I then move on to consider the evidence as to the incidence of reported deaths of three or more infants in the same family attributed to unidentified natural causes. The doubt or question that gave rise to this Inquiry was in relation to such evidence.

Sudden Infant Death Syndrome

At trial

3. At the time of the trial in 2003, a widely accepted definition of SIDS was that published in 1991 by the National Institute of Child Health and Human Development Group in the United States.[1] That definition referred to SIDS as:

> *the sudden death of an infant under one year of age which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history.*[2]

4. Professor Hilton was the Head of the Institute of Forensic Medicine at Glebe for 12 years and at the time of trial was in the process of retiring.[3] He gave a working definition of SIDS at trial: "the unexpected death of any infant or a young child for which a thorough autopsy fails to reveal a cause of death or pathology sufficient to cause death."[4] Professor Hilton gave evidence that the vast majority of SIDS deaths were under 12 months, with occasional deaths of older infants.[5] He identified that SIDS was much more likely to occur in infants aged between two and four months, was much less common in infants over six months and was extremely uncommon in an infant over one year old.[6] He also said that a thorough death scene investigation was necessary.[7]

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1 Exhibit D, Roger W Byard, 'Sudden Infant Death Syndrome: Definitions' in Jhodie R Duncan and Roger W Byard, *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 1, 4, n 37.

2 Exhibit D, Roger W Byard, 'Sudden Infant Death Syndrome: Definitions' in Jhodie R Duncan and Roger W Byard, *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 1, 4, n 37.

3 14 April 2003 T615.21-46.

4 14 April 2003 T654.54-57.

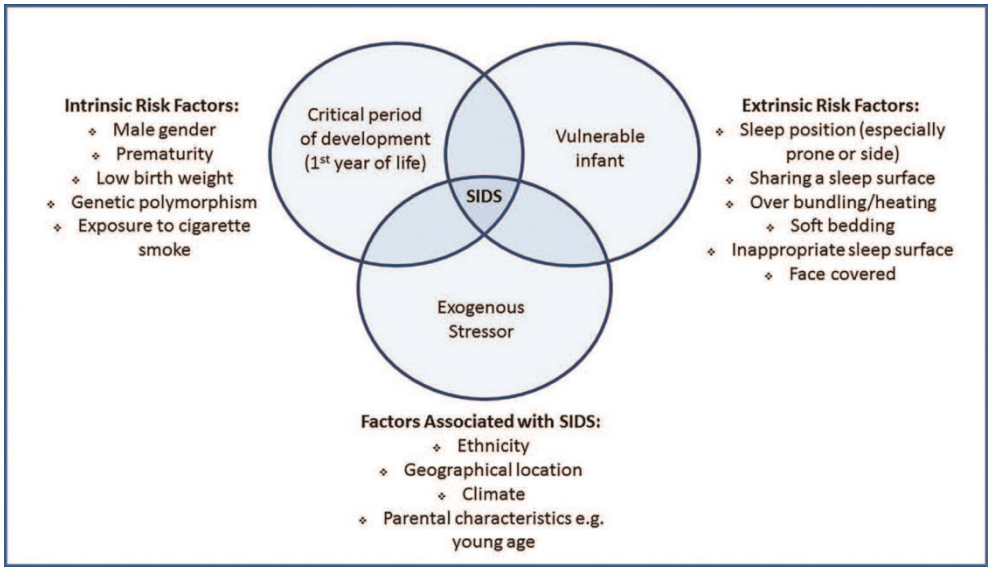
5 14 April 2003 T655.1-16.

6 14 April 2003 T655.1-16.

7 14 April 2003 T655.1-16.

5. Dr Beal, who had particular expertise in SIDS research, described SIDS as the death of an infant or young child who was apparently completely well at the time of death, where the death scene examination and the autopsy show nothing which could be responsible for the death.[8]

6. In 1994 Filiano and Kinney developed the "triple risk model" which reflected the belief that SIDS is multifactorial in origin.[9] This model proposes that when a vulnerable infant, such as one born preterm or exposed to maternal smoking, is at a critical but unstable developmental period in homeostatic control,[10] and is exposed to an exogenous stressor such as being placed to sleep prone, then SIDS may occur.[11] It further proposes that infants will die of SIDS only if they possess all three factors, and that the vulnerability lies dormant until they enter the critical developmental period and are exposed to an exogenous stressor.[12]



8 5 May 2003 T1134.50-58.

9 Transcript of the Inquiry, 18 March 2019 T25.42-45, T26.8, T31.30-31; Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 1, citing J J Filiano and H Kinney, 'A Perspective on Neuropathologic Findings in Victims of the Sudden Infant Death Syndrome: The Triple Risk Model' (1994) 65 *Biology of the Neonate* 194.

10 Homeostatic control is "the maintenance of a relatively stable internal environment by an organism in the face of a changing external environment and varying internal activity using negative feedback mechanisms to minimise an error signal": Harold Modell et al, 'A Physiologist's View of Homeostasis' (2015) 39(4) *Advances in Physiology Education* 259, 264.

11 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 1.

12 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 1.

7. The main risk factors at the time the model was developed, and in 2003, were:
    a. prematurity;
    b. low birth weight;
    c. exposure to maternal smoke;
    d. sleeping on one's stomach;
    e. shared sleeping platforms;
    f. being over bundled or overheated;
    g. soft bedding;
    h. covered faces;
    i. age of mother; and
    j. socio-economic circumstances.[13]

8. The factors in the model are risks, not causes.[14]

Advances and risk factors

Advances generally

9. In the years since Ms Folbigg's trial, SIDS has remained a diagnosis of exclusion based on an absence of any other cause and is understood as being multifactorial.[15]

10. A key advance has been the introduction of sub-categories of SIDS in 2004, along with new definitions and criteria for each sub-category. This change necessitates considering all the deaths and the ALTE, Caleb and Sarah's deaths in particular, in light of this development. The 2004 definition and sub-categorisation are discussed further below from [27].

11. Relevant evidence on the contemporary understanding of SIDS was received in the Inquiry through Duncan and Byard (2018). This publication is a multidisciplinary volume which covers a wide range of aspects of sudden infant and early childhood death, including changes in definitions, epidemiology and risk factors.

12. Evidence about advances was also given by Professors Horne and Elder in the Inquiry. Broadly, evidence they gave was consistent with relevant articles in Duncan and Byard (2018) that contemporary research has advanced in terms of the sophistication of understanding or beliefs of how intrinsic, extrinsic and additional risk factors may be identified, operate and interact.[16]

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13 Exhibit D, Jhodie R Duncan and Roger W Byard, 'Sudden Infant Death Syndrome: An Overview' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 15, 18-27.

14 Transcript of the Inquiry, 18 March 2019 T26.42-27.1, T52.16-27.

15 Transcript of the Inquiry, 18 March 2019 T 23.45-46, T26.8, T47.12-13, T53.4; Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 1.

16 Exhibit D, Jhodie R Duncan and Roger W Byard, 'Sudden Infant Death Syndrome: An Overview' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 15, 19-27; Exhibit J, Report of Professor Rosemary Horne (10 February 2019) pp 1, 3-6; Exhibit K, Report of Professor Dawn Elder (15 February 2019) [20]; Transcript of the Inquiry, 18 March 2019 T26.8-14.

Risk factors and the triple risk model

13. Professor Horne stated that SIDS occurs during sleep, with 90 per cent of deaths occurring in the first six months of life with a peak incidence between two to four months and fewer babies dying in the first month.[17] She explained that this is a time when sleep patterns and cardiorespiratory control are rapidly maturing.[18] The final pathway to SIDS is widely believed to involve immature cardiorespiratory control in conjunction with a failure of arousal from sleep.[19]

14. Professor Horne further identified numerous physiological studies showing that the major risk factors for SIDS (prone sleeping, maternal smoking, prematurity, head covering) have significant effects on blood pressure and heart rate and their control and impairment of arousal from sleep. It is observed that these are largely extrinsic risk factors. In addition, Professor Horne stated that babies who subsequently died from SIDS had incomplete arousal responses and altered autonomic cardiovascular control.[20]

15. Professor Horne described how the Back to Sleep campaign in the 1990s reduced the incidence of SIDS by more than 85 per cent, but said the incidence since about 2006 seems to have plateaued.[21] Figures produced by the Australian Institute of Health and Welfare show that in 2017 there were 87 deaths, or a rate of 0.3 deaths per 1,000, which were classified as Sudden Unexpected Death in Infants ("SUDI").[22] In New Zealand, figures show that in the year 2000 it was 1-1.5 per 1,000 live births, and in 2015 it was 0.7.[23]

16. Professor Elder said that the majority of infant deaths now occur when the infant is in an unsafe sleep position, with the highest risk where this is combined with maternal smoking.[24] Other risks are reflected in the triple risk model: male gender, low birth weight, ethnicity, geographical location, climate, genetic polymorphism and parental characteristics.

17. In this regard, Professor Duflou also referred to the "so-called" triple risk model as "a central theory of SIDS" which hypothesises that a SIDS event requires the simultaneous occurrence of three factors:

    a. infant sleeping during a critical stage of development;

    b. underlying abnormality (e.g. abnormal medullary (brainstem) serotonergic system); and

    c. presence of exogenous homeostatic stressor, including for example, soft mattress, face down, parental smoking, minor respiratory tract infection.[25]

18. Dr Cala also stated the risk factors for SIDS are believed to be premature birth, maternal smoking, bed-sharing, and prone sleeping position.[26]

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17 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 1, nn 4-8.

18 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 1, n 9.

19 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 1, n 10.

20 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 1, nn 11-12.

21 Transcript of the Inquiry, 18 March 2019 T21.18-29, T29.28.

22 Transcript of the Inquiry, 18 March 2019 T29.8-16. SUDI is a useful term that refers to all sudden and unexpected infant deaths and not just to SIDS. "SUDEP" refers specifically to sudden unexpected death in epilepsy. Exhibit D, Roger W Byard, 'Sudden Infant Death Syndrome: Definitions' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018)p 7, 52.

23 Transcript of the Inquiry, 18 March 2019 T29.45-48.

24 Transcript of the Inquiry, 18 March 2019 T43.32-49, T51.12-13.

25 Exhibit L, Report of Professor Johan Duflou (13 February 2019) pp 47-48, citing Kim A Collins and Roger W Byard (eds), *Forensic Pathology of Infancy and Childhood* (Springer, 2014).

26 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 2.

19. Professor Horne gave evidence that environmental cigarette smoke and paternal smoking increases the risk slightly to about 1.1.[27] Formula feeding increases the risk to 1.5.[28] Professor Elder opined that the most profound effect is exposure to maternal smoking in utero, which seems more related to alteration of the infant's arousal responses.[29]

Other advances

20. In her report, Professor Horne identified further advances in the understanding of SIDS. One is that research suggests that autonomic failure plays a role in the fatal event. Arousal from sleep has long been considered a vital survival response for restoring homeostasis in response to various life-threatening situations, such as prolonged hypoxia or hypotension.[30] Any impairment of protective responses may render an infant vulnerable to respiratory and cardiovascular instabilities common during infancy, and postulated to occur in SIDS.

21. A second advance is that characteristic findings prior to the SIDS event include short lasting but profound bradycardia (slowing of the heart).[31] Infants who had been studied previously and who subsequently died from SIDS had variabilities in heart rates or autonomic control or balance of the heart. Others had fewer spontaneous arousals from sleep and impaired arousal responses.

22. Professor Cordner told the Inquiry that the genetic aspect has blossomed since 2003.[32] Professor Horne also stated that in recent years there have been significant advances in identifying genetics and neuropathology which are associated with SIDS, and explained in some detail a number of those advances in her report.

23. According to Professor Horne, advances have established an increasing number of inherited or sporadic gene mutations, which can cause either sudden unexpected death (e.g. cardiac arrhythmias) or polymorphisms, which likely contribute to death but which require additional factors.[33]

24. Professor Horne also stated that:

> [t]*here is convincing evidence that a slight infection and an activated immune system are involved in SIDS. Almost half of SIDS infants had a mild respiratory infection in the last days prior to death. Genetic variations in cytokine genes are most likely involved.*[34]

25. Genetic studies indicate that up to 35 per cent of SIDS cases might be explained by familial or genetic diseases, such as cardiomyopathies, ion channelopathies or metabolic disorders that remained undetected during conventional forensic autopsy procedures.[35] Overall, however, the underlying cause of the majority of SIDS cases still remains elusive and is likely due to a multifactorial aetiology, triggered by a combination of different genetic and environmental risk factors.[36]

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27 Transcript of the Inquiry, 18 March 2019 T28.17-18, citing R Carpenter et al, 'Bed Sharing when Parents Do Not Smoke: Is there a Risk of SIDS? An Individual Level Analysis of Five Major Case-Controlled Studies' (2013) 3 *British Medical Journal Open* e002299:1-11; 18 March 2019 T44.46-45. This means that environmental smoking and paternal smoking increases the odds of SIDS by a factor of 1.1, holding other risk factors constant.

28 Transcript of the Inquiry, 18 March 2019 T31.23-25.

29 Transcript of the Inquiry, 18 March 2019 T45.23-36.

30 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 3, nn 35-36.

31 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 3, nn 37-43.

32 Transcript of the Inquiry, 19 March 2019 T94.30-31.

33 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 3.

34 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 4, nn 54-55.

35 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 4, n 56.

36 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 4.

26. Professor Elder, a clinical practitioner, gave a different perspective on SIDS in contemporary practice. She said that genetic variation is not a topic applicable to most of the deaths that she sees. Rather, her practical experience is that they are usually children who have been in unsafe sleep circumstances.[37]

2004 definition and sub-categorisation

Application of 2004 definition

27. In 2004 Krous et al published the current definition of SIDS, and also introduced sub-categorisation.[38] The current general definition describes SIDS as:

> *the sudden unexpected death of an infant < 1 year of age, with onset of the fatal episode apparently occurring during sleep, that remains unexplained after a thorough investigation, including performance of a complete autopsy and review of the circumstances of death and the clinical history.*[39]

28. As can be seen, the 2004 definition includes that SIDS occurs during sleep and adds the necessity of a thorough death scene investigation.[40] Professor Horne explained in the Inquiry that the sub-categories are mainly a subdivision for research purposes and to encompass cases where information is lacking for a firm diagnosis.[41]

29. Professor Cordner stated that despite wide recognition, the categorisation or classification of SIDS has not been so widely implemented.[42] Similarly, Dr Cala described the categories as being for academic purposes, and not to improve understanding of "this mysterious condition".[43] He did not believe this is helpful and it "may just be confusing".[44]

30. Professor Duflou also relied on the definition and noted that it is used by NSW Health.[45]

31. Professor Cordner cited a book by Byard (2010) to state that our understanding is still incomplete.[46] He stated that epidemiological factors include:

    a. young age (95 per cent less than six months, peak two to four months);
    b. males > females (approx 55:45);
    c. prematurity (not shown in all studies);
    d. maybe, history of poor prenatal care;
    e. most often, in cot after sleeping;
    f. often high in birth order;

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37 Transcript of the Inquiry, 18 March 2019 T48.46-49.3.

38 Henry F Krous et al, 'Sudden Infant Death Syndrome and Unclassified Sudden Infant Deaths: A Definitional and Diagnostic Approach' (2004) 114(1) *Pediatrics* 234.

39 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 1, n 1.

40 Transcript of the Inquiry, 18 March 2019 T20.49-21.9, T21.48-22.22.

41 Transcript of the Inquiry, 18 March 2019 T21.3-9, T22.7- 23.48, T24.20-29, T24.49-50, T25.10.

42 Exhibit Q, Report of Professor Stephen Cordner (undated) p 27.

43 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 2.

44 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 2.

45 Exhibit Q, Report of Professor Stephen Cordner (undated) p 27; Exhibit L, Report of Professor Johan Duflou (13 February 2019) pp 38-39.

46 Exhibit Q, Report of Professor Stephen Cordner (undated) p 29, citing Roger W Byard, *Sudden Death in the Young* (Cambridge University Press, 2010) 560.

g. a history of minor respiratory or gastrointestinal illness in the days leading up to death, although "this is no longer a consistent finding";

h. that twins have two to fourfold increased risk attributed to low birth weight, prematurity and exposure to similar environmental factors;

i. prone sleeping position;

j. cigarette smoke exposure;

k. poor weight gain;

l. bed-sharing;

m. tendency for younger mothers, lower socio-economic status; and

n. generally more common colder/southern climates.[47]

32. Dr Cala referred to the NSW Child Death Review Team definition in his report, which is consistent with the 2004 definition:

> *The sudden and unexpected death of an infant less than one year of age, with the onset of the lethal episode apparently occurring during sleep; that remains unexplained after a thorough investigation including performance of a complete autopsy, review of the circumstances of death and the clinical history.*[48]

33. Dr Cala stated that there must be no convincing cause of death found after a thorough autopsy.[49] SIDS deaths are typically two to four months, with 90 per cent within six months of age.[50] Further, younger than two months is unusual, and Dr Cala considered that younger than one month of age or older than 12 months would not be SIDS.[51]

34. Dr Cala highlighted that the term SIDS "is purely a descriptive one that lacks both symptoms and physical signs. It is a 'label' with no actual medical meaning", and is an "invented term" which was "an attempt to provide parents/caregivers with 'an explanation' for the death."[52]

Subcategorisation

35. The descriptions of the sub-categories as set out in Duncan and Byard (2018) are set out below.[53]

47 Exhibit Q, Report of Professor Stephen Cordner (undated) p 30.

48 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 2, citing NSW Child Death Review Team, *Annual Report 2014* (NSW Ombudsman, October 2015) 59.

49 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 2.

50 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 6, citing Vincent J M DiMaio and Dominick D J DiMaio, *Forensic Pathology* (CRC Press, 2nd ed, 2001) 349.

51 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 6.

52 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 2

53 Exhibit D, Roger W Byard, 'Sudden Infant Death Syndrome: Definitions' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 1, 5-6.

Category 1A SIDS

36. Category 1A SIDS is described as follows:

> *An infant death that meets the requirements of the general definition with all of the following:*
>
> Clinical: *Older than 21 days and under 9 months; a normal clinical history, including term pregnancy (≥37 weeks gestational age); normal growth and development; no similar deaths in siblings, close genetic relatives (uncles, aunts and first-degree cousins), or other infants in the custody of the same caregiver.*
>
> Circumstances: *Investigation of the various scenes where incidents leading to death may have occurred, and determination that they do not provide an explanation for death found in a safe sleeping environment with no evidence of accidental death.*
>
> Autopsy: *Absence of potentially lethal pathological findings; minor respiratory system inflammatory infiltrates are acceptable; intrathoracic petechial hemorrhages are a supportive but not an obligatory or diagnostic finding; no evidence of unexplained trauma, abuse, neglect, or unintentional injury; no evidence of substantial thymic stress effect (i.e. thymic weight less than 15 g, and/or moderate to severe cortical lymphocyte depletion). Occasional "starry sky" macrophages or minor cortical depletion are acceptable; toxicology, microbiology, radiology studies, vitreous chemistry and metabolic screening studies are negative.*

37. As can be seen, Category 1A SIDS invokes classic features of SIDS with a complete investigation, a child who was older than 21 days and under nine months, and no similar deaths in the family or with the same caregiver.[54] It is the most definite case of SIDS – "the perfect SIDS case".[55]

38. Professor Horne said that if an infant is not in a safe sleeping environment, the death is usually defined as accidental suffocation or unascertained, and not Category 1A SIDS.[56] However, the definition does permit findings of minor respiratory system inflammatory infiltrates.[57] In addition, it permits the occasional "starry sky" macrophage (inflammatory cells seen on the thymus under a microscope) unless this is obviously from pronounced thymic stress.[58]

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54 Transcript of the Inquiry, 18 March 2019 T22.11-35.

55 Transcript of the Inquiry, 19 March 2019 T74.12-14.

56 Transcript of the Inquiry, 18 March 2019 T23.17-22.

57 Exhibit D, Roger W Byard, 'Sudden Infant Death Syndrome: Definitions' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 1, 6.

58 Transcript of the Inquiry, 19 March 2019 T82.19-49.

Category 1B SIDS

39. Category 1B SIDS is described as follows:

> *An infant death that meets the requirements of the general definition and also meets all of the above criteria for Category IA except that: investigation of the various scenes where incidents leading to death may have occurred was not performed, and/or one or more of the following analyses was not performed: toxicology, microbiology, radiology, vitreous chemistry, and metabolic screening.*

40. Category 1B SIDS comprises classic features of SIDS but with an incomplete investigation.[59] Professor Horne said that a death of this type may also be able to be described as undetermined or unascertained.[60]

Category 2 SIDS

41. Category 2 SIDS is described as follows:

> *An infant death that meets Category I criteria except for one or more of the following:*
>
> Clinical: *Age range — outside Category IA or IB, i.e. 0 to 21 days or 270 to 365 days; similar deaths of siblings, close relatives, or other infants in the custody of the same caregiver that are not considered suspicious for infanticide or for recognized genetic disorders; neonatal and perinatal conditions (e.g. those resulting from preterm birth) that have resolved by the time of death.*
>
> Circumstances of death: *Mechanical asphyxia or suffocation by overlaying not determined with certainty.*
>
> Autopsy: *Abnormal growth and development not thought to have contributed to death; marked inflammatory changes or abnormalities not sufficient to be unequivocal causes of death.*

42. As such, in Category 2 SIDS the infant may be of an age outside the classic SIDS range, that is, younger than 21 days or between nine months and 12 months. Similar deaths may have occurred, provided they are not suspicious for infanticide or for recognised genetic disorders. Mechanical asphyxia or suffocation by overlaying may not be determined with certainty. Marked inflammatory changes may be present upon autopsy but not sufficient to be an unequivocal cause of death.

Application of the SIDS sub-categories

43. Evidence received in the Inquiry indicates that there is some difference between how definitions and sub-categorisation of SIDS are viewed for research purposes and how they are applied in forensic pathology practice and as between forensic pathologists.

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[59] Transcript of the Inquiry, 18 March 2019 T23.24-27.

[60] Transcript of the Inquiry, 18 March 2019 T23.36-40.

44. Professor Horne, for instance, described SIDS as excluding ***all*** possible causes whether environmental or involving some intrinsic factor.[61] However, Professor Cordner stated that SIDS does not exclude an unnatural cause and that there is always a possibility of an unnatural or natural explanation that is not uncovered. He stated that however people use the term SIDS, smothering is not excluded on forensic pathology grounds when SIDS is the diagnosis.[62]

45. Professor Duflou considered that unless he found "good evidence" for overlaying or a form of mechanical asphyxia, which removed it from SIDS, he would place such a death in Category 2 SIDS.[63] He would not describe such a death as "undetermined", whereas Dr Cala and Professor Hilton would.[64]

46. Professor Cordner considered that Category 2 SIDS would not exclude an unnatural death and described it as a "weaker" category – more flexible and enabling inclusion.[65] Dr Cala said SIDS could be understood to be a death with unidentified natural causes, but not suspected unnatural causes.[66]

47. It was also impressed in evidence by the forensic pathologists in the Inquiry that it is necessary to consider the whole picture of the death under investigation.[67] Most of the time, a forensic pathologist is given a relevant history to do with the death, such as symptoms the person may have experienced.[68] This is reflected in the SIDS definitions, and the requirement to look at clinical features, circumstances and the autopsy.[69] Dr Cala said that it is important to take into consideration the circumstances leading to death, and both Dr Cala and Professor Duflou would take family history into consideration.[70] Dr Cala would, in a case where there are no signs, look to the clinical matters and circumstances consistent with the SIDS definitions.[71]

48. This may reflect the practical reality of uncertainties which attend practice, and an appreciation that an autopsy is sometimes quite a "blunt tool", as Dr Cala described in the Inquiry and as Professor Duflou indicated in his written and oral evidence.[72]

49. At the time of the trial, the terms "undetermined" or "unascertained" were more frequently used by forensic pathologists than appears to be the case now. The expanded subcategories accommodate some deaths which may previously have been ascribed as "undetermined", "unascertained" or similar. Evidence was available from a number of experts at the time of the trial as to when the term "undetermined" may be ascribed as a cause of death rather than SIDS.

50. For example, in her evidence at the trial Dr Beal said that a death of a child outside the age range of one to six months would probably be undetermined.[73]

51. Dr Cala gave evidence at the trial that a death with an undetermined cause could include natural, inflicted and also accidental causes.[74]

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61 Transcript of the Inquiry, 18 March 2019 T23.45-48.
62 Transcript of the Inquiry, 18 March 2019 T23.45-48.
63 Transcript of the Inquiry, 19 March 2019 T113.14-21.
64 Transcript of the Inquiry, 19 March 2019 T113.39-40, T114.10-28.
65 Transcript of the Inquiry, 19 March 2019 T130.24-132.28.
66 Transcript of the Inquiry, 19 March 2019 T76.35-38.
67 Transcript of the Inquiry, 19 March 2019 T78.31-79.1.
68 Transcript of the Inquiry, 19 March 2019 T86.17-25.
69 Transcript of the Inquiry, 19 March 2019 T86.27-31.
70 Transcript of the Inquiry, 19 March 2019 T86.4-31; 21 March 2019 T251.7-11.
71 Transcript of the Inquiry, 19 March 2019 T116.1-6.
72 Transcript of the Inquiry, 19 March 2019 T86.7-9 (Dr Cala), T84.30-85.44 (Professor Duflou); Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 51.
73 5 May 2003 T1135.19-23.
74 15 April 2003 T721.15-20, T721.40-48.

52. In the Inquiry, Dr Cala said "undetermined" encompasses unidentified natural causes and unnatural causes.[75] Professor Duflou's view was that "undetermined" may be employed where there is worry about trauma, describing as an example a circumstance where a child has a fractured rib: not causative of death, but worrying.[76]

Submissions of counsel assisting

53. Counsel assisting submitted that the evidence at trial and in the Inquiry was that prone sleeping in an unsafe sleeping environment was and remains the single greatest risk for SIDS.[77]

54. Counsel assisting submitted that I should therefore accept the evidence of Professors Horne and Elder that all of the Folbigg children were at low risk of SIDS because they were found supine, none of them had their head covered or were on an unsafe sleeping surface, at no relevant stage did Ms Folbigg smoke and Mr Folbigg smoked outside the house.[78]

Submissions of Ms Folbigg

55. Ms Folbigg submitted that the expert opinions at trial about typical factors in "SIDS cases" were flawed because they:

    a. failed to draw a distinction between SIDS and the sudden death of an infant[79]; and

    b. treated SIDS as a diagnosis or a cause of death in and of itself.[80]

56. For example, in closing the Crown prosecutor said:

> *SIDS means: We don't have any suspicious circumstances. We don't have any doubts about this case, but we cannot find a cause of death. So we write it down as SIDS.*[81]

Ms Folbigg submitted this was likely to mislead and confuse because the definition of SIDS at the time of trial and now included unnatural causes of death.[82]

57. Ms Folbigg next submitted that there was

> *no epidemiological information at the Inquiry as to the incidence of death of children under two years from infection, myocarditis, neurological disorder or genetic disorders and any effect such epidemiology would had* [sic] *on the various SIDS research material*.[83]

58. Ms Folbigg also submitted that there is a difference between not being able to identify any cause of death, and having two or more potential causes of death and not being able to determine which was the cause.[84] The terms "unexplained" and "undetermined" can be used in both cases depending on the forensic pathologist.[85] Nor should it be assumed such terms are directly interchangeable with "SIDS".[86]

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75 Transcript of the Inquiry, 19 March 2019 T76.45-48.
76 Transcript of the Inquiry, 19 March 2019 T77.2-9.
77 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 5, [104].
78 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 5, [103].
79 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [12].
80 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [39].
81 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [49]; 13 May 2003 T1310.42-1311.6.
82 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [50].
83 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [42].
84 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [43].
85 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [44]-[46].
86 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [46].

Findings

59. I accept Ms Folbigg's submission that SIDS is not a diagnosis or a condition itself.[87] This is made clear by the definition of SIDS as a "sudden unexpected death of an infant < 1 year of age… that remains unexplained".[88]

60. However, risk factors for SIDS are simply factors that have been identified by experts as making an infant more susceptible to a sudden and unexpected death during sleep, with no obvious cause. References to these risk factors do not imply that SIDS itself is a cause of death.[89]

61. For the reasons set out in Chapter 5, I accept the evidence of Professors Horne and Elder that all of the Folbigg children were at low risk of SIDS.[90]

Recurrence

Introduction

62. The doubt or question that gave rise to this Inquiry was in relation to evidence adduced at Ms Folbigg's trial as to the incidence of reported deaths of three or more infants in the same family attributed to unidentified natural causes. That evidence was given by a number of medical experts who said they had never experienced or read about three or more such instances.

63. That evidence gave rise to the submission to the jury by the Crown prosecutor that "it has never been recorded that the same person has been hit by lightning four times"[91] and "I can't disprove that one day some piglets might be born with wings and that they might fly. Is that a reasonable doubt?"[92]

64. The trial judge dealt with those submissions by directing the jury as follows:

> *SIDS deaths are rare in the community. There is no authenticated record of three or more such deaths in a single family. This does not mean, of course, that such events are impossible. It is an illustration of the rarity of deaths diagnosed as SIDS.*[93]

65. Set out below is a summary of the evidence given at trial, followed by the Inquiry's investigations into this issue.

Trial

Awareness of three or more deaths in a single family

66. Dr Beal gave evidence before the jury that as far as she was aware, there had never been three or more deaths from SIDS in the one family, either from her experience or literature.[94] Asked whether she had ever come across a family in which there had been three or more children who had died suddenly from natural causes in the way that the Folbigg children had died, she said "no".[95]

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87 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [37]-[42].

88 Exhibit D, Roger W Byard, 'Sudden Infant Death Syndrome: Definitions' in Jodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 1, 5.

89 Transcript of the Inquiry, 18 March 2019 T26.42-27.1.

90 Transcript of the Inquiry, 18 March 2019 T34.45, T36.10, T38.21, T40.40-43 (Professor Horne), T35.34-35, TT37.1-2, T38.25, T40.28-29 (Professor Elder).

91 13 May 2003 T1364.43-44.

92 13 May 2003 T1375.27-30.

93 19 May 2003 T24-25.

94 5 May 2003 T1136.50-56; Exhibit H, Forensic pathology tender bundle, Statement of Dr Susan Beal (8 December 1999) p 5.

95 5 May 2003 T1143.52-1144.2

67. Professor Herdson gave evidence in the trial that from his experience and the literature, he was not aware of three or more, thoroughly investigated, infant deaths from SIDS in one family.[96]

68. Professor Berry gave evidence before the jury that he was not aware of any case, from his experience or literature, where three or more children in one family had suffered sudden death from no obvious injury or disease, from a combination or variety of diseases.[97] In his first report, he noted that nonetheless, it was important to explore this possibility.[98] Except for some reports many years prior which did not withstand scrutiny, Professor Berry was unaware in contemporary literature, or from his practice or research, of any families with three or more deaths from SIDS.[99] Nor was he aware of any three or more kindred children, previously fit, who had died suddenly due to another medical condition.[100]

69. Professor Byard had never heard of a case in which three or more children in one family had died or had an ALTE suddenly, unexpectedly, during a sleep period at home.[101] He had never heard of a case in which four children in one family had died suddenly and unexpectedly from four different natural causes.[102]The rare cardiac and metabolic conditions that may account for this had been excluded.[103]

70. In a report prepared at the request of the defence, Professor Busuttil stated that it was extremely unusual and quite unprecedented to have four deaths of siblings in the same family over a period of eight years – he had never seen or heard of this occurrence in over 30 years of practice in pathology.[104]

Key expert evidence of recurrence of SIDS/unexplained deaths in the trial

71. Dr David Cooper was formerly the Head Paediatrician at the Paediatric Respiratory and Sleep Service at John Hunter Hospital and performed sleep studies on Patrick and Sarah. He was concerned with the clinical evaluation of infants and children suffering apparent sleep and breathing disorders.[105] At the time of the trial he was the Director of Paediatric Respiratory and Sleep Medicine at the Mater Children's Hospital in Brisbane, and Associate Professor at the University of Queensland.[106]

96 1 May 2003 T1049.51-56, T1080.5-33, T1081.27-1082.21; Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Herdson (17 January 2002) p 3.

97 1 May 2003 T1066.36-1067.11; Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 25.

98 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 25.

99 1 May 2003 T1066.36-39. See also Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 26.

100 1 May 2003 T1066.53-58.

101 7 May 2003 T1222.42-46.

102 7 May 2003 T1253.3-13.

103 7 May 2003 T1223.14-23.

104 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 5.

105 Exhibit H, Forensic pathology tender bundle, Statement of Dr David Cooper (6 December 1999) pp 1-2; 14 April 2003 T585.48-586.18.

106 14 April 2003 T585.34-37.

72. Dr Cooper gave evidence in the trial that a familial or inherited link in SIDS was extremely improbable.[107] Compared with the 1970s, by 2003 an increased risk of recurrence of SIDS in a family could not absolutely be excluded but the likelihood of recurrence was thought to be probably no higher than the general population.[108] There was debate about whether it was a little higher or no higher at all (putting aside multiple pregnancies – twins, triplets, etc – with combined increased risk factors of prematurity and multiple pregnancy).[109] In relation to the likelihood of a second SIDS death in a family, as analyses became more critical and better studies were done, he thought that having one SIDS death did not predispose the family to another.[110] Whereas 10 years prior the literature would have described the risk as "several-fold", by 2003 the recurrence incidence was thought to be only very slightly increased or not increased at all.[111]

73. Dr Cooper gave evidence that the causes of SIDS were undoubtedly multifactorial.[112] He said that environmental factors can raise the incidence of SIDS, and referred in this context to smoking, socio-economic status, colder climates, drug use during pregnancy and maternal age.[113] He said that SIDS was much more likely to occur in a child between two to four months of age, was much less common after six months and was extremely uncommon after one year.[114]

74. In his evidence, Dr Cooper outlined SIDS research from the 1970s by Dr Alfred Steinschneider in the United States who promulgated the notion, firstly, that SIDS was often familial, secondly that one might be able to investigate and predict it in families, and thirdly that these children should have a home apnoea monitor.[115] As a result, doctors were widely encouraged to provide apnoea monitors to parents of children considered at risk of SIDS or ALTEs.[116] The term "ALTE" evolved from what people initially called near-miss SIDS.[117]

75. Although apnoea monitors were widely encouraged, over time the medical community developed the view that they were well-meaning but did not save lives.[118] No link was shown between apnoea and death from SIDS (other than in premature babies), nor between ALTEs and death from SIDS.[119]

76. The Back to Sleep campaign, introduced in Australia in the early 1990s, saw a dramatic effect on the prevalence of death from SIDS around the world – it was more than halved.[120] Notwithstanding the lack of identified link, the number of so-called ALTEs also went down.[121] From the success of the Back to Sleep campaign, Dr Cooper concluded that "if you can reduce something by more than 50 per cent by doing a simple social intervention, it can't be anything to do with breeding."[122]

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107 14 April 2003 T611.39-42, T612.6-51, T614.45-47, T615.1-4.
108 14 April 2003 T590.31-40, T591.4-592.18, T608.8-49, T608.52-57, T610.47-611.2, T614.40-47.
109 14 April 2003 T608.8-49.
110 14 April 2003 T610.47-611.2, T614.40-47.
111 14 April 2003 T610.56-58.
112 14 April 2003 T614.51-57.
113 14 April 2003 T593.54-594.43.
114 14 April 2003 T595.26-34.
115 14 April 2003 T590.22-40.
116 14 April 2003 T590.57-591.5.
117 14 April 2003 T590.45-46.
118 14 April 2003 T591.4-592.18.
119 14 April 2003 T592.53-593.1.
120 14 April 2003 T593.3-24.
121 14 April 2003 T593.25-26.
122 14 April 2003 T614.45-47.

Inquiry

Investigations conducted

77. The Inquiry conducted a number of investigations into this issue. First, it commissioned reports from Professors Horne and Elder, each of whom has expertise (Professor Horne in research, Professor Elder in both research and clinical practice) in SIDS. The Professors gave oral evidence in the Inquiry, addressing contemporary medical and research knowledge of the rarity or otherwise of SIDS and recurrence of SIDS in the community.

78. Secondly, the Inquiry conducted its own research, and invited parties and relevant experts who gave evidence before the Inquiry, to identify literature – both before and after 2003 – referencing instances of recurrence of SIDS or other sudden unexplained infant deaths. In this regard, the Inquiry received considerable assistance from those representing Ms Folbigg, who provided an extensive collection of literature relevant to this issue and other issues.

79. Thirdly, the Inquiry received reports from forensic pathologists Professors Cordner, Hilton and Duflou, and Dr Cala, all of whom also gave oral evidence in the Inquiry, which included their collective and contemporary experience and expertise on the incidence and recurrence of sudden unexplained infant deaths.

Evidence

Awareness of three or more deaths in a single family

80. Dr Cala gave evidence in the Inquiry that he has not received a case of three deaths in the one family since 2004.[123] In describing SIDS risk factors, Dr Cala noted that some doctors do not believe that SIDS "runs in families", and some question whether diagnosis of SIDS can occur twice or more in a family – there has been intense disagreement for many decades.[124]

81. Professor Hilton said that he had not directly been involved in any cases with a subsequent death since 2004 (noting that Professor Hilton is retired but remained a consultant in forensic medicine until 2016).[125] Professor Duflou recalled two cases before 2004 in which he had found two sudden infant deaths in a family.[126]

82. Professor Horne stated that (for the purposes of her engagement by the Inquiry) she had asked a number of colleagues in the USA and none could recall more than three deaths in a family which were attributed to SIDS.[127] She identified literature reporting instances of multiple deaths from SIDS in a single family.[128] Literature on this topic reviewed by the Inquiry is summarised below.

Recurrence of SIDS/unexplained deaths in the Inquiry

83. In the Inquiry, Professor Horne gave evidence that SIDS itself is rare, and instances of recurrence are very rare.[129] In her report, she referred to evidence of the risk of a subsequent SIDS being greater given that the same risk and genetic factors occur in the same family. She noted that it has been estimated that the risk of SIDS is between five to 10 times greater for infants where a sibling has already been a SIDS victim (referring to Hill, 2004, below at [150]).[130] Other literature referred to by Professor Horne is included in the summary of literature below.

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123 Transcript of the Inquiry, 19 March 2019 T76.17-24.

124 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 3.

125 Transcript of the Inquiry, 19 March 2019 T77.11-15; T62.7-21.

126 Transcript of the Inquiry, 19 March 2019 T75.47-50.

127 Professors Fern Hauck, Rachel Moon, Carrie Shapiro-Mendoza, and Carl Hunt and Betty McEntire for the American SIDS Institute. See Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 6.

128 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) pp 6-7, nn 96-100.

129 Transcript of the Inquiry, 18 March 2019 T32.27-33.40.

130 Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 6, n 95.

84. Professor Horne noted that there is no relationship between having an ALTE and dying of SIDS. Although ALTEs were originally thought to be precursor and were called near-miss SIDS, we now know the aetiologies of both conditions are very different.[131]

85. Professor Elder also gave evidence that the risk of recurrence is affected by genetic and environmental factors, mainly a prone sleeping position, bed sharing or maternal smoking.[132]

86. Duncan and Byard (2018) describe the risk of recurrence as "small".[133] Taken to this in the Inquiry, Professor Duflou and Dr Cala took no issue with the description of "small", agreeing that Professor Byard is extremely well-regarded – if not the foremost expert – in the field.[134] However, Dr Cala said that he would not call a third death in a family SIDS. In a second death, he would look very carefully at autopsy for any missing genetic, metabolic or other abnormality and also exclude suspicion of foul play, before giving SIDS as a cause of death.[135]

87. Professor Duflou considered that the chances of a second or third SIDS in a family is unchanged with each sibling and a second death in a family may be SIDS Category 2 if there are no circumstances of concern.[136] Professor Hilton, however, agreed that there appears to be some slight risk for a subsequent SIDS death in a family.[137]

88. Professor Duflou gave oral evidence that while he tries to stay clear of medical statistics, he thought that the chances of a first, second or third SIDS death in a family remained unchanged with each sibling.[138] He described this as a great advance in understanding.[139] In his opinion the controversy in respect of this issue, between Professors Carpenter and Bacon, addressed below, was almost peripheral to that advance.[140] He observed that there is a lot of difficulty relating to definitions of what is meant by "natural" and "non-natural".[141] He agreed that Professor Byard is extremely well-regarded, if not foremost in the field.[142]

89. Professor Cordner suggested in his report that Caleb, Sarah and Patrick's deaths ultimately be considered together.[143] He referred in this context to the issue of multiple SIDS in one family and to controversy attached to this subject.[144] This controversy arose in literature discussed below from [157]; in short, it turned on competing conclusions as to whether repeated unexpected infant deaths are most probably natural (per Carpenter et al, 2007) or whether this cannot confidently be ascertained.[145] Professor Cordner stated in this regard that:

> [t]*he overall point is that as far as the research literature is concerned, more than half the subsequent deaths in families who have sustained a SIDS death are natural deaths and the remaining one third are largely unexplained, not necessarily homicides. Where is the evidence to "think dirty" in families with multiple sudden unexpected and unexplained deaths in infancy?*[146]

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131 Transcript of the Inquiry, 18 March 2019 T36.14-17.

132 Transcript of the Inquiry, 18 March 2019 T33.44-34.20.

133 Exhibit D, Jhodie R Duncan and Roger W Byard, 'Sudden Infant Death Syndrome: An Overview' in Jhodie R Duncan and Roger W Byard (eds), SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future (University of Adelaide Press, 2018) 15, 27.

134 Transcript of the Inquiry, 19 March 2019 T98.48-99.25.

135 Transcript of the Inquiry, 19 March 2019 T76.21-33.

136 Transcript of the Inquiry, 19 March 2019 T75.24-50.

137 Transcript of the Inquiry, 19 March 2019 T99.35-44.

138 Transcript of the Inquiry, 19 March 2019 T98.7-29.

139 Transcript of the Inquiry, 19 March 2019 T98.27-28.

140 Transcript of the Inquiry, 19 March 2019T98.30-36. See below at [153]-[201].

141 Transcript of the Inquiry, 19 March 2019 T98.36-39.

142 Transcript of the Inquiry, 19 March 2019 T98.49-99.2.

143 Exhibit Q, Report of Professor Stephen Cordner (undated) p 83.

144 Transcript of the Inquiry, 19 March 2019 T90.48-91.7, citing R G Carpenter et al, 'Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?' (2005) 365 *Lancet* 29 and C J Bacon, 'Repeat Sudden Unexpected Infant Deaths' (2005) 365 *Lancet* 1137.

145 See Exhibit Q, Report of Professor Stephen Cordner (undated) p 33.

146 Referring to "thinking dirty", a phrase criticised by the Goudge Inquiry in Canada: Exhibit Q, Report of Professor Stephen Cordner (undated) p 34.

90. Professor Cordner accepted the risk of recurrence described as rare or small.[147] While he observed that the literature referred to by Duncan and Byard (2018) is not recent, he also noted that Professor Byard is Australia's expert in the overlap between forensic and paediatric pathology and that he was not in a position to argue with Professor Byard's conclusion.[148]

Evidence of recurrence of SIDS/unexplained deaths in literature prior to 2003

Peterson, Chinn and Fisher (1980) – abstract only

91. The earliest publication on recurrence reviewed by the Inquiry was dated 1980. It reported on data of 1,263 families in which a (at the time, so-called) "cot death" had occurred. Of the 1,194 families without twins or triplets, 18 had lost a child born subsequently. The abstract of the article does not identify any instance of three or more deaths. The abstract indicates that based on the data collected, the risk of repetition was 2 per cent.[149]

Irgens, Skjaerven and Peterson (1984) – abstract only

92. In 1984 it was reported from research conducted in Norway that the recurrence risk in previous publications were overestimates. This research drew upon data on file in the Medical Birth Registry of Norway: 1,062 (1.3/1,000) infants died of SIDS, five were a second case in a family. The article reported that the recurrence risk for a second sibling after a SIDS death was 5.6/1,000 and for all subsequent siblings, 4.8/1,000 – "which would seem encouragingly low from a counselling point of view".[150] The abstract does not identify any instance of three or more deaths.

93. A later Norwegian study in 1996 by Øyen, Skjaerven and Irgens, found that the SIDS rate for second babies was nearly six times higher if the first baby had died of SIDS.[151] As the autopsy rate was poor, diagnoses other than SIDS might have been missed and the rate inflated.[152]

Peterson, Sabotta and Daling (1986) – abstract only

94. The study reported in this article was conducted in Washington from 1969-1984. It yielded results similar to the Norway study, also finding that SIDS rates among siblings were substantially lower than prior estimates. The rate of SIDS in subsequent infants did not differ significantly from the rate among control infants. The article found that it appeared that earlier estimates of the risk of SIDS in siblings were inflated. The abstract does not identify any instance of three or more deaths.[153]

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147 Transcript of the Inquiry, 21 March 2019 T296.35-39.

148 Transcript of the Inquiry, 19 March 2019 T94.8-11.

149 Donald R Peterson, Nina M Chinn and Lloyd D Fisher, 'The Sudden Infant Death Syndrome: Repetitions in Families' (1980) 97 *Journal of Pediatrics* 265.

150 Lorentz M Irgens, Rolv Skjaerven and Donald R Peterson, 'Prospective Assessment of Recurrence Risk in Sudden Infant Death Syndrome Siblings' (1984) 104(3) *Journal of Pediatrics* 349, 349.

151 Nina Øyen, Rolv Skjaerven and Lorentz M Irgens, 'Population-Based Recurrence Risk of Sudden Infant Death Syndrome Compared with Other Infant and Fetal Deaths' (1996) 144(3) *American Journal of Epidemiology* 300, 303.

152 C J Bacon et al, 'How Common is Repeat Sudden Infant Death Syndrome?' (2008) 93 *Archives of Disease in Childhood* 323, 325.

153 Donald R Peterson, Eugene E Sabotta and Janet R Daling, 'Infant Mortality Among Subsequent Siblings of Infants who Died of Sudden Infant Death Syndrome' (1986) 108(6) *Journal of Pediatrics* 911.

Emery (1986)

95. Professor John Emery, from the Department of Paediatrics, University of Sheffield reported on a survey of 12 families with two or more cot deaths. In two families, the deaths were completely unexplained; in three, the babies probably had a familial developmental disorder; in two, the care of the infants was seriously at fault; in five, filicide was probable.[154]

96. Professor Emery outlined the circumstances of the reported families. The first family was socially very deprived. The children (two months, three months) had each had upper acute respiratory infections, albeit insufficient to explain death. The second family were most likely filicide. In the third family, each death was at three months, and both were completely unexplained. In the fourth, two children died; post-mortem findings indicated an unrecognised multiple organ dysplasia syndrome.

97. The fifth family, in which three children died (two at one month, the third aged 12 days) seemed to have some form of pulmonary dysplasia. In the sixth, two children each died at two months, examinations revealing Werdnig Hoffman disease.

98. In the seventh family, in which both deaths were at seven months, filicide was possible. The eighth family involved four babies who died, with two different fathers. Professor Emery described the circumstances as follows:

> *The first death occurred at 3 months and was ascribed to gastroenteritis. The second death occurred 2 years later, again at 3 months, as a cot death at home. I found a respiratory tract infection, mild achondroplasia, and mild hydrocephalus. These were insufficient to be the final cause of death, which was registered as a cot death. 2 years later the third baby also died unexpectedly at home at 2 months; he had a mild respiratory tract infection and was registered as a cot death. This third child had been born after an antepartum haemorrhage, and had been taken home against advice at 13 days. During his 2 months of life he had been admitted to hospital twice, once for gastroenteritis and once with burns. The fourth child died 5 years later at the age of 1 year 9 months. The mother had left a bath half filled with water into which she had put bleach, intending to scour it later. She went into the garden and on her return she found the child in the bath drowned. Necropsy revealed multiple old and new abrasions including recent fresh bruising of the scalp, but no fractures. There was severe cerebral oedema but no dysplasias. The respiratory tract showed severe catarrhal changes compatible with the bleach story… I ascribed the death to drowning… This family shows most of the problems encountered in the diagnosis of cot death – 2 of the children had non-lethal dysplasias; the variety of presentation is a little against a diagnosis of an unrecognised metabolic disturbance; and the whole picture makes filicide a likely differential diagnosis.*[155]

99. Professor Emery was unable adequately to investigate this family.[156]

100. In the ninth family, the two deaths were ascribed as cot death, with a "definite question mark" about how they died.[157] In the tenth, both children died at six months and were terminally ill but died completely unexpectedly. The two deaths in the eleventh family were likely filicide. In the final, twelfth family, the first child presented as cot death at the age of three months. The home circumstances were "appalling" and standard of healthcare very low. The next baby died at two weeks – he was born prematurely, the issued monitor was not used, the heart showed small areas of lymphocyte infiltration insufficient to account for death.

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154 John L Emery, 'Families in which Two or More Cot Deaths have Occurred' (1986) 327 *Lancet* 313, 313.
155 John L Emery, 'Families in which Two or More Cot Deaths have Occurred' (1986) 327 *Lancet* 313, 313-314.
156 John L Emery, 'Families in which Two or More Cot Deaths have Occurred' (1986) 327 *Lancet* 313, 315.
157 John L Emery, 'Families in which Two or More Cot Deaths have Occurred' (1986) 327 *Lancet* 313, 314.

101. Professor Emery observed that:

> *Although these 12 families are too small a group for statistical generalisation, my experience of similar deaths in other parts of this country and abroad suggests a similar range of causes elsewhere.*[158]

102. Professor Emery relied on work earlier in the 1980s to state that the risk of cot death in an ordinary baby was about one in 500, and the risk of a second cot death probably about thrice as much. He said there were probably less than 50 repeat cot deaths a year in Britain. It should be kept in mind that Professor Emery's paper preceded the Back to Sleep campaign and consequent drastic reduction in SIDS deaths.

Diamond (1986)

103. In this article the author reported on a single case of five consecutive siblings whose deaths were ascribed to SIDS.[159] The circumstances of the family and the deaths bear examination.

104. The mother had a premature girl when aged 18. The baby had multiple episodes of apnoea in the nursery and initially required ventilator support. She was discharged when she was one month old, fed poorly and had multiple episodes of upper respiratory infection. She was found apnoeic and cyanotic (blue) in her crib when she was five months old.

105. The second baby was born about three years later from a different father. Her early life was uneventful, but the mother found her dead in her bed aged four months. The third infant was born full term, 18 months later. Her mother found her apnoeic and cyanotic in her crib when she was six weeks old.

106. The mother remarried and two years later the fourth baby was born healthy. At age 13 months, he was found dead in his crib after a midday nap. Two years later again, the fifth baby was born with normal findings on physical examination. A sleep study at 10 days had normal results. At age nine months, he began to have episodes of bradycardia three or four times a night, but sleep studies again were normal. He was kept on theophylline for four months until he was 13 months old. He then had home monitoring for a further six months. There were no episodes of apnoea or bradycardia during that time. He was in good health until, at age two years, he developed a mild afebrile upper respiratory infection and was started on erythromycin. He died during a day nap at home. A six month investigation found no evidence of deliberate causes.

107. The author suggested that:

> [i]*t is possible that this subgroup of repeated SIDS deaths in siblings may be etiologically distinct from the general population of SIDS deaths in infancy.*[160]

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158 John L Emery, 'Families in which Two or More Cot Deaths have Occurred' (1986) 327 *Lancet* 313, 314.

159 Eugene F Diamond, 'Sudden Infant Death in Five Consecutive Siblings' (1986) 170 *Illinois Medical Journal* 33.

160 Eugene F Diamond, 'Sudden Infant Death in Five Consecutive Siblings' (1986) 170 *Illinois Medical Journal* 33, 34. This paper was addressed in Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 6, n 96.

Oren, Kelly and Shannon (1987)

108. The authors of this publication reported on 73 infants born to families who had two or more previous siblings who had either died of SIDS or who were monitored because they had apnoea of infancy – an ALTE – or for abnormal results on polysomnogram and/or pneumogram recording.[161] The infants were selected from a population of 1,341 infants who had been monitored at home under supervision in Massachusetts between 1973 and 1986.[162]

109. Thirteen infants had subsequent severe episodes of apnoea. Five other infants died during a subsequent episode; all deaths occurred in families who had two or more SIDS victims.[163]

110. The authors relied upon other data to state that it indicated that the risk of SIDS is increased by 3.6 to tenfold in subsequent siblings of SIDS victims, with the pathophysiology leading to repeated deaths unknown and the role of genetic or environmental factors being debated.[164]

111. The study grouped infants according to their family history.[165] Group One identified 28 infants who were born to families who had two or more siblings (or half-siblings) who died of SIDS based on history and a review of the written autopsy results. Group Two (17 infants) were born to families in which one infant died of SIDS and a second had apnoea of infancy. Group Three (19) had a previous SIDS death and a second infant with an abnormal pneumogram or polygraph findings.

112. Of the 73 infants, 37 had abnormal results on pneumogram and/or polysomnogram recordings during evaluation. Eighteen infants had an adverse outcome; 13 of these had subsequent severe episodes of apnoea and/or bradycardia.

113. Five other infants died during a subsequent episode. The diagnosis of SIDS was confirmed by autopsy in three of these, and was suggested by a review of the circumstances of the other two.[166] All the deaths occurred in Group One; that is, the only deaths in the study occurred in families with two or more previous infants who died of SIDS.[167]

114. Thus, the authors stated that there was a distinct difference in mortality rate between infants who were siblings of two or more previous SIDS victims and the remaining 45 infants.[168] The death rate for siblings of two or more SIDS victims in the study (17.9 per cent) was higher than as reported by Peterson, Chin and Fisher (1.9 per cent) and Irgens, Skjaerven and Peterson (0.56 per cent) for siblings of one SIDS victim.[169]

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161 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355.

162 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 355.

163 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 355.

164 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 355.

165 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 356.

166 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 356.

167 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 357.

168 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 357.

169 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 357, citing D R Peterson, N M Chinn and L D Fisher, 'The Sudden Infant Death Syndrome: Repetitions in Families' (1980) 97 *Journal of Pediatrics* 265 and Lorentz M Irgens, Rolv Skjaerven and Donald R Peterson, 'Prospective Assessment of Recurrence Risk in Sudden Infant Death Syndrome Siblings' (1984) 104(3) *Journal of Pediatrics* 349.

115. The authors concluded that it was apparent that:

> *There is a tendency for familial aggregation of SIDS victims. Whether this is due to genetic or environmental factors, or is prenatal or postnatal in origin, is unknown. Current evidence does not support a genetic etiology for SIDS. It is conceivable that most SIDS events are sporadic and that the majority of siblings have the same risk as the general population, but that a relatively small number of families with a high incidence of SIDS alters the overall average risk.*[170]

116. No significant associations were found with social and environmental factors, although mothers of multiple SIDS victims tended to be younger and there may have been indication of disruptive social situations in families with clustering of SIDS cases.[171]

117. The authors referred to a report by Rosen et al (1983) which was based on a family with three previous SIDS victims and which suggested that some cases of SIDS were parentally induced.[172]

118. The Oren study included two families with four SIDS victims and two families with three SIDS victims. The authors were unable to identify any evidence of child abuse in those families.[173]

119. The authors concluded that the risk of adverse outcome is significantly greater for an infant born into a family with two or more previous infants with SIDS and/or ALTEs than for infants in families with only one symptomatic infant and a second infant with abnormal tests.[174] They suggested that the increased risk of death among infants in families with two or more previous infants who died of SIDS might be the result of a defect in physiology or in the parental response to the event.[175]

Beal and Blundell (1988)

120. Dr Beal (who gave evidence in Ms Folbigg's trial) and Dr H K Blundell in 1988 reported the results of a study they conducted in South Australia between 1970 and 1986.[176]

121. Of the families studied, 603 had their first infant die from SIDS. Of these, there were eight subsequent deaths in the same age range (7-364 days), or an incidence in the subsequent children estimated as a rate of 6.7-8.3/1,000 (2.5-4.4 times the rate expected, which was 1.9/1,000).[177] This was similar to the relative risk recorded in Norway, which had a reliable data collection system.[178]

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170 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 357.

171 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 357.

172 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 357, citing Carol Lynn Rosen et al, 'Two Siblings with Recurrent Cardiorespiratory Arrest: Munchausen Syndrome by Proxy or Child Abuse?' (1983) 71(5) *Pediatrics* 715.

173 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 357.

174 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 357.

175 Joseph Oren, Dorothy H Kelly and Daniel C Shannon, 'Familial Occurrence of Sudden Infant Death Syndrome and Apnea of Infancy' (1987) 80(3) *Pediatrics* 355, 357.

176 S M Beal and H K Blundell, 'Recurrence Incidence of Sudden Infant Death Syndrome' (1988) 63 *Archives of Disease in Childhood* 924.

177 S M Beal and H K Blundell, 'Recurrence Incidence of Sudden Infant Death Syndrome' (1988) 63 *Archives of Disease in Childhood* 924, 929.

178 S M Beal and H K Blundell, 'Recurrence Incidence of Sudden Infant Death Syndrome' (1988) 63 *Archives of Disease in Childhood* 924, 929. Relative risk is the ratio of probability of an event in an exposed group to the probability of an event in a non-exposed group: Miquel Porta (ed), *Dictionary of Epidemiology* (6th ed, 2014) 'Relative risk'.

122. The authors observed that both genetic and environmental factors seemed to contribute to recurrence.[179] They noted that 626 (92 per cent) of the 677 families identified as having had a SIDS death between January 1970 and September 1986, had their first infant death from SIDS between two weeks and 12 months, were not severely socially deprived, had no family history of sudden unexpected unexplained deaths, and no bronchomalacia at necropsy.[180] Five of these families had a subsequent infant die from SIDS (i.e. an incidence 1.6 times that expected).[181] They went on to say that "[i]t seems reasonable to reassure these families that it is unlikely to happen again."[182]

123. The authors concluded that the risk for a few families (less than 10 per cent of SIDS) is significantly increased. Of the study groups, recurrent sibling deaths were only found in three out of four families with a history of sudden unexpected deaths in children or young adults, four of 35 families in whom the infant who died from SIDS was over 12 months of age at the time of death, two of the 14 families who were felt to be severely socially deprived, and the only family with bronchomalacia found at necropsy. The authors suggested that in families with those factors (and particularly with history of miscarriage or bleeding during pregnancy), the subsequent child should be considered "at risk".

124. It was therefore noted by Beal and Blundell that:

> *For most families (92%) in which an infant died from SIDS the risk of recurrence is small (less than twice the expected risk). We have identified a small subgroup (8%) with a significantly increased risk of recurrence.*[183]

125. In a 2008 article, Professor Bacon and colleagues considered that the rate of recurrence reported by Beal and Blundell may have been exaggerated because they included babies up to two years of age (instead of 12 months) and also autopsies were absent in some cases.[184]

Beal (1989)

126. In 1989 Dr Beal published again in relation to the incidence of SIDS in twins, reporting that it is higher than among singleton infants.[185] Between 1970 and 1988 in South Australia, 33 twin infants were found unexpectedly dead while their co-twin was alive – of these, 31 died of SIDS.[186]

Meadow's letter (1989)

127. In January 1989 Professor Sir Roy Meadow wrote to the editors of *Archives of Disease in Childhood* in relation to Dr Beal's (1988) article. Referring to their observation that both genetic and environmental factors seem to contribute to recurrence, Professor Meadow stated that

> *It is a pity that the authors do not confront the issue more squarely and acknowledge that some of these deaths will have been caused directly by the mothers – that is, filicide or homicide. In order to understand the epidemiology of SIDS better, and also to prevent deaths, it is important to recognise that a small proportion of children labelled as "SIDS" are killed by their parents, usually their mother.*[187]

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179 S M Beal and H K Blundell, 'Recurrence Incidence of Sudden Infant Death Syndrome' (1988) 63 *Archives of Disease in Childhood* 924, 929.
180 S M Beal and H K Blundell, 'Recurrence Incidence of Sudden Infant Death Syndrome' (1988) 63 *Archives of Disease in Childhood* 924, 930.
181 S M Beal and H K Blundell, 'Recurrence Incidence of Sudden Infant Death Syndrome' (1988) 63 *Archives of Disease in Childhood* 924, 930.
182 S M Beal and H K Blundell, 'Recurrence Incidence of Sudden Infant Death Syndrome' (1988) 63 *Archives of Disease in Childhood* 924, 930.
183 S M Beal and H K Blundell, 'Recurrence Incidence of Sudden Infant Death Syndrome' (1988) 63 *Archives of Disease in Childhood* 924, 924.
184 C J Bacon et al, 'How Common is Repeat Sudden Infant Death Syndrome?' (2008) 93 *Archives of Disease in Childhood* 323, 324.
185 Susan Beal, 'Sudden Infant Death Syndrome in Twins' (1989) 84(6) *Pediatrics* 1038, 1041.
186 Susan Beal, 'Sudden Infant Death Syndrome in Twins' (1989) 84(6) *Pediatrics* 1038, 1043.
187 R Meadow, 'Recurrent Cot Death and Suffocation' (1989) 64 *Archives of Disease in Childhood* 179, 179.

128. Professor Meadow referred to Emery's (1985) report of 12 families and his suggestion that between two per cent and 10 per cent of cot deaths are filicide.[188]

129. Professor Meadow referred to his own study of 21 families in which the mother had suffocated a young child, stating that it had "become apparent how often that child has originally been labelled as SIDS and contributed to false national statistics",[189] and that within these families, a recurrence of unexplained or definitely homicidal deaths in other siblings was high. He also noted that the age of children previously labelled SIDS was more variable, and higher, than is usual for SIDS.

Guntheroth, Lohmann and Spiers (1990) – abstract only

130. The authors of this article studied 251,124 live births from Oregon from a 10 year period. They found five recurrences of SIDS among 385 siblings (13/1,000 live births). Families with infant deaths from causes other than SIDS had similar recurrence rates – this suggested that the phenomenon was not specific to SIDS. The overall mortality rate for subsequent siblings after a sudden death event was 20.8/1,000.[190] They found that the risk of SIDS for next and subsequent siblings was four to five times that for the population.[191]

Wolkind et al (1993)

131. This report co-authored by Wolkind, Taylor, Waite, Dalton and Emery, published in 1993, was a study of families which had experienced two or more unexpected infant deaths in England and Wales.[192] It drew from the Foundation for the Study of Infant Deaths ("FSID") programme launched in 1980.

132. Fifty-seven deaths were studied. Twenty-four families had experienced two deaths and three had experienced three deaths. Of the total deaths, 11 (19 per cent) were explained, seven (12 per cent) were probably accidental, 31 (55 per cent) were probably filicide, five (nine per cent) were considered to be true idiopathic SIDS, and in three (five per cent) there was insufficient information to draw a conclusion.

133. The authors noted that in England and Wales an estimated 50 families a year experienced a second unexpected infant death.[193] Deaths in infants are often of multifactorial cause, and frequently due to the coincidence of several disease processes, some of which in isolation would not necessarily be fatal.[194] They cited as an example, where the amount of inflammatory change of the respiratory tract that would put a child with a moderate degree of bronchopulmonary dysplasia into failure would not be expected to kill a normal child.[195]

134. In relation to the diagnosis of possible or probable filicide, the authors observed that in many SIDS deaths the pathology findings are indistinguishable from those of asphyxia, but cannot carry that diagnosis except as a possibility. In diagnosing filicide, other factors were considered such as presentation of Munchausen syndrome by proxy in relation to siblings.[196] Diagnosis of possible filicide in the study was only made if that was the unanimous opinion of those taking part in the case discussion.[197]

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188 R Meadow, 'Recurrent Cot Death and Suffocation' (1989) 64 *Archives of Disease in Childhood* 179, citing J L Emery, 'Infanticide, Filicide and Cot Death' (1985) 60 *Archives of Disease in Childhood* 505.

189 R Meadow, 'Recurrent Cot Death and Suffocation' (1989) 64 *Archives of Disease in Childhood* 179, 179.

190 Warren G Guntheroth, Rüdiger Lohmann and Phillip S Spiers, 'Risk of Sudden Infant Death Syndrome in Subsequent Siblings' (1990) 116(4) *Journal of Pediatrics* 520. This paper was addressed in Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 6, n 98.

191 Warren G Guntheroth, Rüdiger Lohmann and Phillip S Spiers, 'Risk of Sudden Infant Death Syndrome in Subsequent Siblings' (1990) 116(4) *Journal of Pediatrics* 524.

192 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 873.

193 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 873.

194 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 873.

195 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 873.

196 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 873-874.

197 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 874.

135. Ten of the 641 families within the FSID programme suffered a second death.[198] One family experienced three deaths.[199] Two of the 21 total deaths, in the same family, appeared to be completely unexplained – true SIDS.[200] Eight deaths were completely or partially explained by natural causes. Two families (four deaths) had a strong history of familial disease which might have contributed. Two babies died during potentially treatable illnesses in families with many other significant and possibly contributory factors. Nine deaths in five families were thought to be most probably filicide. Two deaths had inadequate information. Four families were living in profound social deprivation.[201]

136. Of 17 families referred to (rather than self-selecting to be involved in) the FSID programme, three of 36 deaths remained completely unexplained – true SIDS.[202] Three were fully or partially explained by natural causes. Seven were thought to probably be due to accidental smothering. In 22 deaths in 12 families, the deaths were assessed to probably be filicide; the article appears to suggest that some of these however may have been attributable to other causes.[203]

137. The authors noted that studies showed that subsequent infants were "at greatly increased risk".[204] However, only five of their total series were assessed as true SIDS deaths, and in only one family were both deaths in this category. This suggested the chance of recurrence was very small, probably no greater than the general occurrence of such deaths.[205] Consistently with other work in child abuse, the authors said they were rarely completely certain, but in all the "probably filicide" cases, conclusions were that filicide was overwhelmingly the most probable cause. The authors also observed that the excess of deaths in the neonatal period and after six months of age showed that families with repeat deaths were not typical of SIDS death families as a whole.[206]

138. The authors concluded that repeat SIDS deaths do not form a homogeneous group but occur over a wider age range than families with a single SIDS death and in families with many other problems.[207]

Pinholster (1994)

139. This article in the journal *Science* was about a then-landmark research paper by Steinschneider in the October 1972 issue of *Pediatrics* which detailed two cases of SIDS in a single family which had experienced three previous sibling deaths.[208] The family was that of Waneta Hoyt, who in 1994 was charged with murder of her two children. The 1972 paper became one of the most widely cited papers in SIDS research, providing support for the theory of inborn abnormality.[209]

198 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 874.
199 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 874.
200 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 874.
201 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 874-875.
202 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 874-875.
203 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 875-876.
204 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 875-876.
205 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 876.
206 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 876.
207 S Wolkind et al, 'Recurrence of Unexpected Infant Death' (1993) 82 *Acta Paediatrica* 873, 876.
208 Alfred Steinschneider, 'Prolonged Apnea and the Sudden Infant Death Syndrome: Clinical and Laboratory Observations' (1972) 50(4) *Pediatrics* 646.
209 G Pinholster, 'SIDS Paper Triggers a Murder Charge' (1994) 264 *Science* 197, 197.

Meadow's law (1997)

140. British paediatrician Sir Roy Meadow proposed a maxim to be applied when considering multiple infant deaths in a single family which came to be known as "Meadow's law". Meadow's law suggested that "one cot death is a tragedy, two cot deaths is suspicious, and until the contrary is proved, three cot deaths is murder."[210] Although attributed to Meadow, the maxim first arose in DiMaio and DiMaio (1989):

> *While a second SIDS death from a mother is improbable, it is possible and she should be given the benefit of the doubt. A third case, in our opinion is not possible and is a case of homicide.*[211]

141. Meadow's law was based on the belief that three infant deaths in a family is so statistically improbable that the only reasonable explanation for the deaths is murder. The use of such reasoning has now been widely discredited, including in the Goudge Inquiry which inquired into paediatric forensic pathology in Ontario in 2008, and in R v Cannings.[212]

Foundation for the Study of Infant Deaths (1998) (Care of Next Infant paper)

142. FSID, referred to above, developed the Care of Next Infant ("CONI") program to help provide organised support to families with children born after a cot death.[213] CONI collected national data from across its centres in the United Kingdom.[214] In addition, CONI was extended to other groups of parents. CONI PLUS included parents who had had a close relative die from SIDS and parents whose child had suffered an ALTE.[215]

143. This paper reported on 5,000 babies in the CONI (but not CONI PLUS) program who had completed a period of surveillance under the program. Forty-four of the 5,000 babies died while on the program or after completion. Thirty-five were unexpected. Two were under one week and two over one year. The 40 post-perinatal deaths (within eight days to one year) were compared with national figures – in England and Wales deaths from all causes in this period fell from 5.15/1,000 in 1988 to 2.80/1,000 in 1996. In CONI, it was 8/1,000.[216]

144. The authors reported that 104 of the parents in the CONI program had experienced two previous baby deaths, and four had experienced three previous deaths.[217] Of those four, one was three SIDS, one was two SIDS plus one other cause, one was one SIDS plus two other causes, one was three other causes.

145. The authors reported on mortality in the program. Although the post-perinatal death rate of 8/1,000 was considerably higher than the national rates, the authors pointed out that they were not dealing with a normal birth population.[218] Of the 35 children who died unexpectedly on the program, eight (1.6/1,000) were finally categorised as true cot deaths.[219] There were eight unexplained deaths, one of which had minimal disease and the others of which had none.[220] Fourteen were non-natural deaths. Two had an inadequate necropsy, seven were not investigated, two were pending investigation.[221]

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210 Roy Meadow (ed), ABC of Child Abuse (BMJ Publishing, 3rd ed, 1997) 27-29.

211 Dominick J DiMaio and Vincent V J DiMaio, Forensic Pathology (CRC Press, 1st ed, 1989) 503.

212 Stephen T Goudge, Report of the Inquiry into Pediatric Forensic Pathology in Ontario (Ontario Ministry of the Attorney-General, 1 October 2008); *R v Cannings* [2004] EWCA Crim 1, [177].

213 Foundation for the Study of Infant Deaths, *Report on 5,000 Babies using the CONI (Care of Next Infant) Programme* (October 1998) 3.

214 Foundation for the Study of Infant Deaths, *Report on 5,000 Babies using the CONI (Care of Next Infant) Programme* (October 1998) 3.

215 Foundation for the Study of Infant Deaths, *Report on 5,000 Babies using the CONI (Care of Next Infant) Programme* (October 1998) 4.

216 Foundation for the Study of Infant Deaths, *Report on 5,000 Babies using the CONI (Care of Next Infant) Programme* (October 1998) 6

217 Foundation for the Study of Infant Deaths, *Report on 5,000 Babies using the CONI (Care of Next Infant) Programme* (October 1998) 6.

218 Foundation for the Study of Infant Deaths, *Report on 5,000 Babies using the CONI (Care of Next Infant) Programme* (October 1998) 20.

219 Foundation for the Study of Infant Deaths, *Report on 5,000 Babies using the CONI (Care of Next Infant) Programme* (October 1998) 20.

220 Foundation for the Study of Infant Deaths, *Report on 5,000 Babies using the CONI (Care of Next Infant) Programme* (October 1998) 27.

221 Foundation for the Study of Infant Deaths, *Report on 5,000 Babies using the CONI (Care of Next Infant) Programme* (October 1998) 27.

146. The families who had enrolled in the CONI program had experienced high rates of infant deaths – 17.5/1,000.[222] The authors stated that "[w]ithout question we are dealing with families where there is increased risk of multiple deaths and this has to be critically assessed".[223]

Evidence of recurrence of SIDS/unexplained deaths in literature after 2003

Bohnert, Große Perdekamp and Pollak (2004)

147. This article reported on a family with three deaths in infancy (11 weeks, seven weeks, two weeks), each attributed to SIDS.[224] A fourth daughter survived and lived separately from the mother, but with her father. The autopsies did not reveal any pre-existing pathological organ findings except for acute pulmonary emphysema and extensive intra-alveolar bleeding. There was a strong suspicion of mechanical suffocation, and subsequently the mother confessed to suffocation of the first two infants, and that the third baby was smothered by the father.

148. The article discusses contemporary learning at the time of the risks of recurrence, noting that some papers had concluded that the probability for recurrence was not higher than the baseline probability (citing Peterson, Sabotta and Daling, 1986), and a very low recurrence risk identified by Beal,[225] but that the Norwegian studies had identified 5.6/1,000 and 5.8/1,000, and similarly in Guntheroth, Lohmann and Spiers (1990).[226] The authors acknowledged issues associated with inclusion, in studies of this type, of infant deaths due to illnesses, and that SIDS diagnoses were not always exactly verified – especially death from asphyxia which was often not ruled out with sufficient accuracy. Multiple authors had pointed out that many recurrent infant deaths in the same family are homicides.[227]

149. Upon review of literature, the authors noted that because the percentage of deaths due to suffocation is difficult to assess, figures in literature ranged from 0.6 per cent to 10 per cent, although it was assumed that about two per cent of deaths primarily classified as SIDS were homicides, with difficulty differentiating between suffocation and SIDS.[228]

Hill (2004)

150. Hill observed that in recent years there appeared to have been a trend to regard multiple deaths with much more suspicion than single deaths, perhaps inspired by Meadow's law.[229] However, Meadow's Law turns traditional British justice on its head.

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222 Foundation for the Study of Infant Deaths, *Report on 5,000 Babies using the CONI (Care of Next Infant) Programme* (October 1998) 20.

223 Foundation for the Study of Infant Deaths, *Report on 5,000 Babies using the CONI (Care of Next Infant) Programme* (October 1998) 20.

224 M Bohnert, M Große Perdekamp and S Pollak, 'Three Subsequent Infanticides Covered up as SIDS' (2004) 119(1) *International Journal of Legal Medicine* 31.

225 Susan M Beal, 'Siblings of Sudden Infant Death Syndrome Victims' (1992) 19(4) *Clinics in Perinatology* 839.

226 M Bohnert, M Große Perdekamp and S Pollak, 'Three Subsequent Infanticides Covered up as SIDS' (2004) 119(1) *International Journal of Legal Medicine* 31, 31.

227 M Bohnert, M Große Perdekamp and S Pollak, 'Three Subsequent Infanticides Covered up as SIDS' (2004) 119(1) *International Journal of Legal Medicine* 31, 31.

228 M Bohnert, M Große Perdekamp and S Pollak, 'Three Subsequent Infanticides Covered up as SIDS' (2004) 119(1) *International Journal of Legal Medicine* 31, 33.

229 Ray Hill, 'Multiple Sudden Infant Deaths – Coincidence or Beyond Coincidence?' (2004) 18(5) *Paediatric and Perinatal Epidemiology* 320, 320. This paper was addressed in Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 6, n 95.

151. Hill's paper, however, had as its main purpose estimating various probabilities in order to establish whether Meadow's Law had any scientific substance.

152. Professor Hill made some salient observations in his paper. He stated that there was no doubt that the occurrence of two or more SIDS in the same family will be a rare event, just as the occurrence of two or more infant murders in the same family will be a rare event.[230]

153. He also considered the increased risk of SIDS if there has already been a SIDS in the family, stating that it was "intuitively clear" that an infant in a family which has already suffered a SIDS will be at increased risk, because many genetic and environmental factors will be the same.[231] He referred to the earlier Confidential Enquiry for Stillbirths and Deaths in Infancy ("CESDI", discussed below), which studied the deaths of babies in five regions of England from 1993 to 1996. According to the CESDI report, of the 323 SIDS families studied, there were five previous SIDS, while among the 1,288 control families, there were two previous SIDS. Professor Hill stated that this suggested a "dependency factor" of about 10 – that a baby was 10 times more likely to be a SIDS victim if a previous sibling had died of SIDS.[232] There was not a great discrepancy between this and the CONI figure of 5.7 – in light of all the data, it seemed reasonable to estimate the risk of SIDS was between five and 10 times greater for a second sibling.[233]

154. Professor Hill used these estimates to calculate, by employing mathematical formulae, whether double SIDS or double homicide was more likely, and whether triple SIDS or triple homicide was more likely.

155. Professor Hill referred to data provided by Carpenter, one of the authors of the draft report on the CONI program.[234] In particular, Carpenter had found that there were nine families that suffered three infant deaths. Carpenter had reported that in eight of the nine, all three deaths were natural, and one was triple homicide.[235] The eight triple natural deaths could be broken down as two cases of triple SIDS, three of double SIDS plus one explained or accidental death, two cases of single SIDS plus two explained deaths, and one case of three explained deaths. On this basis, Professor Hill considered it noteworthy that within the set of nine families, the cases agreed exactly with a two to one ratio of triple SIDS to triple homicide.[236]

156. Professor Hill returned to recurrent SIDS in his 2005 paper.[237] He discussed the *Cannings* case (raised by Ms Folbigg in her 2004 appeal to the Court of Criminal Appeal)[238] as well as the cases of *R v Patel* and *R v Clark*, and in particular the misuse of statistics in the *Clark* trial.[239]

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230 Ray Hill, 'Multiple Sudden Infant Deaths – Coincidence or Beyond Coincidence?' (2004) 18(5) *Paediatric and Perinatal Epidemiology* 320, 321.

231 Ray Hill, 'Multiple Sudden Infant Deaths – Coincidence or Beyond Coincidence?' (2004) 18(5) *Paediatric and Perinatal Epidemiology* 320, 321-322.

232 Ray Hill, 'Multiple Sudden Infant Deaths – Coincidence or Beyond Coincidence?' (2004) 18(5) *Paediatric and Perinatal Epidemiology* 320, 322.

233 Ray Hill, 'Multiple Sudden Infant Deaths – Coincidence or Beyond Coincidence?' (2004) 18(5) *Paediatric and Perinatal Epidemiology* 320, 322.

234 Ray Hill, 'Multiple Sudden Infant Deaths – Coincidence or Beyond Coincidence?' (2004) 18(5) *Paediatric and Perinatal Epidemiology* 320, 323, citing A Waite et al, 'Repeat Sudden Infant Deaths in Families Enrolled on to a Support Programme' (Draft manuscript, November 2002).

235 Ray Hill, 'Multiple Sudden Infant Deaths – Coincidence or Beyond Coincidence?' (2004) 18(5) *Paediatric and Perinatal Epidemiology* 320, 324, citing R G Carpenter et al, 'Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?' (2005) 365 *Lancet* 29.

236 Ray Hill, 'Multiple Sudden Infant Deaths – Coincidence or Beyond Coincidence?' (2004) 18(5) *Paediatric and Perinatal Epidemiology* 320, 324.

237 Ray Hill, 'Reflections on the Cot Death Cases' (2005) 2 *Significance* 13.

238 See Chapter 3 at [315]-[344] and below at [247]-[250].

239 *R v Cannings* [2004] EWCA Crim 1; *R v Patel* (Reading Crown Court, Jack J, 11 June 2004); *R v Clark* [2003] EWCA 1020. See below at [240]-[246].

Carpenter et al (2005)

157. In 2005, Carpenter and colleagues published this paper in which the authors reported the proportion of natural and unnatural infant (<1 year) deaths occurring in families enrolled in the CONI program, and which subsequently became the focus of criticism.[240]

158. The authors further analysed recurrent deaths recorded by the CONI program by conducting interviews and post-mortem investigations as part of a continuing audit of the CONI program.[241]

159. By December 1999, 6,373 babies from 5,229 families had completed the CONI program. Fifty-seven (8.9/1,000) babies died under the age of one year. Forty-eight were sudden and unexpected, seven of which were classified as probable homicides and 41 (including one second CONI death, or a third in the family) were "natural sudden unexpected deaths in infancy". Therefore, the proportion of SUDI deaths was 5.86 times greater than the proportion of probable homicides.[242] The relative risk of recurrence as compared with the general population was at least 5.71 (confidence interval 4.10-7.74).

160. Enquiries were completed for 27 of the remaining 40 first CONI deaths (i.e. second death in the family), and partially completed for the balance of 13. Of the 27, five were explained (e.g. by very-long-chain acyl-coA dehydrogenase deficiency ("VLCAD") or long QT syndrome) and 22 were SIDS.[243] Of those 22 cases of SIDS, 18 involved two cases of SIDS – in four, the previous (pre-CONI) death was explained.[244] The repeat death in 18 families gave a relative risk of 5.9, although the accuracy of diagnosis was subsequently questioned in papers discussed below.

161. In all of the 18 families with two cases of SIDS, the risk factors for SIDS were high – smoking, illicit drug use, unemployment; many of the families demonstrated disorganisation and social deprivation. In at least six, there were mental health concerns. Nine babies of the 36 deaths were in bed with one or both parents; in nine further, pathological findings were compatible with an asphyxial component to the death.

162. In four of the 18 families, the CONI baby who died was the second or third CONI baby in the family.[245]

163. In 13 families with two deaths, data was insufficient to show whether the deaths were explained or due to SIDS.[246] Seven families (14 deaths) declined enquiries, however, one had congenital abnormalities and one had rib fractures. One mother had 10 live births – two previous SUDI and a CONI death, after which all the children were taken into care. Four of these 14 deaths were in an unsafe sleep environment. The remaining six of the 13 families were lost to investigation: the mother was murdered in one; there were extensive legal investigations in two; and of the remaining three there was no evidence of injury, although two were found in an unsafe sleep environment.

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240 R G Carpenter et al, 'Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?' (2005) 365 *Lancet* 29.
241 R G Carpenter et al, 'Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?' (2005) 365 *Lancet* 29, 30.
242 R G Carpenter et al, 'Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?' (2005) 365 *Lancet* 29, 31.
243 R G Carpenter et al, 'Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?' (2005) 365 *Lancet* 29, 31.
244 R G Carpenter et al, 'Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?' (2005) 365 *Lancet* 29, 32.
245 R G Carpenter et al, 'Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?' (2005) 365 *Lancet* 29, 32.
246 R G Carpenter et al, 'Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?' (2005) 365 *Lancet* 29, 32.

164. The authors concluded that of the 46 first unexpected CONI deaths, five were probably filicides, one was homicide by a babysitter, and 40 were SUDI, "none of which were deemed to have been unnatural".[247]

> **Findings** 57 (8·9 per 1000) CONI infants died. Nine deaths were inevitable, and 48 were unexpected. 44 families lost one child, and two families lost two children. Of the 46 first CONI deaths, 40 were natural; the other six were probable homicides, five committed by one or both parents (two criminally convicted). The ratio of 40 natural to six unnatural deaths is 6·7 (95% CI 2·8–19·4). Enquiries identified 18 families with two SIDS (sudden infant death syndrome) deaths and two families with probable covert double homicides (ratio 9·0 [2·2 to 80·0]). There were no convictions in 13 incomplete cases. Families with three deaths are reported.
>
> **Interpretation** Repeat unexpected infant deaths are most probably natural.

Figure 2: Extract of summary from Carpenter et al (2005): Exhibit AN in the Inquiry.

165. The report identified two families who lost two CONI infants, i.e. lost three infants including the index death which resulted in them being in the program. In one, all the deaths were homicides. In the other, twins died six weeks apart. A third family in which there had previously been two sudden unexpected deaths had a CONI death – all three were registered in SIDS categories. The authors identified a further six families who had lost three infants before participating in CONI – of these, one had three SIDS cases and the other five had at least one explained death. After January 2000, a further family was identified as having had a second death on the CONI program, and all three deaths remained unexplained. Therefore, there were four families with three SIDS or unexplained deaths.

	Final classification by					Final allocation			
						Total homicide		Total natural	Total
	Coroner and CONI panel	Coroner's court only	Family court	Criminal court	Appeal court	Due to parents (filicide)	Due to others (not family)		
First CONI death									
Overt homicide	..	..	3	1	..	3	1	..	4
Probable covert homicide									
Initially SIDS	(1)	..	..	1*	..	1	..	..	1
Initially unascertained	..	..	1	..	..	1	..	..	1
Natural (explained/SIDS)	27	..	..	..	..		..	27	27
Natural (incomplete information)	..	11†	..	1	1	..		13	13
Second CONI death									
Confessed homicide	..	..	..	1		1	..	..	1
SIDS	1	..	..	..		..	..	1	1
Total						6	1	41	48

Excludes four families in which either the index death or the CONI death occurred after 1 year of age. *Initially registered as SIDS, but the father was subsequently convicted of three homicides, including this child. †Some of the families were the subject of Social Service case conferences. We have no information that any were the subject of Family Court or criminal proceedings in relation to the death of the CONI baby.

Table 1: A general grouping of the deaths

Figure 3: Table representing the 48 unexpected deaths on the CONI program reported in Carpenter et al (2005): Exhibit AN in the Inquiry

166. One object of the enquiries in the study was to identify cases of probable filicide. Seven deaths were classified as probably unnatural – in three there were convictions, three were due to overt injuries, and in one a family court found the father responsible. In the 27 cases the authors considered there was no suspicion of filicide, including 18 where both deaths were unexplained albeit there may have been less than optimum care and concern about safety of subsequent children.

167. The authors concluded that the proportion of homicides appeared much lower than in previous studies of recurrence. They acknowledged that differences of definition probably accounted for discrepant findings, and that they had counted as SIDS several cases in which asphyxia might have been a contributory factor – however, there was no way to distinguish reliably between SIDS and unnatural death without a confession or evidence of violence.[248]

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247 R G carpenter et al, 'Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?' (2005) 365 *Lancet* 29, 32.

248 R G Carpenter et al, 'Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?' (2005) 365 *Lancet* 29, 33.

168. Overall, the authors stated that their data suggested that second deaths were not rare and the majority – 80-90 per cent (40 in 45; or 18 in 20) – were natural. Also, there were families who had experienced three unexpected deaths.[249] They concluded that in their belief, the occurrence of a second or third sudden unexpected death in infancy within a family, although relatively rare, was in most cases from natural causes.

Bacon (2005)

169. Soon after the Carpenter article was published, in separate letters to *The Lancet,* Professor Christopher Bacon and Professor Vincent DiMaio challenged the findings.[250]

170. Professor Bacon was concerned that the authors had not taken sufficient account of two possible bias sources. First, as both participation in CONI and cooperating with enquiries of Carpenter and his colleagues were voluntary, there were two stages of self-selection.

171. Secondly, and more problematically, there was scope for bias in decisions as to whether a death was natural or unnatural. Recognition of covert homicide in infancy is "notoriously difficult";[251] the history may be fictitious, the autopsy unhelpful, and diagnostic criteria were not agreed. Judgments were largely speculative.

172. In this context, several deaths attributed to SIDS by Carpenter et al had features which "must give rise to concern".[252] Thus, Professor Bacon stated, instead of dichotimising the cases into unnatural or natural, it would have been more accurate to have a grey area of uncertainty. In science, presumption and benignity had no place, all possibilities compatible with the evidence had to be considered. The data did not support such clear-cut conclusions as promoted by the authors, and instances of homicide may be higher than the Carpenter analysis suggested.

DiMaio (2005)

173. Professor Vincent DiMaio was also critical of the Carpenter article. Views previously published by him and Dr Dominick DiMaio were characterised in the Carpenter article as being that siblings have the same risk as the general population, but with a squared or cubed probability to find that a third case was not possible and was homicide.[253]

174. In his letter to *The Lancet,* Professor DiMaio disavowed that he would advocate ruling multiple infant deaths in a family solely on the basis of statistical probabilities, statistics being unreliable. He described the data used by Carpenter et al as "dubious" – they had failed to comprehend that a diagnosis of SIDS actually means that one does not know why a child has died. It is, he said, a "wastebasket" diagnosis.[254] Most such deaths are probably due to a natural disease of some sort, some due to overlaying and others to deliberate smothering.

175. Professor DiMaio noted that within the "natural" 40 deaths, Carpenter et al seemed to include cases in which overlaying may have occurred, and cases for which a natural cause of death was determined which would not be regarded as unexplained. Being SIDS, some (or all) could have been natural, some (or all) could have been accidents, some (or all) could have been homicides.

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249 R G Carpenter et al, 'Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?' (2005) 365 *Lancet* 29, 34.

250 C J Bacon, 'Repeat Sudden Unexpected Infant Deaths' (2005) 365 *Lancet* 1137; Vincent J M DiMaio, 'Repeat Sudden Unexpected Infant Deaths' (2005) 365 *Lancet* 1137.

251 C J Bacon, 'Repeat Sudden Unexpected Infant Deaths' (2005) 365 *Lancet* 1137; Vincent J M DiMaio, 'Repeat Sudden Unexpected Infant Deaths' (2005) 365 *Lancet* 1137, 1137.

252 C J Bacon, 'Repeat Sudden Unexpected Infant Deaths' (2005) 365 *Lancet* 1137; Vincent J M DiMaio, 'Repeat Sudden Unexpected Infant Deaths' (2005) 365 *Lancet* 1137, 1138.

253 R G Carpenter et al, 'Repeat Sudden Unexpected and Unexplained Infant Deaths: Natural or Unnatural?' (2005) 365 *Lancet* 29, 29, citing V J DiMaio and D DiMaio, *Forensic Pathology* (CRC Press, 1st ed, 1989).

254 Vincent J M DiMaio, 'Repeat Sudden Unexpected Infant Deaths' (2005) 365 *Lancet* 1137, 1137.

Carpenter et al (2005) – Authors' reply

176. Professor Carpenter and the other authors (except Professor Emery, who died in 2000) wrote in reply to *The Lancet*.[255] They did not accept a grey category would be appropriate, arguing that just because high risk factors applied, this did not imply intentional filicide. They also did not accept Professor DiMaio's assertion that "SIDS deaths appear to occur in families at random",[256] because it would follow that a repeat could not be natural. They acknowledged that some SIDS cases might be due to filicide, but argued that their enquiries were complete in 27 pairs of deaths, and court findings were relied upon in 13. They stood by their conclusion.

CESDI and South West Infant Sleep Scene Study[257]

177. Between 1993 and 1996, the CESDI commissioned a case-control study to monitor the characteristic profiles of infant deaths following the dramatic reduction in death rates after the Back to Sleep campaign in England and Wales in November 1991.[258] The CESDI study was conducted via the University of Bristol across a third of England. The South West Infant Sleep Scene ("SWISS") Study was similar, conducted between 2003 and 2006 in a smaller geographical area.

178. The Back to Sleep campaign reduced the SIDS rate in England and Wales from 1,597 SIDS deaths in 1988 to 531 by 1992. The CESDI and SWISS studies provided an evidence base for further SIDS risk reduction, to 212 SIDS deaths in 2014. Both studies involved notification networks to enable rapid response to incidents to document narrative accounts of circumstances of the deaths, and further consultation with families to obtain broader information about the death.

CESDI

179. During the CESDI study period, 456 SUDI cases were identified. Ninety-three were later fully explained and 363 were classified as SIDS. The study found there were four significant predictors: maternal smoking during pregnancy, low maternal age (<26 years), larger families (three or more children) and poor socioeconomic status.[259] Three or more of these predictors identified 42 per cent of SIDS families from eight per cent of the population. Infants put on their side or front for sleep were at risk.[260] Significantly more of the SIDS infants were wrapped too warmly and used a duvet.[261] Significantly more SIDS infants slept with a parent in the parental bed or on a sofa, and unobserved outside the parental bedroom.[262]

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255 Professor Emery was included as a co-author after his death due to his work on the CONI program which generated the data.

256 R G Carpenter et al, 'Repeat Sudden Unexpected Infant Deaths' (2005) 365 *Lancet* 1138, 1138.

257 This summary of the CESDI and SWISS studies is taken from Exhibit D, Peter S Blair, Anna S Pease and Peter J Fleming, 'Observational Investigations from England: The CESDI and SWISS Studies' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 325.

258 Exhibit D, Peter S Blair, Anna S Pease and Peter J Fleming, 'Observational Investigations from England: The CESDI and SWISS Studies' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 325, 326.

259 Exhibit D, Peter S Blair, Anna S Pease and Peter J Fleming, 'Observational Investigations from England: The CESDI and SWISS Studies' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 325, 330.

260 Exhibit D, Peter S Blair, Anna S Pease and Peter J Fleming, 'Observational Investigations from England: The CESDI and SWISS Studies' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 325, 333.

261 Exhibit D, Peter S Blair, Anna S Pease and Peter J Fleming, 'Observational Investigations from England: The CESDI and SWISS Studies' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 325, 333.

262 Exhibit D, Peter S Blair, Anna S Pease and Peter J Fleming, 'Observational Investigations from England: The CESDI and SWISS Studies' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 325, 333.

180. The majority of both SIDS and control babies were generally well in the 24 hours prior to the last sleep, although 11 per cent of SIDS infants needed a doctor or emergency medical attention compared with four per cent of controls.[263]

SWISS

181. The SWISS study identified 157 SUDI cases, with 90 meeting the criteria for SIDS (0.49/1,000, similar to the national rate at the time).[264] The age distribution of the SIDS infants was significantly different from the CESDI study, with a median age of 66 days which was almost a month younger than the SIDS infants in CESDI.[265] Of the study group, 54 per cent died co-sleeping (compared with 20 per cent of controls); in 31 per cent parental use of alcohol was recent (three per cent in controls). These environmental factors were found to be virtually identical to their prevalence in the CESDI study.[266]

Gornall (2006)

182. In a further critique of the Carpenter et al (2005) publication, Professor Gornall claimed that the authors had re-categorised deaths that Professor Emery – who died two years before the first draft was completed – had classed as unnatural or indeterminate, and that Professor Emery would not have supported its conclusion.[267] Professor Emery was one of the early proponents of the theories that SIDS involves inter-related causal spheres of influence, proposing risk factors that contributed to Filiano and Kinney's triple risk model in 1994.[268]

183. The author pointed out that when Professor Emery published on CONI data in 1998 (when 5,000 babies had been through the program), 14 of 35 unexpected deaths were unnatural. However, of the 46 unexpected deaths identified by Carpenter and colleagues, the authors concluded that only six were unnatural. Dr Waite, one of the authors, had explained that six deaths had been re-categorised, two where coroners had recorded open verdicts and four where overlying infants had died while sleeping with their parents.[269] Deaths of children over one year had been disregarded. No explanation was given for recategorising all 13 deaths for which there was insufficient information to reach a conclusion.

184. The conclusion that 87 per cent of repeat sudden infant deaths are natural, moreover, did not reflect that the authors themselves had stated they could not exclude the possibility that some were covert homicide.

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263 Exhibit D, Peter S Blair, Anna S Pease and Peter J Fleming, 'Observational Investigations from England: The CESDI and SWISS Studies' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 325, 334.

264 Exhibit D, Peter S Blair, Anna S Pease and Peter J Fleming, 'Observational Investigations from England: The CESDI and SWISS Studies' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 325, 338.

265 Exhibit D, Peter S Blair, Anna S Pease and Peter J Fleming, 'Observational Investigations from England: The CESDI and SWISS Studies' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 325, 339.

266 Exhibit D, Peter S Blair, Anna S Pease and Peter J Fleming, 'Observational Investigations from England: The CESDI and SWISS Studies' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 325, 340.

267 Jonathan Gornall, 'Was Message of Sudden Infant Death Study Misleading?' (2006) 333 *British Medical Journal* 1165, 1165.

268 See Exhibit D, Roger W Byard, 'Sudden Infant Death Syndrome: Definitions' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 1, 2.

269 Jonathan Gornall, 'Was Message of Sudden Infant Death Study Misleading?' (2006) 333 *British Medical Journal* 1165, 1166.

185. In a communication with the *British Medical Journal*, Professor Carpenter was clear that "unnatural" meant filicide – everything else was "natural".[270] Professor Gornall described this as creating an illogical corollary that all the deaths that were not unnatural must be natural; this approach was correct in a criminal court, but not in scientific research. Professor Emery himself had recognised the importance of the distinction in his 1993 paper (referred to above at [131]).

186. Shortly before he died, Professor Emery had expressed the view in a report prepared at the request of Ms Clark's legal representatives (the trial was in November 1999), that he had been further confidentially studying the occurrence of repeat unexpected deaths, and in 100 of such deaths all the families had two cot deaths.[271] He found that approximately a third were due to a series of rare natural causes missed at necropsy; a third were associated with child abuse and had features indicating that they were unnatural; a third involved no suspicion of unnatural death but no finding of a natural cause. He had concluded that the occurrence of two unexpected deaths raised a definite suspicion of unnatural death, in his experience confirmed in a third of such cases.[272]

187. Professor David Hall, then immediate past president of the Royal College of Paediatrics and Child Health, had written to *The Lancet's* editor expressing alarm at the Carpenter paper. He described the analysis as seriously flawed, and the findings as "seriously misleading".[273]

188. Gornall identified the following "summary points" in the paper:

> *an analysis of sudden infant deaths suggested that almost 90% of second deaths in the same family are natural;*
>
> *classification of deaths in the study was changed after the death of one of the senior authors;*
>
> *deaths of indeterminate cause were counted as natural;*
>
> *the study was used as evidence in murder appeals.*[274]

Carpenter et al (2007)

189. In a response to Professor Gornall's article, Carpenter and colleagues denied that they materially changed the cause of death for any case which Professor Emery knew about. Inter alia, they stated that in their study, four infants who died in bed with parents were initially classified as "non-natural" but were subsequently categorised as SIDS in line with the CESDI SUDI study (see above at [171]).[275]

Bacon and Hey (2007)

190. The debate over the Carpenter publication continued in an article by Bacon and Hey in 2007 in which the authors re-analysed deaths described by Carpenter and colleagues as "natural".[276] "Natural", for their purposes, were deaths arising from disease or a wholly accidental event; "unnatural" were homicide; "covert homicide" were unnatural deaths that were not initially so recognised.

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270 Jonathan Gornall, 'Was Message of Sudden Infant Death Study Misleading?' (2006) 333 *British Medical Journal* 1165, 1166.
271 Jonathan Gornall, 'Was Message of Sudden Infant Death Study Misleading?' (2006) 333 *British Medical Journal* 1165, 1166-1167.
272 Jonathan Gornall, 'Was Message of Sudden Infant Death Study Misleading?' (2006) 333 *British Medical Journal* 1165, 1167.
273 Jonathan Gornall, 'Was Message of Sudden Infant Death Study Misleading?' (2006) 333 *British Medical Journal* 1165, 1167-1168.
274 Jonathan Gornall, 'Was Message of Sudden Infant Death Study Misleading?' (2006) 333 *British Medical Journal* 1165, 1168.
275 R G Carpenter et al, 'Author's Reply. Special Report Adds Nothing New, Say Paper Authors' (2007) 334 *British Medical Journal* 7.
276 C J Bacon and E N Hey, 'Uncertainty in Classification of Repeat Sudden Unexpected Infant Deaths in Care of Next Infant Programme' (2007) 335 *British Medical Journal* 129.

191. Bacon and Hey observed that one of the difficulties in distinguishing between SIDS and covert homicide is that both tend to occur against a background of social disadvantage. The 18 families with two deaths could have been associated with either. Five of the incompletely investigated 13 families had been categorised as "natural" deaths following previous prosecution without conviction in two and State intervention to take children into care in three, but Bacon and Hey categorised the five cases as undetermined. In another seven, classification of all the deaths as natural seemed unwarranted. In the remaining family the mother was murdered.

192. Overall, Bacon and Hey concluded with 13 per cent probably unnatural, 43 per cent probably natural, and 43 per cent undetermined. This contrasted with 87 per cent natural as found by Carpenter and colleagues and was closer to Emery and to Wolkind and colleagues (above at [131]).[277]

193. So reconsidered, the authors' purpose was to show how a comparatively small change of perspective could result in a large change to conclusions. They also introduced notions of "probably", and "undetermined", being concerned that a simple dichotomy of "natural" and "unnatural" glossed over complexities and uncertainties and fostered polarisation. "Uncertainty may be uncomfortable, but it is truer to reality, more conducive to scientific inquiry, and safer for children."[278]

Bacon (2008)

194. Professor Bacon revisited the issue of recurrence in the context of the evidence led in prosecutions in the United Kingdom of estimates of recurrence ranging from one in 73 million to one in 200.[279] These estimates were both reliant upon error.

195. Professor Bacon re-examined the eight published studies of recurrent SIDS conducted in Australia, Norway, the United Kingdom and the United States between 1965 and 1999. Professor Bacon concluded that flaws in the studies invalidated the authors' estimates of the risk of recurrent SIDS, and the figures suggested were mainly too high.[280] He accepted, however, that on theoretical grounds a family who had lost an infant to SIDS may well be at an increased (but unquantifiable) risk because of the persistence of genetic and environmental influences.[281]

196. Professor Bacon concluded that risk varies widely between families. Above all, it is essential that the first death be thoroughly investigated, looking particularly for conditions that might recur, such as familial disease, covert homicide, or major risk factors for SIDS. If these could be excluded and the family takes reasonable precautions, they could be assured that the chance of recurrence is very small.[282]

Bacon et al (2008)

197. Professor Bacon co-authored a further article in *Archives of Disease in Childhood* that examined eight studies of recurrent SIDS published in England since 1970. This included reviewing again the Carpenter study and taking into account the CESDI study results which showed wide differences in SIDS risk between subgroups of the same population.[283]

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277 C J Bacon and E N Hey, 'Uncertainty in Classification of Repeat Sudden Unexpected Infant Deaths in Care of Next Infant Programme' (2007) 335 *British Medical Journal* 129, 131.

278 C J Bacon and E N Hey, 'Uncertainty in Classification of Repeat Sudden Unexpected Infant Deaths in Care of Next Infant Programme' (2007) 335 *British Medical Journal* 129, 131.

279 Christopher Bacon, 'Recurrence of Sudden Infant Death Syndrome' (2008) 122 *Pediatrics* 869, 869.

280 Christopher Bacon, 'Recurrence of Sudden Infant Death Syndrome' (2008) 122 *Pediatrics* 869, 869.

281 Christopher Bacon, 'Recurrence of Sudden Infant Death Syndrome' (2008) 122 *Pediatrics* 869, 869.

282 Christopher Bacon, 'Recurrence of Sudden Infant Death Syndrome' (2008) 122 *Pediatrics* 869, 869.

283 C J Bacon et al, 'How Common is Repeat Sudden Infant Death Syndrome?' (2008) 93 *Archives of Disease in Childhood* 323, 323.

198. The authors stated that of about 350 SUDI deaths in England and Wales each year, about two thirds are unexplained, and if thought natural are usually designated as SIDS. "SIDS", for the purposes of the paper included "unexpected infant deaths that are natural but have no specific cause, but not deaths from specific causes, whether natural or unnatural, that have not been detected." They felt that attribution to SIDS of deaths in the latter category falsely elevated the recurrence rate.

199. A summary of the studies identified by the authors was set out in the following table:

Table 1 Studies of recurrence of sudden infant death syndrome

		Siblings		Relative risk (95% CI)	
Study	**Place and date**	**Total**	**SIDS**	**vs population**	**vs controls**
Froggatt *et al*[13]	Northern Ireland, 1965–67	**362** p	4–8*	3.7 to **7.4**	**4.8 to 9.6**
Peterson *et al*[7]	USA up to 1978	839 s	18	**9.5**	
		582 n	11	**8.6**	
Irgens *et al*[6]	Norway, 1967–80	1043 s	5	3.7	
		712 n	4	4.4	
Peterson *et al*[9]	Washington state, 1969–84	810 s	6	3.4	1.9 (0.6 to 6.0)
		566 n	4	**3.2**	1.7 (0.4 to 6.6)
Beal and Blundell[10]	South Australia, 1970–85	660 p	14	10.1	
		637 n†	7	5.2‡	
Guntheroth *et al*[11]	Oregon, 1975–84	385 s	5	6.0 (2.8 to 12.8)	
		253 n	3	5.4 (2.0 to 14.8)	
Oyen *et al*[12]	Norway, 1967–88	426 n	5	**7.3**	5.8 (2.1 to 13.2)
Carpenter *et al*[5]	UK, 1988–99	6373 s	18	5.9 (4.1 to 8.1)	

Figures are those given by the authors, except for figures in bold, which are calculated from the authors' data.
Confidence intervals are given only when presented by the authors.
*Comprising four definite and four probable sudden infant death syndrome (SIDS); †maximum figure; ‡minimum figure.
n, next siblings; p, all previous siblings; s, all subsequent siblings.

Figure 4: Studies of reccurence of sudden infant death syndrome identified in Bacon et al (2008)

200. The authors set three basic criteria to gauge the accuracy of the studies – completion and accuracy of ascertainment, thorough investigation, and control comparisons. They found that all the studies fell short of these criteria.[284]

201. The importance of an autopsy was discussed in order to detect familial diseases and covert homicide. Homicide was not generally acknowledged as a possible cause of SUDI until Emery's work in the 1980s. Its detection in any case is problematic.[285]

202. The authors also observed that the importance of using controls matched for degree of risk for SIDS only became apparent in 2000 with the CESDI report. Without stratification, repeat SIDS in high-risk families will give a false impression of an increased risk for the population as a whole.[286]

203. The authors found that all of the studies were flawed in ways that would mostly have caused overestimation of the risk of SIDS recurrence. With the fallen incidence, it would be very difficult to mount a study large enough to yield a reliable figure. There remained, however, a theoretical argument that a family who has lost one baby by SIDS is likely to be more at risk than other families because many of the same genetic and environmental influences will apply for subsequent children.

[284] C J Bacon et al, 'How Common is Repeat Sudden Infant Death Syndrome?' (2008) 93 *Archives of Disease in Childhood* 323, 325.
[285] C J Bacon et al, 'How Common is Repeat Sudden Infant Death Syndrome?' (2008) 93 *Archives of Disease in Childhood* 323, 325.
[286] C J Bacon et al, 'How Common is Repeat Sudden Infant Death Syndrome?' (2008) 93 *Archives of Disease in Childhood* 323, 325.

204. The authors concluded that a family's risk for a second SIDS death was probably greater than the risk for a first death for their subgroup, but that the increase cannot be quantified and was almost certainly less than that suggested by most of the previous studies.[287] They cautioned against expressing opinions about repeat SIDS in the absence of good evidence, especially in the courts.

205. The authors recommended that families whose initial SIDS death was fully investigated and who have no major risk factors can be advised that, although the risk of a second death may be slightly increased, it remains very small. The continuing decline in SIDS incidence added reassurance – by 2005 in England and Wales it had fallen to 1/3,300 live births. Therefore, even if the near sixfold increase in recurrence rate found by Carpenter et al (2005) and by Øyen, Skjaerven and Irgens (1996) were correct, the predicted overall rate for subsequent children would still be less than one in 500.[288]

Campbell et al (2008)

206. The authors of this study investigated stratification by risk factors in computing the probability of a second SIDS death in a family.[289] Founded on the CESDI findings of major social risk factors – smoking, maternal age <27 and parity >1,[290] and unemployment – the authors investigated whether it was reasonable to assume that the risk of a second event was independent of the risk of the first.

207. The authors found that it was important to consider environmental factors to investigate risk of recurrence – in a community with high rates of risk factors, most second SIDS cases would occur in high-risk families and overall recurrence rate would be high, whereas in a community where these factors were not common, high-risk families would account for only a minority of second SIDS:

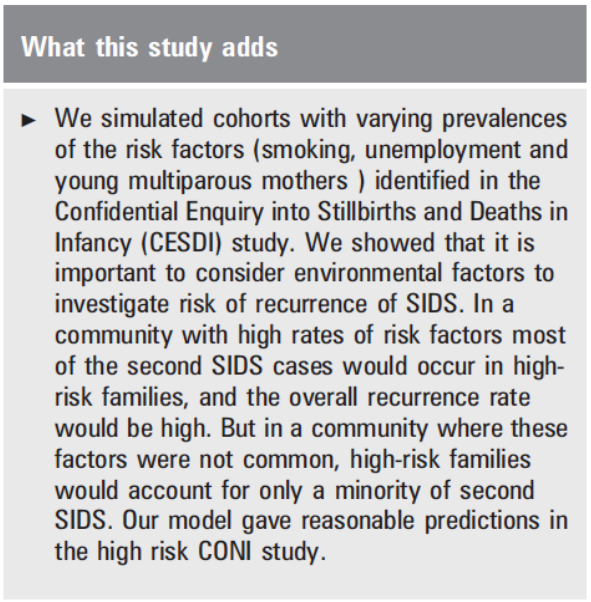

Figure 5: Summary of findings of Campbell et al (2008)

287 C J Bacon et al, 'How Common is Repeat Sudden Infant Death Syndrome?' (2008) 93 *Archives of Disease in Childhood* 323, 325.

288 C J Bacon et al, 'How Common is Repeat Sudden Infant Death Syndrome?' (2008) 93 *Archives of Disease in Childhood* 323, 326.

289 M J Campbell et al, 'Recurrence Rates for Sudden Infant Death Syndrome (SIDS): The Importance of Risk Stratification' (2008) 93 *Archives of Disease in Childhood* 936.

290 Parity is "the number of pregnancies that attained the gestation of viability irrespective of outcome": E Opara and J Zaidi, 'The Interpretation and Clinical Application of the Word "Parity": A Survey' (2007) 114 *BJOG: An International Journal of Obstetrics and Gynaecology* 1295, 1295.

208. Referring to eight previous studies that addressed the issue of recurrence, the authors observed that all eight studies appeared to assume that recurrence risk would be the same in all families – all reported an increase in risk after one SIDS.[291]

209. The authors suggested that the question to be asked was, rather, "what is the risk of a second SIDS in a given family if risk factors pertaining to that family at the time of the first SIDS persist subsequently?"[292] They agreed with Hill that it was a mistake to square the probability of a single SIDS to obtain the probability of two successive SIDS, since the events are not independent – however, conditional on known risk factors, it may be reasonable to multiply the risks. This formed the basis of their model, in which they applied risks of SIDS in families with zero, one, two or three risk factors from CEDSI in a predictive model.

210. Campbell et al observed that the steep social gradient in the risk and the dramatic change in incidence after the importance of sleeping position was recognised, together suggested that environmental influences played a major role in the pathogenesis of SIDS. The fact of a previous SIDS death increased the probability that the family had raised risk factors, but even taking these risk factors into account, the risk of a second event may be raised further since even a thorough review may fail to identify a familial disorder or covert homicide.[293]

211. Comparing the rate of second and subsequent deaths with the rate predicted from the risk factors applicable to the index cases, the analysis showed that the predicted number of second SIDS and distribution of index cases and second SIDS cases depended on the distribution of risk factors in the community. Therefore, in a community with high rates of risk (smoking, etc), the overall recurrence rate would be high. Whereas, in a community with low rates of risk, high-risk families would be a minority and overall recurrence rate would be much lower.[294]

212. The modelling suggested that that the risk of a second SIDS in families with no risk factors was very low.[295] They pointed out that recurrence risk studies based on a cohort identified by members having experienced a SIDS event had to adjust the predicted risk to that of the cohort rather than using risks in the general population.[296]

Blair and Fleming (2008)

213. The authors of this paper acknowledged at the outset the multifactorial nature of SIDS and the difficulty in trying to calculate recurrence risk for death due to disparate and largely unknown causes which potentially involve several independent mechanisms.[297]

214. Complexities included any genetic predisposition being presumed to interact with infections at a vulnerable developmental stage, as in the triple risk model – in such children, there was probably a relatively low risk of recurrence.[298] Cases of limited pathological investigations could mean failure to identify metabolic disorders, but the risk of recurrence in future siblings would be relatively high. The frequency of covert homicide is impossible to ascertain with certainty.[299]

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291 M J Campbell et al, 'Recurrence Rates for Sudden Infant Death Syndrome (SIDS): The Importance of Risk Stratification' (2008) 93 Archives of Disease in Childhood 936, 936.

292 M J Campbell et al, 'Recurrence Rates for Sudden Infant Death Syndrome (SIDS): The Importance of Risk Stratification' (2008) 93 *Archives of Disease in Childhood* 936, 936.

293 M J Campbell et al, 'Recurrence Rates for Sudden Infant Death Syndrome (SIDS): The Importance of Risk Stratification' (2008) 93 *Archives of Disease in Childhood* 936, 936.

294 M J Campbell et al, 'Recurrence Rates for Sudden Infant Death Syndrome (SIDS): The Importance of Risk Stratification' (2008) 93 *Archives of Disease in Childhood* 936, 938.

295 M J Campbell et al, 'Recurrence Rates for Sudden Infant Death Syndrome (SIDS): The Importance of Risk Stratification' (2008) 93 *Archives of Disease in Childhood* 936, 938.

296 M J Campbell et al, 'Recurrence Rates for Sudden Infant Death Syndrome (SIDS): The Importance of Risk Stratification' (2008) 93 *Archives of Disease in Childhood* 936, 938.

297 Peter S Blair and Peter J Fleming, 'Recurrence Risk of Sudden Infant Death Syndrome' (2008) 93 *Archives of Disease in Childhood* 269, 269.

298 Peter S Blair and Peter J Fleming, 'Recurrence Risk of Sudden Infant Death Syndrome' (2008) 93 *Archives of Disease in Childhood* 269, 269.

299 Peter S Blair and Peter J Fleming, 'Recurrence Risk of Sudden Infant Death Syndrome' (2008) 93 Archives of Disease in Childhood 269, 269.

215. Moreover, Bacon et al revealed another layer of complexity in underlying wrong assumptions that families in population-based studies are broadly representative of the population.[300] As pointed out by Bacon et al, studies had shown similar characteristic profiles of SIDS families, so recurrence risk can also be dependent upon the particular population. Campbell et al, acknowledging dependency of SIDS risk upon birth into a high-risk or low-risk environment, considered that it was difficult to fix a recurrence rate to a single estimate given death is most likely also due to a range of difference causes, the authors noted. Bacon et al observed, sensibly, that on a population level there are too many variants to resolve the issue.[301]

216. The authors concluded that consideration of risk of a subsequent SIDS should always take into account the known risk factors. The evidence suggested that for most families the chances of recurrence are low, although this should not negate an extensive review of whether familial disease contributed to the death, or the importance of advice and support on avoiding modifiable risk factors.[302]

The Lullaby Trust (2009)

217. The 10,000th infant to be registered on CONI was born on December 2006, providing an opportunity for review.[303]

218. Among babies enrolled in CONI from 2000, there had been 14 infant deaths (i.e. at least the second in the family) of which 13 were sudden and unexpected – none were overt homicides, one was a confessed homicide, and no covert homicides had come to light.[304] Therefore, over seven years the sudden unexpected infant death rate among CONI babies had declined by 47 per cent and probable homicides declined from 1.1/1,000 to 0.3/1,000. However, given the small figures this could have been due to chance.

219. The report observed that the data indicated that in CONI families compared with national data, inter alia, mothers were younger, twice as many parents were smokers, and unemployment of partners was over five times higher.[305] Referring to Carpenter et al, the report noted that ongoing analysis of CONI deaths suggested that infants in particular social classes were generally at even higher risk than the general CONI population.[306] A persistent five per cent of CONI babies continued to be laid to sleep on their fronts in the first three months after birth, and a similar proportion on their sides.[307] The report observed significant variability in breast feeding, birth weight, and monitoring efforts.[308]

Waite, McKenzie and Daman-Willems (2011)

220. In a defence of the continued relevance of the CONI program, Waite, McKenzie and Daman-Willems evaluated its data in light of the continued prevalence of risk factors and comfort offered to high-risk populations by the program. The authors observed that compared with national data, families in CONI had more factors associated with increased risk of cot death, which confirmed that a history of cot death identified a particular high-risk group. They noted that there were 312 cot deaths registered in the United Kingdom in 2008 (0.39/1,000).[309] At the time of the article, epidemiological studies showed that cot death was predominantly occurring in lower demographic social classes.[310]

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300 Peter S Blair and Peter J Fleming, 'Recurrence Risk of Sudden Infant Death Syndrome' (2008) 93 *Archives of Disease in Childhood* 269, 269.
301 Peter S Blair and Peter J Fleming, 'Recurrence Risk of Sudden Infant Death Syndrome' (2008) 93 *Archives of Disease in Childhood* 269, 270.
302 Peter S Blair and Peter J Fleming, 'Recurrence Risk of Sudden Infant Death Syndrome' (2008) 93 *Archives of Disease in Childhood* 269, 270.
303 The Lullaby Trust, *Report on 10,000 Babies using the CONI (Care of Next Infant) Programme* (September 2009) 2.
304 The Lullaby Trust, *Report on 10,000 Babies using the CONI (Care of Next Infant) Programme* (September 2009) 15.
305 The Lullaby Trust, *Report on 10,000 Babies using the CONI (Care of Next Infant) Programme* (September 2009) 16.
306 The Lullaby Trust, *Report on 10,000 Babies using the CONI (Care of Next Infant) Programme* (September 2009) 16.
307 The Lullaby Trust, *Report on 10,000 Babies using the CONI (Care of Next Infant) Programme* (September 2009) 16.
308 The Lullaby Trust, *Report on 10,000 Babies using the CONI (Care of Next Infant) Programme* (September 2009) 19.
309 Alison Waite, Angela McKenzie and Charlotte Daman-Willems, 'CONI: Confirmation of Continuing Relevance after 20 Years' (2011) 84 *Community Practitioner* 25, 25.
310 Alison Waite, Angela McKenzie and Charlotte Daman-Willems, 'CONI: Confirmation of Continuing Relevance after 20 Years' (2011) 84 *Community Practitioner* 25, 28, citing Peter S Blair et al, 'Major Epidemiology Changes in Sudden Infant Death Syndrome: A 20-Year Population-Based Study in UK' (2006) 367(9507) *Lancet* 314.

Eminoglu et al (2011)

221. Originating in Turkey, the authors discussed a one month old female referred to their clinic after three siblings had died of an unknown cause in the newborn period (two within 24 hours, a third at 14 days).[311] Metabolic investigations of the patient found results consistent with VLCAD, with two mutations in the VLCAD gene, one of which had never before been detected. The patient died suddenly at another hospital one month after the diagnosis. The authors discussed that progression of unrecognised VLCAD deficiency may be rapid and fatal secondary to cardiac involvement.[312]

Waite et al (2015)

222. This report considered the CONI PLUS program, established to support parents anxious because of SUDI in their extended family or an ALTE in their baby.

223. The study found that the number of SUDI deaths in CONI PLUS babies was higher than expected from United Kingdom averages.[313] It found that deaths in babies enrolled because of family history of SUDI were mostly associated with inappropriate sharing of a sleep surface at night and mostly outside the peak age range for sudden infant death.

224. Of 6,387 babies enrolled between 1996 and 2010, 37 (5.7/1,000) died in their first year. This showed a slightly raised mortality, but was not statistically significant.[314] Eighteen presented as SUDI, seven were attributed to SIDS, four were unascertained, four were due to infections, and three to other identified natural causes.[315]

225. Two thousand seven hundred and eighty-nine SUDI related babies were enrolled; six died suddenly and unexpectedly (2.15/1,000), however, four were sharing a bed or sofa at night with parent(s) who smoked or had consumed alcohol.

226. Of the 1,882 babies enrolled following an ALTE, five died suddenly and unexpectedly (2.66/1,000). Four were unexplained (one was due to infection). None were sharing a sleep surface and at least three died during the day.

227. While the number of these deaths was small, findings suggested a different mechanism for death in the two groups. Overall the number of deaths was slightly greater than expected; data suggested, however, that the population had a higher than average risk for SIDS.[316]

Child Death Review Team (2016)

228. In 2015, 42 infant deaths in NSW were classified as SUDI. SUDI was defined to apply to infants aged less than 12 months, with a death that is sudden and unexpected where the cause was not immediately apparent at the time of death.[317]

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311 Tuba F Eminoglu et al, 'Very Long-Chain Acyl CoA Dehydrogenase Deficiency which was Accepted as Infanticide' (2011) 210 *Forensic Science International* e1:1-3.

312 Tuba F Eminoglu et al, 'Very Long-Chain Acyl CoA Dehydrogenase Deficiency which was Accepted as Infanticide' (2011) 210 *Forensic Science International* e1:1-3, 1.

313 Alison J Waite et al, 'Mortality of Babies Enrolled in a Community-Based Support Programme: CONI PLUS (Care of Next Infant Plus)' (2015) 100(7) *Archives of Disease in Childhood* 637, 642.

314 Alison J Waite et al, 'Mortality of Babies Enrolled in a Community-Based Support Programme: CONI PLUS (Care of Next Infant Plus)' (2015) 100(7) *Archives of Disease in Childhood* 637, 639.

315 Alison J Waite et al, 'Mortality of Babies Enrolled in a Community-Based Support Programme: CONI PLUS (Care of Next Infant Plus)' (2015) 100(7) *Archives of Disease in Childhood* 637, 639.

316 Alison J Waite et al, 'Mortality of Babies Enrolled in a Community-Based Support Programme: CONI PLUS (Care of Next Infant Plus)' (2015) 100(7) *Archives of Disease in Childhood* 637, 642.

317 NSW Child Death Review Team, *Child Death Review Report 2015* (NSW Ombudsman, November 2016) 40.

229. SUDI has declined overall since 2001, the rate of 0.46 in 2015 being as low as it had been. The rate had not, however, changed significantly since 2008.[318] The 42 who died were aged from two days to seven months, with 27 aged between 28 days and three months. The cohort residing in areas of greatest socio-economic disadvantage had 22 deaths, more than twice that of other socio-economic cohorts.[319]

230. In 11 deaths a cause was unable to be determined.[320] All of them evinced more than one known risk factor for SIDS/SUDI.[321]

Milroy and Kepron (2017)

231. The authors referred to previous research that suggested a figure of two to 10 per cent of all SIDS cases were homicide (and other figures as high as 20-40 per cent), and re-evaluated these figures following Back to Sleep campaigns. The higher figures were, the authors argued, inconsistent with the fall in death rates following the Back to Sleep campaign. They found that current data suggested a figure much lower than 10 per cent of SIDS cases.[322]

232. The authors considered literature on the rate of covert homicide in SIDS, and also considered the related question of whether multiple deaths classified as SIDS are really homicides. In this context, they discussed high profile cases of women having been tried for multiple deaths of infants in their families, including the Folbigg case.

233. The authors concluded that the use of a percentage was problematic and of reduced value in view of the fallen rates of SUDI.[323] Literature suggested that initial figures were overestimates, but was clear that some SUDI deaths – a low percentage – are covert homicides.

234. The question of whether multiple cases of SIDS are "all homicides or could be from natural causes remains controversial".[324] The authors observed that different approaches to the problem may reflect jurisdictional differences, and also postulated that some medical examiners or forensic pathologists may have been influenced by a "think dirty" philosophy. This approach was rejected by the Goudge Inquiry: the correct approach is to be objective and "think truth".[325] Having regard to observations by appeal courts in the relevant high profile cases, evidence beyond simply the multiplicity of deaths appeared to now be a requirement before the deaths would be prosecuted.[326]

Christensen et al (2016)

235. This paper reported on an analysis of SIDS conducted by a large cohort of medical experts using the Utah Population Database ("UPDB").[327]

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318 NSW Child Death Review Team, *Child Death Review Report 2015* (NSW Ombudsman, November 2016) 40.

319 NSW Child Death Review Team, *Child Death Review Report 2015* (NSW Ombudsman, November 2016) 43.

320 NSW Child Death Review Team, *Child Death Review Report 2015* (NSW Ombudsman, November 2016) 45.

321 NSW Child Death Review Team, *Child Death Review Report 2015* (NSW Ombudsman, November 2016) 48.

322 Christopher M Milroy and Charis Kepron, 'Ten Percent of SIDS Cases are Murder – Or are they?' (2017) 7(2) *Academic Forensic Pathology* 163, 163.

323 Christopher M Milroy and Charis Kepron, 'Ten Percent of SIDS Cases are Murder – Or are they?' (2017) 7(2) *Academic Forensic Pathology* 163, 169.

324 Christopher M Milroy and Charis Kepron, 'Ten Percent of SIDS Cases are Murder – Or are they?' (2017) 7(2) *Academic Forensic Pathology* 163, 169.

325 Stephen T Goudge, Report of the Inquiry into Pediatric Forensic Pathology in Ontario (Ontario Ministry of the Attorney General, 1 October 2008).

326 Christopher M Milroy and Charis Kepron, 'Ten Percent of SIDS Cases are Murder – Or are they?' (2017) 7(2) *Academic Forensic Pathology* 163, 169.

327 Erik D Christensen et al, 'Sudden Infant Death "Syndrome" – Insights and Future Directions from a Utah Population Database Analysis' (2017) 173 *American Journal of Medical Genetics Part A* 177. This paper was addressed in Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 6, n 97.

236. The authors observed that it had been suggested that some five per cent of SIDS may be due to disorders of fatty acid oxidation and organic acidemias.[328] Channelopathy and cardiomyopathy variants were recently suggested as accounting for around one third of SUDI.[329] Thus, in view of the balance of cases, the diagnostic category SIDS remained a heterogeneous group of causal entities with common presentation and unknown recurrence risk.[330]

237. The authors conducted a search of the UPDB for death certificate diagnoses of SIDS and their family relationship. Information on the database was a valuable resource for studying the heritability of SIDS as it included population, pedigree, and selected clinical information (which included causes of death) for over eight million individuals. Even so, the authors emphasised that an accurate recurrence risk and heritability assessment in a given family required the specific diagnosis of a disease which may present as SIDS.

238. The authors used relationships among affected relatives to assess relative risk of SIDS recurrence by estimating the odds ratio for SIDS – that is, the odds that a relative of a SIDS death will die of SIDS compared with the odds of a control.[331] The analyses were performed on pre- and post-Back to Sleep campaign periods.

239. The following figure illustrates the changing incidence over the relevant periods

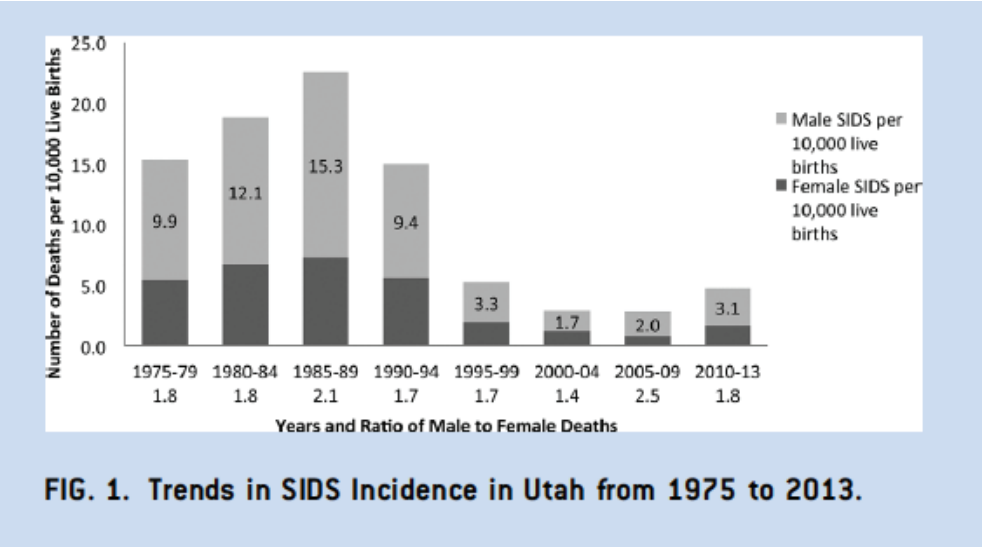

Figure 7 Trends in SIDS Incidence in Utah from Christensen et al (2016)

328 Erik D Christensen et al, 'Sudden Infant Death "Syndrome" – Insights and Future Directions from a Utah Population Database Analysis' (2017) 173 *American Journal of Medical Genetics Part A* 177, 178, citing M Bennett and P Rinaldo, 'The Metabolic Autopsy Comes of Age' (2001) 47 *Clinical Chemistry* 1145 and D H Chace et al, 'Electron Spray Tandem Mass Spectrometry for Analysis for Acylcarnitines in Dried Postmortem Blood Specimens Collected at Autopsy from Infants with Unexplained Cause of Death' (2001) 47 *Clinical Chemistry* 1166; Stephen T Goudge, Report of the Inquiry into Pediatric Forensic Pathology in Ontario (Ontario Ministry of the Attorney-General, 1 October 2008).

329 Erik D Christensen et al, 'Sudden Infant Death "Syndrome" – Insights and Future Directions from a Utah Population Database Analysis' (2017) 173 American Journal of Medical Genetics Part A 177, 178, citing C L Hertz et al, 'Genetic Investigations of Sudden Unexpected Deaths in Infancy Using Next-Generation Sequencing of 100 Genes Associated with Cardiac Genes' (2016) 24 *European Journal of Human Genetics* 817.

330 Erik D Christensen et al, 'Sudden Infant Death "Syndrome" – Insights and Future Directions from a Utah Population Database Analysis' (2017) 173 *American Journal of Medical Genetics Part A* 177, 178.

331 Erik D Christensen et al, 'Sudden Infant Death "Syndrome" – Insights and Future Directions from a Utah Population Database Analysis' (2017) 173 *American Journal of Medical Genetics Part A* 177, 178.

240. The incidence data mirrored national trends. The authors found incidence in the order of 2.8/10,000 after the Back to Sleep campaign, similar to recent worldwide ranges of 1-8/10,000.[332] The authors hypothesised that the residual post-Back to Sleep cases are "relatively enriched for underlying genetic causes of SIDS."[333]

241. Recurrence risks for SIDS were found to be modestly increased within families that had a prior SIDS, with a sibling odds ratio of 4.2, which was slightly more conservative than the five to six determined by Guntheroth, Lohmann and Spiers (1990) and Øyen, Skjaerven and Irgens (1996).[334] This was qualified by low numbers of cases occurring in large families, and exclusive reliance upon death certificates for diagnosis. A similar ratio (4.8) was found to apply to SIDS and other or unknown deaths to siblings at any age. This was about threefold higher than the overall (post- and pre-Back to Sleep) odds ratio of 2.95, which supported the notion that post-1995 SIDS cases are more likely to have a greater genetic contribution given the reduction in environmental factors.[335] This led to the observation that, given the role of genetics, true family-specific recurrence risk requires an accurate underlying diagnosis.

242. The authors commented that given that SIDS *rarely recurs* in families, most genetic analyses involve screening candidate genes in sporadic cases.[336] However, the full extent to which underlying genetic factors may interact with environmental factors remains to be determined.[337]

Duncan and Byard (2018)

243. Sibling deaths was discussed in Duncan and Byard (2018) in the following passage, set out in full with citations:

> *The association of SIDS deaths amongst siblings is still debated. There have been reports of an increase in the incidence of SIDS of between two and ten times in infants who have had a sibling or twin death, including an increase risk based on the presence of SIDS in second- and third-degree relatives (104, 105).*[338] *However, some of these outcomes have been explained once environmental and maternal factors have been controlled for and these families may only represent a small subgroup of individuals with increased vulnerability. There have also been reports of simultaneous sudden death in siblings supporting a genetic basis (106),*[339] *although the importance of environmental factors should be taken into consideration under these circumstances. In addition, a report by Diamond et. al. indicated five consecutive sibling deaths in the same family (107)*[340]

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332 Erik D Christensen et al, 'Sudden Infant Death "Syndrome" – Insights and Future Directions from a Utah Population Database Analysis' (2017) 173 *American Journal of Medical Genetics Part A* 177, 179, citing R Y Moon, R S Horne and F R Hauck, 'Sudden Infant Death Syndrome' (2007) 370 Lancet 1578 and B G Winkel et al, 'Sudden Unexpected Death in Infancy in Denmark' (2011) 45 *Scandinavian Cardiovascular Journal* 14.

333 Erik D Christensen et al, 'Sudden Infant Death "Syndrome" – Insights and Future Directions from a Utah Population Database Analysis' (2017) 173 *American Journal of Medical Genetics Part A* 177, 179.

334 Erik D Christensen et al, 'Sudden Infant Death "Syndrome" – Insights and Future Directions from a Utah Population Database Analysis' (2017) 173 *American Journal of Medical Genetics Part A* 177, 179.

335 Erik D Christensen et al, 'Sudden Infant Death "Syndrome" – Insights and Future Directions from a Utah Population Database Analysis' (2017) 173 *American Journal of Medical Genetics Part A* 177, 180.

336 Erik D Christensen et al, 'Sudden Infant Death "Syndrome" – Insights and Future Directions from a Utah Population Database Analysis' (2017) 173 *American Journal of Medical Genetics Part A* 177, 181.

337 Erik D Christensen et al, 'Sudden Infant Death "Syndrome" – Insights and Future Directions from a Utah Population Database Analysis' (2017) 173 *American Journal of Medical Genetics Part A* 177, 181.

338 (104): S Beal, 'Sudden Infant Death Syndrome in Twins' (1989) 84(6) Pediatrics 1038; (105): S M Beal and H K Blundell, 'Recurrence Incidence of Sudden Infant Death Syndrome' (1988) 63(8) *Archives of Disease in Childhood* 924.

339 (106): S Ladham et al, 'Simultaneous Sudden Infant Death Syndrome: A Case Report' (2001) 22(1) *American Journal of Forensic Medicine and Pathology* 33.

340 (107): Eugene F Diamond, 'Sudden Infant Death in Five Consecutive Siblings' (1986) 170(1) *Illinois Medical Journal* 33.

> *However, the authors feel that multiple deaths within the same family should raise concerns about other possible inherited conditions such as prolonged QT interval or metabolic disorders, homicide or potentially misclassified deaths of known cause. Thus, while multiple SIDS deaths in the one family may represent a genetic component in the etiology of SIDS, for 92% of families the risk of recurrence is considered small.*[341]

Recurrence of SIDS/unexplained deaths in previous cases

R v Clark

244. In 1999 lawyer Sally Clark was convicted in the Crown Court at Chester, England, of the murder of her infant sons in 1996 and 1998. Professor Sir Roy Meadow, Emeritus Professor of Paediatrics and Child Health at St James' University Hospital, had given evidence that the calculated risk of two infants dying of SIDS in the family by chance was "approximately a chance of 1 in 73 million."[342]

245. Ms Clark's appeal against conviction was dismissed in 2000. The Court of Appeal had considered whether the jury might have focussed on the figure given by Professor Meadow to the exclusion of the "real and compelling" evidence in the case. While the Court concluded that there was some substance to the criticism, in looking at all of the evidence there was an "overwhelming case" against Ms Clark.[343]

246. In 2002 the Criminal Cases Review Commission ("the CCRC") referred the case back to the Court of Appeal following the emergence of previously undiscovered results of microbiological testing that had been performed on one of the children. Expert opinion was that the results suggested one of the Clark children may have died from natural causes.[344]

247. Following referral, two grounds of appeal were raised for Ms Clark:

    a. first, that the failure to disclose the information contained in the microbiology reports meant important aspects of the case were never considered at trial; and

    b. secondly, that statistical information given to the jury about the likelihood of two sudden and unexpected deaths of infants from natural causes misled the jury and painted a picture that considerably overstated the rarity of two such events happening in the same family.[345]

248. The Court of Appeal found that the medical evidence that was not before the jury might have caused the jury to reach a different verdict in respect of that charge, and that if that child's death may have been from natural causes, it followed that no safe conclusion could be reached that the other child had died of unnatural causes. On that basis it quashed Ms Clark's convictions.[346]

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341 Exhibit D, Jhodie R Duncan and Roger W Byard, 'Sudden Infant Death Syndrome: An Overview' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 15, 26-27. This chapter was addressed in Exhibit J, Report of Professor Rosemary Horne (10 February 2019) p 6, n 100.

342 *R v Clark* [2000] EWCA Crim 54, [9].

343 *R v Clark* [2000] EWCA Crim 54, [256].

344 *R v Clark* [2003] EWCA Crim 1020.

345 *R v Clark* [2003] EWCA Crim 1020, [6].

346 *R v Clark* [2003] EWCA Crim 1020, [134]-[135].

249. However the Court also found the following in respect of the statistical evidence:

> *On the material before us, we think it very likely that it* [the figure of 1 in 73 million] *grossly overstates the chance of two sudden deaths within the same family from unexplained but natural causes… Quite what impact all this evidence will have had on the jury will never be known but… it may have had a major effect on their thinking notwithstanding the efforts of the trial judge to downplay it.*
>
> *The Court of Appeal on the last occasion would, it seems clear to us, have felt obliged to allow the appeal but for their assessment of the rest of the evidence as overwhelming. In reaching that conclusion the Court was as misled by the absence of the evidence of the microbiological results as were the jury before it. We are quite satisfied that if the evidence in its entirety, as it is now known, had been known to the Court it would never have concluded that the evidence pointed overwhelmingly to guilt.*
>
> *Thus it seems likely that if this matter had been fully argued before us we would, in all probability, have considered that the statistical evidence provided a quite distinct basis upon which the appeal had to be allowed.*[347]

R v Patel

250. In June 2003 Trupti Patel was tried and acquitted of the murder of her three children. There was evidence of a possible genetic explanation, with a history of unexplained infant deaths on both sides of the family. Also, significantly, one of the prosecution expert witnesses changed his mind partway through the trial about the likelihood of a baby's ribs being broken during resuscitation.[348]

R v Cannings

251. Ms Cannings had four children, three of whom died in infancy. She was charged with the murder of two children and the Crown adduced evidence that three of the children, including the daughter who had survived, had suffered ALTEs. The defence suggested that the deaths were natural, and should be classified as SIDS deaths. Ms Cannings was convicted of the two counts of murder.[349]

252. In 2004 the Court of Appeal quashed her convictions as a result of fresh evidence which fundamentally undermined the Crown case at trial. Medical evidence in the appeal demonstrated that the expert opinions at trial understated the frequency of:

    a. recurrent unexpected infant deaths from natural causes in a single family;

    b. finding a child dead or suffering an ALTE shortly after them appearing to be in good health; and

    c. the pattern of ALTEs preceding SIDS which was observed in Ms Cannings' children.

    Further evidence was also given by Ms Cannings' half-sister which raised a realistic possibility of a genetic problem within the family having caused the children's deaths.[350]

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347 *R v Clark* [2003] EWCA Crim 1020, [178]-[180].

348 *R v Patel* (Reading Crown Court, Jack J, 11 June 2004).

349 *R v Cannings* [2004] EWCA Crim 1, [2].

350 *R v Cannings* [2004] EWCA Crim 1, [34].

253. The Court confirmed that the exclusion of currently known natural causes of infant death does not establish that the death or deaths resulted from the deliberate infliction of harm: if on examination of all of the evidence every possible known cause has been excluded, the cause remains unknown.[351]

254. The Court observed:

> *We recognise that the occurrence of three sudden and unexpected infant deaths in the same family is very rare, or very rare indeed, and therefore demands an investigation into their causes. Nevertheless the fact that such deaths have occurred does not identify, let alone prescribe, the deliberate infliction of harm as the cause of death. Throughout the process great care must be taken not to allow the rarity of these sad events, standing on their own, to be subsumed into an assumption or virtual assumption that the dead infants were deliberately killed, or consciously or unconsciously to regard the inability of the defendant to produce some convincing explanation for these deaths as providing a measure of support for the prosecution's case. If on examination of all the evidence every possible known cause has been excluded, the cause remains unknown.*[352]

R v Anthony

255. In 2005 the CCRC referred the case of *R v Anthony* to the Court of Appeal. Donna Anthony had been convicted in 1998 of the murder of two of her infant children via smothering.[353]

256. Following the referral, the Court of Appeal allowed the appeal on the basis that "the balance of the medical evidence appears… to be much less unfavourable to Mrs Anthony than was the case at trial."[354] The Court also found that if this evidence and the judgment in *Cannings* had been available at trial, the evidence given by the experts would have taken a different route, the Judge would have summed up differently, and the jury verdict might have been different.[355]

257. However, the Court cautioned:

> *care must be taken not to transpose judicial comment on matters of evidence in the Cannings case into formal judicial precedent in a different case where the combined effect of the evidence, whether extraneous to or linked with or arising from the medical evidence, is different.*[356]

R v Kai-Whitewind and R v Mark

258. *R v Kai-Whitewind*[357] and *R v Mark*[358] are both 2005 cases from the English Court of Appeal where *Cannings* was distinguished.

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351 *R v Cannings* [2004] EWCA Crim 1, [13], [177].
352 *R v Cannings* [2004] EWCA Crim 1, [177].
353 *R v Anthony* [2005] EWCA Crim 952.
354 *R v Anthony* [2005] EWCA Crim 952, [96].
355 *R v Anthony* [2005] EWCA Crim 952, [97].
356 *R v Anthony* [2005] EWCA Crim 952, [81].
357 *R v Kai-Whitewind* [2005] 2 Cr App R 31.
358 *R v Mark* [2005] EWCA Crim 2257.

259. In *Kai-Whitewind* the Court held that while there had been conflicting expert opinion about the probable cause of death of Ms Kai-Whitewind's baby, unlike in *Cannings,* there had also been sufficient additional evidence before the jury to justify the verdict that she was guilty of his murder.[359]

260. In *Mark* the Court noted that the case was not one of conflicting medical evidence or one in which the evidence against Mr Mark was solely derived from the experts. Statistical figures given, including by Professor Meadow, were not relied upon as a statistical demonstration of the defendant's guilt and no direction over and above that given by the Judge was called for.

R v Matthey

261. In the Victorian case of *R v Matthey*,[360] Ms Matthey was charged with the murder of her four children. The charges were ultimately withdrawn by the prosecution following a decision by Coldrey J to exclude a number of pieces of evidence and limiting the evidence that could be given by medical experts:

> *Experts can point to the rarity of four unexpected and unexplained deaths in the one family on the basis of their experience and knowledge of the literature, but to utilise that factor in allocating a cause of death in an individual case is to indulge in "impermissible coincidence reasoning"… The rarity of the phenomenon of four unexpected and seemingly unexplained deaths in one family cannot, of itself, provide a cause of death.*[361]

Use of evidence of recurrence of sudden unexplained infant deaths

262. There are limits to the relevance of evidence given by experts and from appropriate sources of the recurrence of sudden unexplained infant deaths to a consideration of the Folbigg case. There are also limits to the weight that may appropriately be given to such evidence. These were issues that arose at trial and they are considered again here.

Expert awareness of other similar incidents

263. At trial the defence objected to this evidence being adduced from expert witnesses.[362] However, in his pre-trial determination of this issue, Wood CJ at CL decided that the experts could give evidence to the effect that SIDS deaths are a relatively infrequent event, and that SIDS deaths and/or multiple unexplained deaths or ALTEs involving infants within any one family are even more infrequent.[363] His Honour also saw no objection to the experts expressing an opinion, subject to "appropriate qualification", as to what was and what was not included in the medical literature regarding the improbable occurrence of multiple deaths of infants or of ALTEs within the same family of unexplained causes, that is, absent some common genetic disorder.[364]

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359 *R v Kai-Whitewind* [2005] 2 Cr App R 31, [90], [92].
360 *R v Matthey* [2007] VSC 398.
361 *R v Matthey* [2007] VSC 398, [188], [191].
362 *R v Folbigg* [2005] NSWCCA 23, [51].
363 *R v Folbigg* [2002] NSWSC 1127, [88].
364 *R v Folbigg* [2002] NSWSC 1127, [89]

264. Wood CJ at CL identified the relevance of this evidence as two of the pieces of circumstantial evidence relevant to the assessment of the probative value of the medical evidence, namely:

    a. the infrequent incidence of SIDS; and

    b. the rarity of repeat incidents of SIDS and of unexplained infant deaths or ALTEs within one family.[365]

    Each of these propositions were legitimate strands of the circumstantial case presented by the Crown.

265. The Court of Criminal Appeal upheld Wood CJ at CL's finding that the evidence was relevant.[366] The evidence of experts' knowledge of other similar cases was relevant to the Crown case that there was no reasonably possible natural cause of death and to infer that the deaths were caused in some unnatural way, namely, by deliberate suffocation.[367]

266. The findings of Wood CJ at CL on these points are of sound application for present purposes, even though rules of evidence do not apply in the Inquiry. Evidence of appropriately qualified experts and appropriate medical literature which describes the rarity or otherwise of SIDS and unexplained infant deaths may be given weight in the Inquiry.

Questions of weight

267. In 2004 in *Cannings*, the English Court of Appeal recognised that the occurrence of three sudden and unexpected infant deaths in the same family is very rare, or very rare indeed. The Court acknowledged that such a sequence of events demands an investigation but cautioned that the fact that the deaths have occurred "does not identify, let alone prescribe, the deliberate infliction of harm" as the cause of death.[368] In a statement with which I agree, the Court stated that:

> *Throughout the process, great care must be taken not to allow the rarity of these sad events, standing on their own, to be subsumed into an assumption or virtual assumption that the dead infants were deliberately killed.*[369]

Evidence that is not relevant, and limits as to weight

268. Wood CJ at CL did not permit expert opinion evidence which was reasoned only upon an exercise of statistical probability – such as one death equals SIDS, two equals unascertained and three equals homicide unless otherwise explained. It would not be appropriate for the likelihood of occurrence of multiple deaths to be expressed in the terms of statistical odds. This could give rise to a risk of those odds being misused in a way similar to the Crown prosecutor's fallacy in relation to DNA evidence.[370]

Counsel assisting's submissions on recurrence

269. Counsel assisting submitted that reasoning on the basis of Meadow's law, as was relied on by Professor Herdson in his report and Dr Beal on the voir dire, should not be accepted or adopted by the Inquiry.[371]

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365 *R v Folbigg* [2002] NSWSC 1127, [107].
366 *R v Folbigg* [2005] NSWCCA 23, [78]-[79].
367 *R v Folbigg* [2005] NSWCCA 23, [81].
368 *R v Cannings* [2004] EWCA Crim 1, [177].
369 *R v Cannings* [2004] EWCA Crim 1, [177].
370 *R v Folbigg* [2002] NSWSC 1127, [90].
371 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [141].

270. Counsel assisting also submitted that on the basis of the evidence given by the expert witnesses at trial, it would have been open to the jury to conclude that there had never been recorded a family where four children had died from natural causes. In fact, at the time of the trial there *had* been reported cases of three or more infants in the same family attributed to unidentified natural causes, or at least not established as attributable to unnatural causes.[372]

271. However, counsel assisting submitted that given:

    a. the weight of the evidence is that any increased risk of recurrence in a sibling is affected by genetic and environmental factors;

    b. there has been no genetic factor identified in the Folbigg family; and

    c. relevant environmental factors in respect of each child gave rise to a low risk of sudden unexplained infant death,[373]

    the observation by the trial judge that such events are not impossible and that they are rare reflected the knowledge as it stood then and remains the case today. Therefore, counsel assisting submitted there is no basis to assert there was a miscarriage of justice or irregularity that would give rise to a reasonable doubt as to Ms Folbigg's guilt.[374]

Ms Folbigg's submissions on recurrence

272. Ms Folbigg submitted that the Crown closing and summing-up at trial were wrong because there were recorded cases of three or more such deaths in the one family at the time of trial. Relying on *Mraz,* she said that such a submission can have an unknown but powerful effect on a jury that may not be capable of amelioration by a direction, and was likely to lead to a mistrial in this case.[375]

273. Putting the factual inaccuracy to one side, Ms Folbigg submitted that the submission regarding an absence of recorded cases was fundamentally unreliable because:

    a. the reporting of events is reliant upon a publisher deciding they warrant publication;

    b. there is no analysis to demonstrate what a "family" is for the purpose of the studies; and

    c. it presupposes that any family who has two children who die of unexplained death or SIDS proceeds to further attempts at having children.[376]

274. Ms Folbigg also submitted that the Crown introduced a syllogistic fallacy by suggesting that if there was no recorded case of three deaths in the one family, that must mean Ms Folbigg smothered her children.[377]

275. In reality, in Ms Folbigg's submission, there were alternatives, including that one or more children died of a genetic cause (as yet unknown), each child died of causes unrelated to the other, or that one or more of the children died of an exogenous stressor.[378]

276. Ms Folbigg also made lengthy submissions in respect of the relevance of previous cases.

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372 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [172].
373 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [173].
374 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [174].
375 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [199], [206], citing *Mraz v R* (1955) 93 CLR 493.
376 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [233].
377 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [208].
378 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [209].

277. She submitted that *Clark* was authority for the proposition that the Crown must establish each charge beyond reasonable doubt and that in the event that one charge fails the others must fall in circumstances where reasoning to the effect of "two deaths in the one family is suspicious" is deployed.[379]

278. Ms Folbigg referred to the judgment of Sully J on appeal which distinguished the Folbigg case from *Cannings* on the basis that in the latter case there was "no suggestion of ill-temper, inappropriate behaviour, ill treatment let alone violence, at any time, with any one of the four children", which could be contrasted with Ms Folbigg's diary entries.[380] Ms Folbigg said that Sully J did not consider the absence of prior abuse, evidence of smothering, ill temper at the time of Caleb's birth and death and the care and attention she afforded to all of her children.[381] She submitted that this should be considered in light of the observation in *Cannings* that:

> *if however the deaths were natural, virtually anything done by the mother on discovering such shattering and repeated disasters would be readily understandable as personal manifestations of profound natural shock and grief*.[382]

279. Ms Folbigg also sought to draw an analogy with the Chamberlain case where the High Court found that the Crown needed to prove its case that Ms Chamberlain murdered her baby and could not simply assert that if a dingo did not take her baby, she must have murdered it.[383] Ms Folbigg submitted that this line of reasoning introduced a high risk that the jury would deal with all of the charges together and fail to satisfy themselves beyond reasonable doubt with respect to each charge.[384]

Findings

280. As seen from the review of literature above, while Duncan and Byard (2018) do not refer to the most recent literature available on the topic of recurrence, nonetheless the evaluative description is consistent with, although perhaps somewhat understated compared with, more recent descriptors of risk. That is, the risk of recurrence is considered small.[385]

281. Professor Cordner's oral evidence on this issue reflected the substance of the discrepancies between experts in the literature between about 2005 and 2008 on issues of recurrence set out in the review of literature above. It is, however, not the case that the overall point from research, as stated by Professor Cordner, is that more than half of subsequent infant deaths in families are natural.[386] The literature makes plain that the issues are much more complex and nuanced, which are not fairly represented by such a statement. In any event, there is no emerging weight of consensus which would support Professor Cordner's statement as to there being an "overall point". Counsel assisting submitted that for these reasons, that statement ought not be accepted.[387] I accept that submission.

282. It is accepted that it is clear from the work of the Inquiry that before 2003 there had been reported cases involving the deaths of three or more infants in the same family attributed to unidentified natural causes, or at least not established as attributable to unnatural causes. To the extent that the Crown case as left to the jury asserted or invited otherwise, that was incorrect.

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379 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [218]-[219].
380 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [224]-[225].
381 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [229].
382 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [229], citing *R v Cannings* [2004] EWCA Crim 1, [11].
383 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [212], citing *Chamberlain v The Queen (No 2)* (1984) 153 CLR 521.
384 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [213].
385 Exhibit D, Jhodie R Duncan and Roger W Byard, 'Sudden Infant Death Syndrome: An Overview' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 15, 27.
386 Exhibit Q, Report of Professor Stephen Cordner (undated) p 34.
387 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [148]-[149].

283. However, the current descriptions in literature and in evidence by experts emphasise the low nature or rarity of recurrence risk, a point which was accurately reflected in the directions of the trial judge. It is also clear that the mere fact of the recurrence of the sudden unexpected death of an infant cannot, by itself, prove beyond a reasonable doubt that the cause was homicide, even in the case of multiple deaths. That is so because our current understanding of the causes of such natural deaths cannot be said to be complete. It is also not helpful to attempt to ascribe a statistical value to the possibility of a recurrence of such an event.

284. I do not accept Ms Folbigg's submission that an analogy can be drawn with *Mraz*, a case where the trial judge misdirected the jury that they could find the accused guilty of manslaughter in circumstances where there should have been a finding of murder or an acquittal. This misdirection created a "fundamental confusion in the minds of the jury as to what constitutes the crime".[388]

285. I note also that the decision of the Court of Criminal Appeal in the present case in 2005 referred to the decision of *Cannings* in the English Court of Appeal in 2004 in which three infant deaths and an ALTE were considered.[389] The Court of Criminal Appeal was thus aware of the existence of such a case when there had been three deaths in the one family but held that the Crown prosecutor's submission that such a case had never been heard of was simply a piece of circumstantial evidence to be added to the other circumstances. I agree with that description. A review of the literature indicates that repeat SIDS deaths are rare.

286. In light of the above, I am satisfied that the treatment of the issue of recurrence at trial has not resulted in a miscarriage of justice or irregularity that gives rise to a reasonable doubt as to Ms Folbigg's guilt.

388 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [199], [206], citing *Mraz v R* (1955) 93 CLR 493, 515.

389 *R v Folbigg* [2005] NSWCCA 23, [117], [133]-[144], citing *R v Cannings* [2004] EWCA Crim 1.

Report of the Inquiry into the convictions of Kathleen Megan Folbigg

July 2019

Volume 1: Chapter 5

Chapter 5: Medical Evidence

Introduction

1. This chapter sets out evidence received in the Inquiry of a medical nature, including expert medical evidence and evidence of treating practitioners, and submissions and findings on medical evidence, concerning the death of each child and of Patrick's ALTE.

2. The volume of medical material received in the Inquiry was significant. It includes relevant clinical records, medical reports both expert and treating available at the time of trial, oral evidence given by medical experts throughout the curial proceedings, medical reports prepared for the purposes of the Inquiry, and oral evidence given by medical experts in the public hearings of the Inquiry.

3. As such, I had available to me a range of medical evidence which exceeds what was before the jury. As observed elsewhere, the Inquiry was not constrained by the rules of evidence.

4. Ambulance and autopsy (and some related histology) reports were tendered as exhibits in the trial and were provided to the jury. Some reports and statements prepared at the request of the Crown and the defence containing expert opinions and accounts were not tendered as exhibits and were not provided to the jury. Further, medical experts gave a significant amount of evidence in the absence of the jury, during the course of arguments about admissibility of aspects of their evidence. The expert opinions and accounts of treating doctors which were adduced in oral evidence at the trial, and which the jury heard, were more confined.

5. I set out in the previous chapter the risk factors for SIDS and the evidence received in the Inquiry as to their meaning and application. In this chapter, I set out evidence received in the Inquiry in relation to how those risk factors applied to the circumstances of each Folbigg child.

Caleb

Caleb's birth and medical history

6. Caleb was 3,280 grams and not underweight when he was born on 1 February 1989, full-term at 40 weeks.[1] At discharge from hospital he was recorded as being artificially fed.[2]

7. Dr Barry Springthorpe, consultant paediatrician, saw Caleb twice, first on 2 February 1989 when Caleb was 14 hours old.[3] He gave evidence at trial that when he first examined Caleb, he had developed respiratory distress which required some oxygen through the night.[4] This "stridor", or noisy breathing, was a common occurrence in newborn children.[5] A chest x-ray was clear, and Caleb's condition improved over the next two days.[6]

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1 Exhibit H, Forensic pathology tender bundle, Neonatal record of Caleb (5 February 1989).
2 Exhibit H, Forensic pathology tender bundle, Neonatal record of Caleb (5 February 1989).
3 7 April 2003 T265.22-28, T266.6-8, T269.25-28.
4 7 April 2003 T265.30-41.
5 Exhibit H, Forensic pathology tender bundle, Statement of Dr Barry Springthorpe (6 December 1999) [5]; 7 April 2003 T265.43-44.
6 Exhibit H, Forensic pathology tender bundle, Statement of Dr Barry Springthorpe (6 December 1999) [5].

8. At the second examination on 17 February 1989, Caleb still had a "mild inspiratory stridor with a little recession which was most marked when he was upset or lying flat on his back".[7] In his oral evidence at trial, Dr Springthorpe said that the stridor was "very, very mild".[8] He diagnosed laryngomalacia, or a "floppy larynx", meaning that the cartilage in the larynx was soft and could collapse on inspiration.[9] It was not apparent when Caleb was at rest but would have been exacerbated when Caleb was unsettled.[10] Dr Springthorpe was informed by Mr and Ms Folbigg that it did not interfere with Caleb's feeding or sleeping, although in a letter noted that it caused some feeding difficulties.[11]

Caleb's death

9. Caleb died on 20 February 1989 aged 19 days old.[12] He was found on his back with his face uncovered in his own bed,[13] in his own room.[14] Mr Folbigg gave evidence at trial that he was woken by Ms Folbigg screaming. He went in and Ms Folbigg was standing at the end of the bassinet screaming.[15] Caleb was in the bassinet, and Mr Folbigg lifted him.[16] Caleb's lips were blue, his eyes were closed and his skin was warm to touch.[17] He was not breathing.[18] Mr Folbigg attempted to resuscitate him and told Ms Folbigg to call an ambulance.[19]

10. In a statement to police, Ms Folbigg stated that she fed Caleb at 1:00am then put him to bed.[20] She recorded in a diary entry that, at about 2:00am, Caleb was "finally asleep!!".[21] At about 2:50am, she checked him and found him cold.[22]

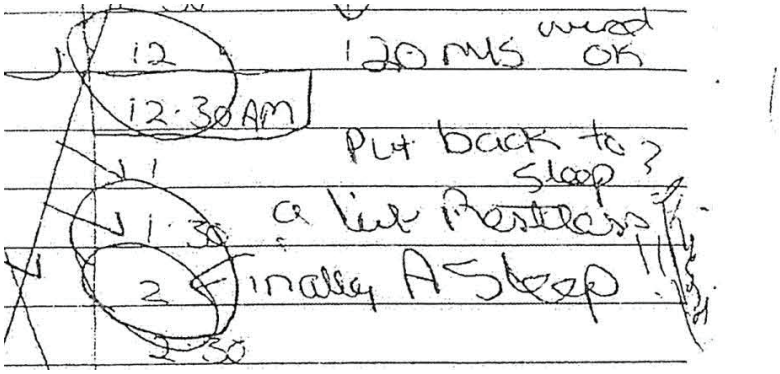

Figure 1: Diary entry 19 February 1989: Exhibit AZ in the Inquiry, p 19

7 Exhibit H, Forensic pathology tender bundle, Statement of Dr Barry Springthorpe (6 December 1999) [6]; 7 April 2003 T268.46-49, T269.44-270.2.
8 7 April 2003 T266.20.
9 7 April 2003 T266.34-39.
10 7 April 2003 T270.37-41.
11 Exhibit H, Forensic pathology tender bundle, Letter from Dr Barry Springthorpe to Dr Dorothy Leeder (21 March 1989); 2 April 2003 T101.38-102.18; 7 April 2003 T266.46-51.
12 Exhibit H, Forensic pathology tender bundle, Report of death to Coroner (20 February 1989).
13 2 April 2003 T104.52-55
14 2 April 2003 T104.5.
15 2 April 2003 T104.1-6.
16 2 April 2003 T104.11-15.
17 2 April 2003 T104.23.34.
18 2 April 2003 T104.37.
19 2 April 2003 T104.40-46.
20 Exhibit E, trial Exhibit AK, Statement of Kathleen Folbigg (undated).
21 Exhibit AZ, Diaries tender bundle, p 19.
22 Exhibit E, trial Exhibit AK, Statement of Kathleen Folbigg (undated).

11. Mr Dave Hopkins and Mr Richard Baines were the first ambulance officers to attend. Mr Hopkins stated that at 2:59am he and Mr Baines arrived at the house.[23] They either went into a room and gathered Caleb, or the woman present (Ms Folbigg) brought Caleb to them. Caleb was dressed in light clothing.[24] Mr Hopkins removed the upper clothing and established that Caleb was in a state of cardiac arrest, i.e. unconscious, was not breathing and was pulseless. He noted that Caleb was warm to touch and pale around the mouth and lips. Mr Baines also stated that Caleb was pale, not breathing and warm to touch upon examination.[25]

12. Mr Hopkins recorded that Caleb's airway was obstructed, although when he gave his statement in 1999 he could not remember exactly what obstructed it.[26] He assumed there may have been saliva or fluid in the airway, which needed to be cleared, which Mr Baines did, prior to inserting a Guedel's airway (a device to keep the airway open).[27]

13. A short time later, two more ambulance officers, Mr Allen Reed and Mr Ron Doherty, arrived. In his statement, Mr Reed said that on arrival at 3:03am either he or Mr Doherty applied an ECG monitor to Caleb which recorded asystole (meaning deceased).[28] Mr Reed stated that the external examination showed that Caleb had blue buccal mucosa which was a blue/purple colouring around the outside of the mouth.[29] In an ambulance report, Mr Reed recorded the skin temperature as cold to touch and the airway clear, and no breathing or circulation present.[30] There was no sweating, vomiting, fitting, burns or blood loss. At trial, Mr Reed had no independent recollection of the incident (after 14 years) and relied on his statement.[31] Nothing was said in his statement as to how long after his arrival he might have touched Caleb.

14. In the ambulance report signed by Mr Baines and Mr Hopkins, the history was recorded as being SIDS.[32] The report recorded that Caleb was pale, warm to touch but not breathing. His airway was recorded as obstructed, breathing absent, circulation absent, and skin temperature as normal, on a series of check boxes. His posture was recorded as supine.

15. The "Report of death to Coroner" (known as a "P79A") was dated 20 February 1989 and was signed by Senior Constable K J Bryant. It recorded the time that Caleb was last seen alive as being 1:00am on 20 February 1989.[33]

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23 Exhibit H, Forensic pathology tender bundle, Statement of David Hopkins (1 October 1999) p 2; Exhibit H, Forensic pathology tender bundle, Statement of Richard Baines (29 October 1999) p 2; 3 April 2003 T141.51-52 (Hopkins); T148.31-34 (Baines).

24 3 April 2003 T142.12-20.

25 Exhibit H, Forensic pathology tender bundle, Statement of Richard Baines (29 October 1999) p 2; 3 April 2003 T148.31-34.

26 Exhibit H, Forensic pathology tender bundle, Statement of David Hopkins (1 October 1999) p 2; 3 April 2003 T142.31-33.

27 Exhibit H, Forensic pathology tender bundle, Statement of David Hopkins (1 October 1999) p 2; 3 April 2003 T142.32-35.

28 Exhibit H, Forensic pathology tender bundle, Statement of Allen Reed (1 September 1999) [5].

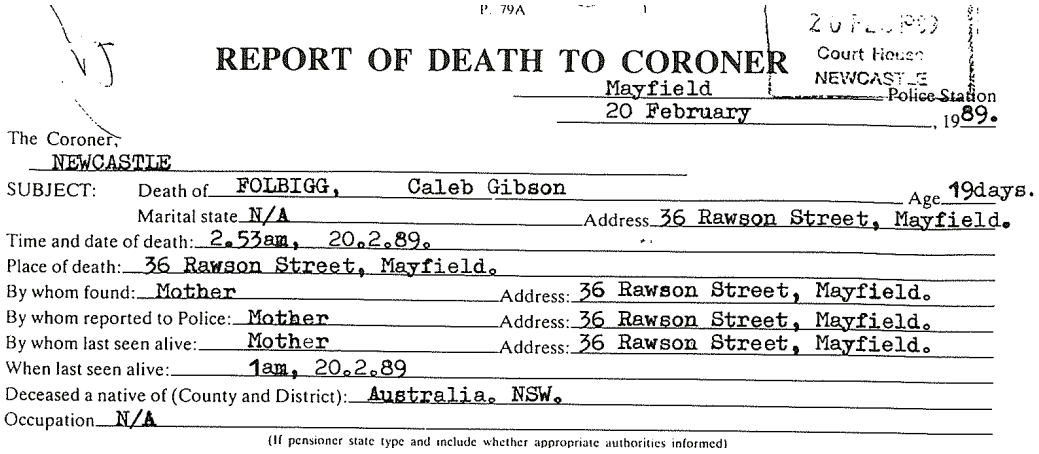
29 Exhibit H, Forensic pathology tender bundle, Statement of Allen Reed (1 September 1999) [5].

30 Exhibit H, Forensic pathology tender bundle, Ambulance report Q006 (20 February 1989).

31 5 May 2003 T1152.46-52.

32 Exhibit H, Forensic pathology tender bundle, Ambulance report Q005 (20 February 1989).

33 Exhibit H, Forensic pathology tender bundle, Report of death to Coroner (20 February 1989).



P. 79A

REPORT OF DEATH TO CORONER

Court House NEWCASTLE Police Station

Mayfield

20 February 1989.

The Coroner,
NEWCASTLE

SUBJECT: Death of FOLBIGG, Caleb Gibson Age 19days.
Marital state N/A Address 36 Rawson Street, Mayfield.
Time and date of death: 2.53am, 20.2.89.
Place of death: 36 Rawson Street, Mayfield.
By whom found: Mother Address: 36 Rawson Street, Mayfield.
By whom reported to Police: Mother Address: 36 Rawson Street, Mayfield.
By whom last seen alive: Mother Address: 36 Rawson Street, Mayfield.
When last seen alive: 1am, 20.2.89
Deceased a native of (County and District): Australia. NSW.
Occupation N/A
(If pensioner state type and include whether appropriate authorities informed)

Figure 2: Extract of Report of Death to the Coroner (P79A) dated 20 February 1989: Exhibit H in the Inquiry, p 4

Autopsy

16. In an interim autopsy report dated 20 February 1989, Dr Royal Cummings, pathologist, gave SIDS as the provisional cause of death.[34]

17. Dr Cummings also gave SIDS as the cause of death in his final autopsy report dated 9 May 1989. His examination of Caleb was recorded as having taken place at 11:45am on 20 February 1989.[35] There were no external signs of injury and Caleb appeared to have been well cared for. A toxicology report was negative.

18. On 20 February 1989, Ms Folbigg called Dr Springthorpe and told him Caleb had been found dead in his cot.[36] Dr Springthorpe asked Dr Cummings to check for any cysts or webs which can cause noisy breathing.[37] Dr Cummings did not identify evidence of these.[38]

19. In 1999, when reviewing the deaths of all four children, Dr Allan Cala, forensic pathologist, conducted a microscopic examination of tissues. All of these were reported to be normal, except for Caleb's lungs, with the report stating that "congestive changes are present with focal areas of haemorrhage present within some alveolar spaces."[39]

[34] Exhibit H, Forensic pathology tender bundle, Interim post-mortem report of Caleb (20 February 1989); Final autopsy report of Caleb (9 May 1989).
[35] Exhibit H, Forensic pathology tender bundle, Final autopsy report of Caleb (9 May 1989).
[36] Exhibit H, Forensic pathology tender bundle, Statement of Dr Barry Springthorpe (6 December 1999) [7].
[37] 7 April 2003 T267.31-46.
[38] 7 April 2003 T267.42-46; Exhibit H, Forensic pathology tender bundle, Final autopsy report of Caleb (20 February 1989).
[39] Exhibit H, Forensic pathology tender bundle, Microscopic examination of tissues by Dr Allan Cala (25 November 1999).

HISTOLOGY

HEART	Normal
LUNGS	Are congested and in places show incomplete aeration, in other sections their alveoli contain extravasated red blood cells and a small amount of eosinophillic exudate.
LIVER	Normal.
KIDNEY	Normal.
SPLEEN	Normal.
THYMUS	Normal.
ADRENAL GLAND	Normal.
LYMPH NODES	Normal.

SUMMARY OF AUTOPSY FINDINGS
Both lungs were moderately moist
No other unusual features were seen

R. CUMMINGS MD, FRCPA
Regional Forensic Pathologist
May 9, 1989

... contd. page 3

Figure 3: Extract from final autopsy report dated 9 May 1989 re Caleb: Exhibit H in the Inquiry, p 10

Death certificate

20. The death certificate recorded "Sudden Infant Death Syndrome" as the cause of Caleb's death.[40]

Evidence at the time of trial

Stridor and laryngomalacia

21. Dr Springthorpe recorded in his statement and gave evidence before the jury that he believed that stridor had nothing to do with Caleb's death.[41] In crossexamination, he said that while the "floppy larynx" condition can cause total obstruction of the airway, in Caleb's case it had been ruled out; otherwise, Caleb would not have been discharged.[42] Dr Springthorpe agreed that the younger the baby the more vulnerable to upper airways obstruction, and that airways can collapse without cysts or webs.[43]

22. Dr Allan Cala gave evidence at trial that he had never heard of a child who had died of a floppy larynx.[44]

40 Exhibit H, Forensic pathology tender bundle, Death certificate of Caleb (1 March 1989).
41 7 April 2003 T267.48-50; Exhibit H, Forensic pathology tender bundle, Statement of Dr Barry Springthorpe (6 December 1999) p3.
42 7 April 2003 T270.53-271.17.
43 7 April 2003 T271.32-52.
44 16 April 2003 T746.41.

23. Dr Susan Beal, a paediatrician and epidemiologist with particular SIDS expertise, considered that in isolation Caleb's floppy larynx would be "[m]ost unlikely" to have played a role in his death.[45] She had never heard of a death from a floppy larynx.[46] Laryngomalacia is normally compatible with life, but can be life threatening – Dr Beal knew of children who would have died without treatment from severe laryngomalacia.[47]

24. Professor Peter Herdson, a consultant forensic pathologist based in the Australian Capital Territory, gave evidence that he had never heard of a child who had died from floppy larynx.[48] He said that "floppy larynx" refers to less cartilage in the larynx than normal, and did not indicate inflammation or infection which could obstruct breathing.[49] In his recorded answers to the "model questions",[50] Professor Herdson stated he did "not believe in a floppy larynx".

25. Professor Peter Berry, a retired consultant paediatric pathologist at the Bristol Royal Hospital for Sick Children, stated in his report of November 2000 that there was nothing to suggest that a floppy larynx was responsible for Caleb's death, a floppy larynx being generally a benign and self-limiting condition.[51] Although "near miss" SIDS events have been attributed to the condition in medical literature, it was not a recognised cause of death.[52] In answers to the model questions, Professor Berry also said that a floppy larynx was a very common occurrence. He had once come across a case of a child with a floppy larynx who died, but in his opinion the floppy larynx did not cause the death.[53]

26. Professor Roger Byard, specialist forensic pathologist with expertise in sudden natural death in infancy and early childhood at the Forensic Science Centre in Adelaide, included Caleb's history of breathing problems with a floppy larynx as a reason for giving "undetermined" (not excluding SIDS) for Caleb's death (below at [51]).[54] In his report, Professor Byard observed there was no histologic examination of the larynx (it not being a routine examination) and so it was uncertain whether there were any structural abnormalities of cartilage present.[55] He was aware of a small number of cases in which infants with laryngomalacia had suffered collapse or reportedly died with a similar condition.[56]

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45 5 May 2003 T1137.30.

46 Responses to Crown model questions – Dr Susan Beal, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.

47 5 May 2003 T1138.5-8.

48 1 May 2003 T1033.55-1034.30.

49 1 May 2003 T1034.20-27.

50 On 22 April 2003 the Crown provided Ms Folbigg's solicitor with a set of model questions and answers from Professors Herdson and Berry and Dr Beal. They were each asked to respond to the questions asked of Dr Cala at trial and their responses were recorded in writing in a standardised format. The model questions and answers were tendered at trial on the voir dire of Dr Beal and in this Inquiry as Annexure C to the 5 March 2019 report of Professor Blackwell (Exhibit T). The set of questions asked each expert, inter alia, what they would diagnose as each child's cause of death individually, what their causes of death were considered together, whether the deaths were consistent with deliberate smothering, and whether there was any natural cause of death that could account for all four deaths.

51 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) pp 23-23; 1 May 2003 T1056.55-1057.35.

52 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) pp 23-24; 1 May 2003 T1056.58-1057.13.

53 Responses to Crown model questions – Professor Berry, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.

54 7 May 2003 T1203.25-1205.39, T1206.7-15, T1234.15-38, T1254.25-32; Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 9.

55 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 9; 7 May 2003 T1203.33-47.

56 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 9; 7 May 2003 T1204.18-25.

27. Professor Anthony Busuttil, professor of forensic medicine and clinical forensic examiner, stated in his report that it was "debateable" whether laryngomalacia would result in episodes of upper airways obstruction given the paediatrician noted the presence of stridor.[57] Imposed airways obstruction could not completely be excluded.[58]

Haemosiderin

28. Before the trial, Professor Berry was provided with 14 stained microscope slides of tissue taken from Caleb's body.[59] He said that some time subsequent to the death, sections of lung had been stained by Perls' method for ferric iron.[60] It was believed that some children, who experience a period of complete occlusion of the airways and recover, bleed into their lungs.[61] Over a period of 36 to 48 hours the blood is converted into haemosiderin, which stains blue via Perls' method.[62] One of the explanations for a positive Perls' stain is that there may have been an episode of previous asphyxia, whatever the cause.[63]

29. The Inquiry sought to determine the origins of the slides provided to Professor Berry, and whether they could be re-examined. The Inquiry was, however, informed that the slides are not now available. When and by whom they were stained is not known.

30. Professor Berry stated in his report that haemosiderin indicated previous haemorrhage into the lungs.[64] In his oral evidence, he referred to the significant amount of haemosiderin in Caleb's lungs, including in air spaces which, he said, was linked to (although not proof of) suffocation.[65] He said that the mere finding of the haemosiderin did not make the death suspicious, but when one had eliminated causes, then it did raise suspicion – he described it as "really a screening test, I think, for picking out problematic cases."[66]

31. Professor Berry said in evidence that haemosiderin would prompt investigation into the possibility of a previous episode of suffocation.[67] In his report, Professor Berry noted that the haemosiderin, together with the diary entry by Ms Folbigg that at 2:00am Caleb was "finally asleep!!",[68] would lead him to suspect suffocation and recommend a full police investigation.[69]

32. In his first report, Professor Byard noted the scattered iron-containing macrophages (cells involved in the detection and destruction of harmful organisms) in lung sections found by Professor Berry.[70] He gave evidence before the jury of a study he had done in which he found haemosiderin in around 20 per cent of SIDS babies' lungs, saying that it just meant that something had happened with bleeding in the past, which could be suffocation or could, for example, be a nose bleed.[71]

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57 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 9.
58 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 9.
59 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 21; 1 May 2003 T1055.27-31.
60 1 May 2003 T1058.32-36.
61 1 May 2003 T1057.50-57.
62 1 May 2003 T1057.57-1058.2.
63 1 May 2003 T1058.2-4.
64 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 24; 1 May 2003 T1057.50-1060.23.
65 1 May 2003 T1058.54-1059.4, T1070.6-19.
66 1 May 2003 T1069.11-14, T1069.54-58.
67 1 May 2003 T1059.44-57; T1060.10-14.
68 Exhibit AZ, Diaries tender bundle, p 19.
69 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) pp 3, 24; 1 May 2003 T1060.10-14.
70 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 9.
71 7 May 2003 T1208.5-35.

No one knows how much iron would get into the lungs from child birth; any inhalation of blood into lungs could cause the presence of iron.[72] This view is also expressed in Duncan and Byard (2018).[73] Professor Byard also said that one of the most common causes of bleeding from *within* the lungs is an asphyxiating event of some sort, and it possibly tended to indicate a previous episode of this type in Caleb.[74]

33. Professor Busuttil stated that the presence of haemosiderin within the lungs raised the question of imposed upper airways obstruction; haemosiderin may appear within days of the obstruction.[75]

SIDS and difficulty differentiating from smothering

34. Dr Springthorpe gave evidence that it is possible to smother a young baby with a pillow and leave no external signs and that a pillow or a hand over the face could have caused Caleb's death without leaving any marks.[76] He said that a pillow over the face "could certainly cause this child's death and not leave any marks whatsoever."[77] It was also possible that a hand could have smothered Caleb without leaving marks, given Caleb's age.[78]

35. Dr Beal, paediatrician and epidemiologist, gave evidence that she would have diagnosed Caleb's death, on its own, as SIDS with the proviso that he was under three weeks of age – the fact that he was supine made SIDS unlikely.[79]

36. Dr Janice Ophoven, a paediatric forensic pathologist based in Minnesota, USA, did not give evidence at trial and her report was not tendered as an exhibit. She cited an absence of commonly recognised risk factors as being one of the reasons for her conclusions that Caleb did not die of SIDS.[80] She discussed the SIDS diagnosis as having been applied variably over the last half century, and that some deaths previously diagnosed as SIDS were now known to be homicides.[81] Dr Ophoven stated that it was common practice to reconsider deaths of previous children in a family, and this applied in Caleb's case.[82]

37. Professor Herdson said that SIDS was uncommon, but not unheard of, in a baby as young as Caleb.[83] It was virtually impossible, there being virtually nothing to find, to differentiate between SIDS and suffocation at post-mortem.[84] He found it extremely difficult to differentiate between SIDS and suffocation based on the presence or absence of petechiae (pinpoint haemorrhages).[85]

38. Professor Berry opined both in his report and in evidence that he would exclude SIDS because of the presence of haemosiderin in alveolar spaces in Caleb's lungs, which he stated was very unusual in infant deaths.[86]

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72 7 May 2003 T1208.26-35.

73 Exhibit D, Roger W Byard, 'The Autopsy and Pathology of Sudden Infant Death Syndrome' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 497, 504-505.

74 7 May 2003 T1235.13-24.

75 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) pp 8-9.

76 7 April 2003 T267.56-268.11.

77 7 April 2003 T268.3-5.

78 7 April 2003 T268.7-11.

79 5 May 2003 T1138.21-40.

80 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 3.

81 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 4.

82 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 4.

83 1 May 2003 T1034.37-45, T1042.13-24.

84 1 May 2003 T1034.47-1035.2; Responses to Crown model questions – Professor Herdson, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.

85 1 May 2003 T1035.4-14. Petechiae are pinpoint haemorrhages: Transcript of the Inquiry, 19 March 2019 T81.37-40.

86 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 24; 1 May 2003 T1068.17-22, T1070.7-19.

39. Professor Byard gave evidence at trial that autopsy findings were the same for suffocation as for SIDS (and so there was no finding that amounted to proof that Caleb was suffocated).[87]

40. Professor Busuttil, forensic pathologist, stated in his report that SIDS was rather unusual at Caleb's age – it should not have been attributed to SIDS.[88]

Opinions on cause of Caleb's death

41. Dr Cala gave evidence at trial that he would have given the cause of Caleb's death as "undetermined"; the findings on Caleb's post-mortem examination were consistent with Caleb having been deliberately smothered.[89] Dr Cala believed it was "likely" that Caleb died from such an event.[90] The lack of positive finding of suffocation did not exclude it, because there are generally no positive signs of suffocation.[91] In correspondence to Detective Senior Constable Ryan before the trial, which was not before the jury, Dr Cala noted that upon post-mortem examination, no significant disease processes were found to explain the death.

42. Dr Beal agreed with the proposition that findings in relation to Caleb were consistent with him having been deliberately smothered.[92] Under crossexamination, she agreed that age was not determinative, and did not exclude SIDS; similarly as to Caleb having been found on his back.[93] In isolation, Dr Beal said she would have looked to see whether Caleb had "a minor congenital heart lesion" to explain his death. She believed that findings on Caleb's postmortem were consistent with him having been deliberately suffocated. If he did not have a heart lesion, a catastrophic asphyxiating event was "most likely".

43. Dr Ophoven opined in her report, "to a reasonable degree of medical certainty", that Caleb did not die of SIDS, that his death was most consistent with suffocation, and that it was probably homicidal assault.[94] She opined that the cause and manner of his death should be listed as undetermined.[95] The findings at autopsy that Dr Ophoven considered to be consistent with suffocation were the absence of thymic petechiae, presence of extensive pulmonary haemorrhage and the blood that Ms Folbigg described on Caleb's face.[96]

44. Professor Herdson gave evidence that the findings on post-mortem were consistent with deliberate suffocation; there being no pathological findings, he could not distinguish between deliberate or accidental.[97] However, Professor Herdson was "quite sure" that Caleb died from a sudden catastrophic event of unknown causes.[98] In his report dated 17 January 2002, Professor Herdson opined that the findings in relation to Caleb left the cause of death undetermined, but apparently consistent with SIDS.[99] He concurred with analyses by Professor Berry and Dr Ophoven.[100]

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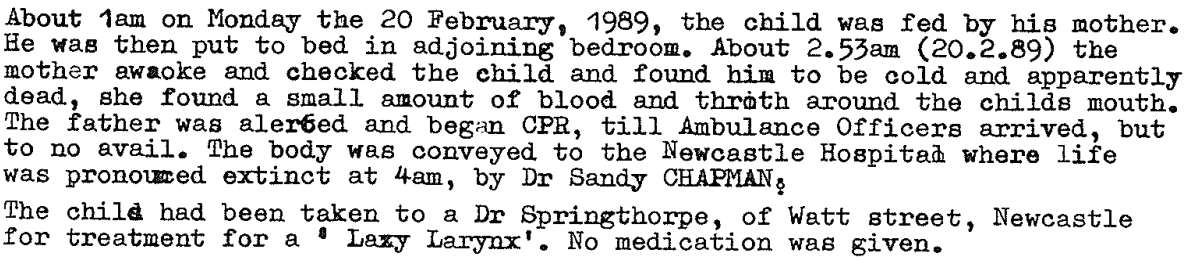
87 7 May 2003 T1205.41-1206.15.
88 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) pp 8-9.
89 16 April 2003 T746.48-53.
90 16 April 2003 T746.55-57.
91 16 April 2003 T752.23-28.
92 5 May 2003 T1138.42-48
93 5 May 2003 T1146.11-29.
94 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 3.
95 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 3.
96 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 3.
97 1 May 2003 T1035.16-21, T1042.26-39.
98 1 May 2003 T1035.23-27.
99 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Herdson (17 January 2002) p 2.
100 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Herdson (17 January 2002) p 2.

45. In his answers to the model questions, Professor Herdson said he would have diagnosed Caleb's death as SIDS or undetermined, although had never used "undetermined" in isolation, because it is so tough on relatives and police. As to whether Caleb had died from a catastrophic asphyxiating event of unknown causes, "of course he did."[101]

46. Professor Berry observed in his report that the post-mortem examination showed a normally grown baby with no natural disease to account for death, and that there were no marks of violence and no features such as facial petechial haemorrhages to suggest suffocation.[102]

47. In the P79A report, Senior Constable Bryant recorded that when Ms Folbigg found Caleb, she found a small amount of blood and froth around his mouth.



About 1am on Monday the 20 February, 1989, the child was fed by his mother. He was then put to bed in adjoining bedroom. About 2.53am (20.2.89) the mother awaoke and checked the child and found him to be cold and apparently dead, she found a small amount of blood and thrøth around the childs mouth. The father was alerted and began CPR, till Ambulance Officers arrived, but to no avail. The body was conveyed to the Newcastle Hospitah where life was pronoumced extinct at 4am, by Dr Sandy CHAPMAN.

The child had been taken to a Dr Springthorpe, of Watt street, Newcastle for treatment for a ' Lazy Larynx'. No medication was given.

Figure 4: Extract from P79A report re Caleb: Exhibit H in the Inquiry, p 4

48. This report on the P79A assumed significance in the Inquiry, with Dr Cala identifying it as a cause of concern (see [70]).

49. At the time of trial, Professor Berry stated in his report that blood stained froth around the nose and mouth is a common finding in sudden infant deaths and in accidental or deliberate suffocation.[103] He stated that milk in Caleb's stomach (found on autopsy) was consistent with Ms Folbigg's account of having recently fed him.[104]

50. The finding of haemosiderin would cause Professor Berry not to call Caleb's death SIDS, but to ask further questions – he would probably call it "unascertained". He considered that Caleb died from a catastrophic asphyxial event of unknown causes "only to the extent that we all do, because we all stop breathing". While there was no histology of Caleb's upper airway, Professor Berry reported that Caleb's lungs showed no evidence of infection.[105]

51. Professor Byard gave evidence that he would have said the cause of Caleb's death was "undetermined" (which did not exclude SIDS).[106] In his first report he stated he would list Caleb's death as "undetermined, with laryngomalacia", because Caleb's autopsy findings could not be taken in isolation.[107]

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101 Responses to Crown model questions – Professor Herdson, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.

102 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 24.

103 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 24.

104 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 24; Autopsy report of Caleb Folbigg (9 May 1989) p 2.

105 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 21.

106 7 May 2003 T1202.17-26, T1209.6-24. This was consistent with his report, however, in his report he stated he would have noted a history of breathing problems involving a floppy larynx (laryngomalacia): Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 4.

107 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 7.

52. The reasons for Professor Byard's conclusion included that there was no death scene examination (and so he would not make a diagnosis of SIDS) and no histology of the brain.[108] There was also a history of breathing problems with the diagnosis of a floppy larynx and a possible cause of death albeit, he agreed under cross-examination, highly unlikely.[109] It was significant, also, that Caleb's brain did not appear to have been examined histologically.[110] As noted above, there was no condition or symptom observed that amounted to proof of suffocation, but the autopsy findings were the same for suffocation as for SIDS.[111]

53. Professor Busuttil stated that in the presence of the floppy larynx known to be giving rise to respiratory obstruction in life, Caleb's death should have been classified as "'undetermined' – sudden infant death",[112] with haemosiderin raising the question of imposed upper airways obstruction. Professor Busuttil observed there was no suggestion that Caleb had any internal metabolic problems, and that genetic studies after death did not show such an anomaly.[113]

54. Dr Richard Hawker, consultant paediatric cardiologist at The Children's Hospital at Westmead, examined "Medi Traces" from Caleb's medical records but could not interpret them because of their poor quality.[114]

Evidence in the Inquiry

SIDS risk factors

55. In her report, Professor Horne, infant sleep and SIDS specialist, referred to the following "potential protective factors for SIDS" in respect of Caleb: Caleb was born at term at a normal weight, was found supine with his face uncovered and in his own bed and was vaccinated. In respect of his age at death of 19 days, she recorded that 10% of SIDS cases in the United States in 2004-2006 died under one month of age. She also referred to an increased risk in mothers younger than 26 years at the time of the baby's birth (Ms Folbigg was 21 when Caleb was born) and paternal smoking.[115]

56. Taking the above into account, Professor Horne gave evidence in the Inquiry that Caleb did not have the major risk factors for SIDS and was at low risk.[116] Similarly, Professor Elder, paediatrician, said Caleb had no clear, classic risk factors, albeit referring to a "little question mark" given respiratory stress at birth, noisy breathing, and laryngomalacia.[117]

Haemosiderin

57. As noted above, the Inquiry was unable to obtain new evidence about the origins of the slides seen by Professor Berry and was informed that they are not now available. Accordingly, they could not be examined by the forensic pathologists who gave evidence in the Inquiry.

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108 7 May 2003 T1202.12-26, T1205.4-39, T1206.7-11; Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 9.

109 7 May 2003 T1203.25-1205.39, T1206.7-15, T1234.15-38, T1254.25-32; Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 9.

110 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 9.

111 7 May 2003 T1205.41-1206.15.

112 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) pp 8-9.

113 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002).

114 Exhibit H, Forensic pathology tender bundle, Statement of Dr Richard Hawker (6 March 2003) p 1.

115 Exhibit J, Expert report of Professor Rosemary Horne (10 February 2019) pp 2-3.

116 Transcript of the Inquiry, 18 March 2019 T34.42-45.

117 Transcript of the Inquiry, 18 March 2019 T35.15-35.

58. However, in the Inquiry (and consistently with Duncan and Byard, 2018) the forensic pathologists said that the view today is that haemosiderin in the lungs is not a positive indicator of superimposed upper airway obstruction.[118] It is still understood that it takes a number of days after the blood is deposited for haemosiderin to become apparent.[119] There is no evidence of where it came from in Caleb.

Opinions on cause of Caleb's death

59. Professors Duflou, Cordner and Hilton would all have given Caleb's death as Category 2 SIDS given the presence of laryngomalacia and because Caleb was younger in age than the classic SIDS range.[120]

60. Professor Cordner observed in his report that had Caleb died two days later, his death by contemporary standards would have been properly diagnosed as SIDS (Category 1B) because of the incomplete level of investigation/documentation according to today's standards.[121] He stated that a floppy larynx is "[a] common condition which resolves with time".[122] He considered that laryngomalacia potentially meant that Caleb was more vulnerable to SIDS.[123]

61. Professor Michael Pollanen, Chief Forensic Pathologist for Ontario, reviewed Professor Cordner's report and gave a short summary of his opinions which were appended to Professor Cordner's report.

62. He stated in relation to Caleb that there were no positive pathologic findings. Professor Pollanen utilised a formulation of "causal relevance" drawn from Spitz and Fisher's *Medicolegal Investigation of Death*,[124] which categorises forensic pathologists' diagnosis of cause of death into five classes:

    a. Class 1. Definitively fatal acute pathologic finding(s) are identified that imply the cause of death, based on correlation with the history and exclusionary findings;

    b. Class 2. Potentially fatal acute pathologic finding(s) are identified that imply the cause of death, based on correlation with the history and exclusionary findings;

    c. Class 3. Potentially fatal chronic pathologic finding(s) are identified that imply the cause of death, based on correlation with the history and exclusionary findings;

    d. Class 4. Non-definitive (non-specific) pathologic findings are present that can explain death based on inference from correlation with history and exclusionary findings; and

    e. Class 5. No pathologic findings are present that can explain death based on inference, despite correlation with history and exclusionary findings.

63. Professor Pollanen ascribed Caleb's death as Class 5.[125]

64. In his report Professor Duflou also ascribed SIDS Category 2 due to Caleb's age and a history of congenital laryngeal stridor almost certainly on the basis of laryngomalacia (although it was not specifically identified at autopsy).[126]

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118 Transcript of the Inquiry, 20 March 2019 T232.30-39.

119 Transcript of the Inquiry, 20 March 2019 T233.16-35; Exhibit D, Roger W Byard, 'The Autopsy and Pathology of Sudden Infant Death Syndrome' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 497, 504-505.

120 Transcript of the Inquiry, 19 March 2019 T130.8-14, T245.25-31 (Professor Duflou), T130.18-20, T244.7-16 (Professor Hilton), T130.37-131.9, T278.12-16(Professor Cordner).

121 Exhibit Q, Report of Professor Stephen Cordner (undated) pp 83, 90.

122 Exhibit Q, Report of Professor Stephen Cordner (undated) p 9.

123 Transcript of the Inquiry, 21 March 2019 T278.12-16.

124 (Charles C Thomas Publishers, 4th ed, 2005).

125 Exhibit C, Report of Professor Michael Pollanen (1 June 2015) p 5.

126 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 29; Transcript of the Inquiry, 19 March 2019 T130.8-14, T140.12-18.

65. Professor Duflou distinguished a floppy larynx from laryngeal stridor or laryngomalacia.[127] He noted that Caleb had a prior medical history of congenital laryngeal stridor, considered to almost certainly be on the basis of laryngomalacia, and a diagnosis on chest x-ray after birth of possible pneumomediastinum (the abnormal presence of air or another gas in the mediastinum).[128] He stated that, arguably, Caleb's death is explained by possible laryngomalacia with upper airway obstruction.[129] He also noted that that autopsy recorded larynx and upper airway as unremarkable.[130]

66. Professor Duflou referred to research that 90 per cent of laryngomalacia is benign and self-limiting, echoing Professor Berry's evidence at trial, but noted that the remainder, if untreated, can prove fatal with death attributed to either upper airway obstruction or heart failure.[131]

67. Professor Hilton said that it could not be proved or disproved whether laryngomalacia played a part.[132]

68. Consistently with his evidence at trial, in the Inquiry Dr Cala did not accept the floppy larynx was a cause of death. He was not convinced laryngomalacia was in any way serious, referring to it as a relatively benign abnormality discovered in the neonatal period which the infant outgrows as cartilage strengthens, and generally disappears with an "observe for now" approach.[133]

69. Dr Cala would not ascribe SIDS for Caleb's death, "even by today's standards".[134] He stated that a diagnosis of SIDS may be made only if there are no suspicious circumstances surrounding the death, the scene examination, the subsequent autopsy and the results of ancillary tests are all subsequently negative; Dr Cala had concern in particular in relation to what may have caused the blood and froth around Caleb's mouth.[135] He stated that given Caleb was 19 days, he would initially be searching for possible underlying infectious, congenital, metabolic or other natural causes of death as well as excluding suspicious scene findings or circumstances.[136]

70. Dr Cala tempered his view from that expressed at trial, where he said that he considered that Caleb was "likely smothered". He maintained that he would ascribe Caleb's death as undetermined, in view of Caleb's age and concern about the report of blood and froth.[137] He observed that the report to the coroner (above at [48]) suggested it was a small amount, but the volume is unknown.[138] He stated it could not have been caused by CPR, because there was no reference to it in the autopsy report, and CPR also only occurred when Mr Folbigg was alerted.[139]

71. Dr Cala said the blood and froth did not exclude the possibility of some external agent having been applied to Caleb's outer airway, whether accidentally or deliberately, particularly given Caleb was in a supine position when found, and decomposition was not a possible cause.[140]

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127 Exhibit L, Report of Professor Johan Duflou (13 February 2019) pp 29-30.
128 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 29.
129 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 45.
130 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 29.
131 Transcript of the Inquiry, 21 March 2019 T245.27-31; Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 30, citing Michael R N Baxter, 'Congenital Laryngomalacia' (1994) 41 *Canadian Journal of Anaesthesia* 332.
132 Transcript of the Inquiry, 21 March 2019 T278.4-6.
133 Transcript of the Inquiry, 21 March 2019 T277.34-41; Exhibit M, Report of Dr Allan Cala (26 November 2018) p 7.
134 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 5; Transcript of the Inquiry, 19 March 2019..
135 Exhibit M, Report of Dr Allan Cala (26 November 2018) pp 5, 7; Transcript of the Inquiry, 19 March 2019 T127.25-128.47.
136 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 6.
137 Transcript of the Inquiry, 19 March 2019 T127.25-128.1, T128.49-129.40; Exhibit M, Report of Dr Allan Cala (26 November 2018) pp 5, 7.
138 Transcript of the Inquiry, 19 March 2019 T128.3-34.
139 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 6.
140 Transcript of the Inquiry, 19 March 2019 T128.36-47.

72. In his first report for the Inquiry, Dr Cala gave a number of reasons for his concern. Oronasal blood may be a sign of accidental or inflicted suffocation. Finding of frank blood at the external airway is unusual and concerning, and differs from oronasal secretions, which is common in SIDS. Assuming that Caleb was supine, which is supported by lack of reports of blood on the pillow or sheets, oronasal blood, however, occurs rarely in SIDS when the infant is sleeping supine in a safe environment. Caleb was fed milk less than two hours earlier and had been apparently well. While early decomposition can sometimes cause a small amount of blood on external airways, this could reasonably be excluded given the timeframe since the feed. Further, a number of possible causes of the froth and blood were excluded: Caleb was not face down after death, was not co-sleeping, and did not have congenital heart disease or pulmonary oedema.[141] Dr Cala referred to seven articles in medical literature as supporting his concern.[142]

73. The other forensic pathologists who gave evidence did not share Dr Cala's views regarding the blood and froth. Professor Duflou said that in the absence of anything else, it did not negate SIDS as an entirely reasonable cause of death.[143] In the Inquiry, and also at trial, Professor Hilton described frothy, bloody fluid at the nose and sometimes mouth as commonplace in SIDS.[144] Professor Cordner opined that such a finding was not particularly exceptional in SIDS.[145] These views are consistent with Professor Berry's evidence at trial.[146]

Time of death

74. Professor Duflou observed that the time of Caleb's death was not formally assessed at autopsy or by ambulance officers but noted that Caleb was described as cold to touch by Ms Folbigg, and either warm or cold to touch by ambulance officers.[147]

75. Professor Duflou also observed Caleb to have a large quantity of curdled milk in his stomach at the time of autopsy, noting that the time taken for the stomach to empty in infants is variable and complex, but in general one to two hours is not unreasonable.[148] He acknowledged, however, that providing opinions based on stomach content is dangerous for forensic pathologists, and there are "probably graveyards full of forensic pathologists who have done that" – Professor Cordner described it as "tiger country".[149] Professor Duflou stated it appeared that Caleb died some short time after he was checked by his mother at 10:00pm, and likely not around the time he was next checked by her at 2:45am.[150]

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141 Exhibit M, Report of Dr Allan Cala (26 November 2018) pp 6-7.

142 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 7, citing Sydney Smith, *Forensic Medicine* (J & A Churchill, 8th ed, 1945) 263; Jay Dix, 'Homicide and the Baby-Sitter' (1998) 19(4) *American Journal of Forensic Medicine and Pathology* 321; C J Polson and D J Gee, *The Essentials of Forensic Medicine* (Pergamon Press, 3rd ed, 1973) 544; David Dolinak, Evan Matshes and Emma Lew, *Forensic Pathology* (Elsevier Academic Press, 1st ed, 2005) 206; Exhibit AO, American Academy of Pediatrics, 'Distinguishing Sudden Infant Death Syndrome from Child Abuse Fatalities' (2006) 118(1) *Pediatrics* 421; D P Southall et al, 'Covert Video Recordings of Life-Threatening Child Abuse: Lessons for Child Protection' (1997) 100(5) *Pediatrics* 735; Andrew M Baker, 'Pediatric Asphyxial Deaths' in Kim A Collins and Roger W Byard (eds), *Forensic Pathology of Infancy and Childhood* (Springer New York, 2014) 207.

143 Transcript of the Inquiry, 19 March 2019 T130.3-10.

144 14 April 2003 T632.46-48; Transcript of the Inquiry, 19 March 2019 T130.16-20.

145 Transcript of the Inquiry, 19 March 2019 T130.31-33.

146 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 24.

147 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 29; Transcript of the Inquiry, 19 March 2019 T141.39-40.

148 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 29; Transcript of the Inquiry, 19 March 2019 T140.45-50.

149 Transcript of the Inquiry, 19 March 2019 T125.40-44 (Professor Duflou), T126.10 (Professor Cordner).

150 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 29; Transcript of the Inquiry, 19 March 2019 T141.4-10.

Counsel assisting's submissions on cause of Caleb's death

76. Counsel assisting submitted that there have been two material changes since the 2003 trial. First, genetic testing has been completed and no genetic variant which is pathogenic or likely pathogenic has been identified to account for Caleb's death (or any of his siblings). Secondly, more recent research on SIDS that maternal smoking and sleeping position pose the highest risks relevantly lowers, in retrospect, the risk posed to Caleb of SIDS.[151]

77. In other respects, in relation to the cause of Caleb's death, counsel assisting submitted that the opinions expressed to the Inquiry, based on the same information available in 2003, remain broadly similar to those given at the trial.[152] Haemosiderin is now considered less likely to be an indicator of suffocation and Dr Cala has somewhat tempered his view as to the likelihood of smothering, while not ruling it out.[153] It remains the case that any contribution to Caleb's death by laryngomalacia is highly unlikely.[154]

78. The weight of the expert opinion at trial was that his death was best described as undetermined. In the Inquiry, apart from Dr Cala the forensic pathology experts preferred Category 2 SIDS.[155]

79. Counsel assisting noted that whereas experts at trial hesitated or qualified ascribing SIDS in large part due to Caleb being 19 days old when he died, and younger than generally acceptable SIDS age at the time, Category 2 SIDS expressly contemplates the death of a child Caleb's age. In the Inquiry Professor Duflou said, Professor Hilton agreeing, that Caleb's death ought to be ascribed as Category 2 SIDS *because of* his age at time of death.[156]

80. However described, SIDS – and particularly Category 2 SIDS – does not answer the cause of Caleb's death. SIDS does not exclude unidentified natural causes. Nor does SIDS, and particularly Category 2 SIDS, described as being more flexible, exclude unnatural causes which are not identifiable at autopsy. It can be virtually impossible to distinguish at autopsy between a SIDS death and a death caused by deliberate or accidental suffocation.[157]

81. Counsel assisting submitted that on forensic pathology evidence, both "undetermined" and SIDS apply to Caleb's death. Both terms leave open the possibility of an unidentified natural cause, or unidentified unnatural cause, of death.[158]

82. Counsel assisting also submitted that the weight of the expert evidence in relation to the blood and froth recorded on the P79A in relation to Caleb is that it could have been present whether Caleb's death was SIDS or the result of accidental or deliberate smothering.[159]

83. In light of Caleb having been fed at around 1:00am, and of the ambulance officers' evidence, counsel assisting submitted Professor Duflou's statement that Caleb likely died around 10:00pm and likely not around 2:45am should not be accepted.[160]

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151 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [24].
152 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [25].
153 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [17].
154 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [26].
155 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [27].
156 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [28]; Transcript of the Inquiry, 19 March 2019 T130.9-20.
157 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [29].
158 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [30].
159 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [6].
160 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [23].

84. In relation to time of death, counsel assisting set out evidence of ambulance officers who attended Caleb. When the first unit arrived at 2:59am he was unconscious, not breathing and pulseless, and warm to touch.[161] Mr Reed, in the second unit, described Caleb's skin temperature as cold to touch, and that he was obviously not breathing and had no circulation.[162] Although Mr Reed recorded his arrival time as 3:03am (shortly after the first unit), he also recorded the airway as "clear" whereas the first officers recorded it as "obstructed". In addition, the second unit used an ECG upon their arrival.[163] Accordingly, there may have been additional time between the two sets of observations of Caleb's skin temperature.[164] Professor Duflou agreed that the body temperature described by ambulance officers was not known, and also said that an assessment of body temperature on the basis of feel is very unhelpful.[165] Professor Hilton doubted that these observations had any relevance as to when Caleb died.[166]

85. Counsel assisting submitted that, in any event, Professor Duflou based his opinion in his report predominantly on Caleb's stomach contents and on information that Caleb was checked at 10:00pm (and that this was the latest time that Caleb was fed). Ms Folbigg gave a statement that Caleb was fed at 1:00am, said in her ERISP that Caleb had had an early morning feed, and recorded in her diary that he was finally asleep at 2:00am. In his oral evidence, Professor Duflou agreed it was obvious that if Caleb was fed at 1:00am, or early in the morning, and finally went to sleep at 2:00am then death could not have occurred prior to 2:00am.[167]

86. Ultimately, counsel assisting submitted that on the medical evidence in 2019 there remains no identified natural (including genetic) cause of Caleb's death and death from unnatural causes cannot be excluded. Most medical experts considered that the death could have been the result of an asphyxiating event. No medical expert excluded asphyxia or smothering.[168]

Ms Folbigg's submissions on cause of Caleb's death

87. Ms Folbigg submitted that the presence of blood and froth on Caleb has no significance because the forensic pathologists could not be sure of its cause.[169]

88. She submitted that Dr Cala's evidence at the Inquiry contradicted that given at trial: that at the trial he maintained that all of the children were deliberately smothered, but at the Inquiry he conceded that Caleb's death fit within Category 2 SIDS.[170]

89. She also submitted that it is not a matter for her to show that asphyxia or smothering can be excluded[171] and in any event "[t]he Inquiry could not be reasonably satisfied, on the evidence before it, that asphyxia or smothering occurred insofar as Caleb is concerned."[172]

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161 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [20]; Exhibit H, Forensic pathology tender bundle, Statement of David Hopkins (1 October 1999) p 2, Statement of Richard Baines (29 October 1999) p 2; 3 April 2003 T142.27-28.

162 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [21]; Exhibit H, Forensic pathology tender bundle, Statement of Allen Reed (1 September 1999) p 13.

163 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [21]; Exhibit H, Forensic pathology tender bundle, Statement of Allen Reed (1 September 1999) p 13.

164 Exhibit H, Forensic pathology tender bundle, Ambulance Report Q005 (20 February 1989) p 1.

165 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [22]; Transcript of the Inquiry, 19 March 2019 T142.4.

166 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [22]; Transcript of the Inquiry, 19 March 2019 T121.44-46.

167 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [19]-[23].

168 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 7, [31]-[32].

169 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [10]-[13].

170 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [16]-[21], citing 15 April 2003 T726.50-727.06, T728.48 and Transcript of the Inquiry, 19 March 2019 T129.40.

171 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [28]-[29], [39].

172 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [30].

90. Ms Folbigg referred to counsel assisting's submission that it is highly unlikely that laryngomalacia had any contribution to Caleb's death, and contended that that submission is either not supported by the evidence or even if it is, laryngomalacia cannot be excluded as a contributor. She pointed to the following matters in support of this argument:

    a. The evidence of the ambulance report of Mr Hopkins which recorded that Caleb's airway was obstructed. Ms Folbigg submitted that the clear inference from this document is that the airway was obstructed by the airway's collapse due to laryngomalacia or laryngospasm.[173] If the airway was obstructed by a solid object, the ambulance officer would have noted it.[174]

    b. There was no study done of the larynx and no histology to determine whether the cartilage was sufficient to prevent obstruction of the upper airway.[175]

    c. When he was reviewed at two weeks of age, Caleb had inspiratory stridor and some recession. If Caleb was having recession in the chest, then it is clear the laryngomalacia was demonstrating some impact on his breathing two days before his death.[176]

    d. According to Professor Byard, the effect of the condition can be worsened by a child lying on its back, and Dr Springthorpe noted that if Caleb was unsettled this also worsened the condition. Caleb was upset on the night of his death and was found lying on his back.[177]

    e. When first born, the position of the larynx and the epiglottis enables simultaneous breathing and drinking but Caleb was unable to simultaneously breathe and drink.[178]

    f. Since the trial there has been a greater focus by otolaryngologists in respect of SIDS and laryngomalacia is now a recognised potential cause of death rather than a theoretical possibility. At the Inquiry, Professors Hilton and Duflou accepted that laryngomalacia could be fatal, and Professor Hilton expressly accepted that it was a potential cause of death in Caleb. Dr Cala conceded he did not have the expertise to form a view about what level of laryngomalacia is likely to kill somebody.[179]

    g. By contrast, Professor Byard's evidence at trial that if Caleb had died of a floppy larynx it would be a world first, probably enabled the jury to put death by laryngomalacia to one side as a reasonable cause of death in Caleb.[180] Had the evidence before the Inquiry been available at trial, the defence would have been able to mount a far stronger argument about its likelihood.[181]

    h. Caleb was not an entirely healthy child as submitted at trial. He had upper respiratory tract issues and Dr Springthorpe decided on conservative management. His assessment could have been inadequate. Dr Springthorpe was unaware of the bleeding in Caleb's lungs. Caleb was "still at an age where improvement could not have been expected to resolve his laryngomalacia".[182]

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173 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [61]-[62], citing Exhibit H, Forensic pathology tender bundle, Ambulance report Q005 (20 February 1989) and 3 April 2003 T142.47, T145.17-146.09, T147.23-46.

174 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [82].

175 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [74].

176 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [68(d), (f)].

177 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [77]-[79], citing 7 April 2003 T269.58-270.05, T270.40 (Dr Springthorpe), 7 May 2003 T1204.1205.03 (Professor Byard).

178 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [93].

179 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [86]-[91], [93].

180 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [80].

181 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [99], citing 14 May 2003 T1420.05-1421.15.

182 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [95].

91. Ms Folbigg concluded by submitting that the evidence at trial and in the Inquiry "demonstrates an almost overwhelming alternative natural cause of death" which:

> *cannot be displaced on the basis of unproven inferences to be derived from theories of 'four deaths in one family' nor… by one interpretation of the diaries seen through the prism of a presumption of guilt.*[183]

92. She also submitted that the jury's verdict of manslaughter, rather than murder, reflects "a perversity of reasoning that was not readily available on the Crown case."[184]

Professor Hilton's submissions on cause of Caleb's death

93. In his submissions in response, Professor Hilton said that Ms Folbigg's submissions fairly reflected his opinion as to the possible cause of death of Caleb.[185]

Findings: Caleb

94. In relation to Caleb's death, having regard to all of the medical evidence set out above, I find as follows.

95. The weight of the expert evidence in relation to the blood and froth recorded on the P79A in relation to Caleb is that it could have been present whether Caleb's death was SIDS or the result of accidental or deliberate smothering. It neither supports nor detracts from either hypothesis as a cause of Caleb's death.

96. Expert evidence does not establish that the presence of haemosiderin is a positive indicator of suffocation. It is a non-specific finding and there is no evidence of what may have caused it.[186] I do not regard the presence of haemosiderin identified by Professor Berry, noting that his views cannot now be verified in the absence of the slides, as indicative of accidental or deliberate smothering in Caleb's case, and evidence received in the Inquiry does not now enable the cause of the haemosiderin to be identified. However, I do not accept Ms Folbigg's submission that Dr Springthorpe's assessment could have been inadequate given he was unaware of it, or that its presence ought to bear on the weight attributed to Dr Springthorpe's evidence at trial.

97. Upon evidence available to the Inquiry, any contribution to Caleb's death by laryngomalacia is very unlikely. Evidence available to the Inquiry does not completely exclude laryngomalacia as a possible contributor, but I find the possibility is so low as to be remote. It is not an "almost overwhelming alternative natural cause of death." In that regard, I note in particular the following evidence:

    a. Dr Springthorpe's evidence that by 17 February 1989, Caleb's inspiratory stridor was mild, or "very, very mild", and the recession was "little" and most marked when Caleb was upset or lying on his back;

    b. the absence of webs or cysts found on autopsy;

    c. research and evidence that laryngomalacia has been recorded as a potential cause of death, but forensic pathologists who gave evidence at trial and in the Inquiry had either never heard of such a case or, in the minority of experts, had seen it or heard of it only on very few occasions;[187] and

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183 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [101].

184 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [102].

185 Submissions of Professor Hilton to the Inquiry (18 June 2019).

186 Transcript of the Inquiry, 20 March 2019 T232.20 (Professor Cordner), T232.35-39 (Dr Cala and Professor Hilton agreeing), T233.11 (Dr Cala), T233.15 (Professor Duflou agreeing).

187 Responses to Crown model questions – Professor Berry, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C; Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 9.

d. no forensic pathologist gave an opinion at trial or in the Inquiry that laryngomalacia caused Caleb's death. Some gave evidence that they were not aware from either their experience or published literature of a child dying of laryngomalacia.[188] Most preferred a view that it did not contribute,[189] or were unable to form a strong view either way.[190]

98. It is true, as Ms Folbigg submits, that Mr Hopkins' ambulance report recorded an obstruction in the airway. In his evidence at trial, Mr Hopkins agreed that it could be taken that in the absence of a mention in his report of any particular substance, there would only have been saliva, fluid or phlegm obstructing the airway.[191] I do not infer from his report, as Ms Folbigg submits I should, that the airway was obstructed by collapse due to laryngomalacia or similar.

99. On the evidence available to me, I agree with the submission of counsel assisting that the two material changes since the 2003 trial are the genetic testing conducted by the Inquiry, and SIDS research.

100. As set out in Chapter 7 of this report, genetic testing of samples retained on autopsy of all the Folbigg children has been completed. No genetic variant which is pathogenic or likely pathogenic has been identified to account for Caleb's death.

101. In relation to SIDS research, more recent research, referred to by Professors Horne and Elder, highlights that maternal smoking, sleeping position and bed sharing pose the highest risks. Neither of these risk factors were present in relation to Caleb. Viewed retrospectively, and with the benefit of contemporary research and understanding of SIDS, the risk posed to Caleb of SIDS was diminished by the absence of either of the major risk factors. I accept Professors Horne's and Elder's evidence that Caleb's risk of SIDS was low, noting Professors Elder's and Cordner's references to the presence of stridor and laryngomalacia.

102. Particularly with low risk factors, evidence available to the Inquiry establishes that "SIDS", and particularly "Category 2 SIDS", is far from complete as an explanation for Caleb's death. SIDS Category 2 more readily accommodates Caleb's death than did the earlier definition by Krous et al,[192] because it expressly extends SIDS to infants of Caleb's age when he died and has a degree of flexibility in its terms compared with the former definition and with SIDS Category 1A and 1B. Ascribing Caleb's death as SIDS acknowledges that on occasion infants of Caleb's age die with no identifiable explanation.

103. I find that both "undetermined" and SIDS can apply to Caleb's death, noting that Caleb's risk factors for SIDS were low but his laryngomalacia may have contributed to a vulnerability in that regard. Both descriptors leave open the possibility of an unidentified natural cause, or unidentified unnatural cause, of death. Expert evidence was consistent in both the trial and the Inquiry that it can be very difficult, indeed virtually impossible, to distinguish between SIDS and suffocation at autopsy.

104. In relation to the time at which Caleb died, I note Professor Duflou's concession in his oral evidence that if Caleb was fed at around 1:00am and went to sleep at 2:00am then death could not have occurred prior to 2:00am. I note that there is no evidence to controvert Ms Folbigg's statement made soon after Caleb's death that he was fed around 1:00am.

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188 15 April 2003 T728.29-36 (Dr Cala); 1 May 2003 T1034.24-30 (Professor Herdson).

189 7 April 2003 T267.48-50 (Dr Springthorpe); 1 May 2003 T1057.15-35 (Professor Berry); Transcript of the Inquiry, 21 March 2019 T245.10, T277.29-41 (Dr Cala).

190 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) [5.8]-[5.9]; Exhibit L, Report of Professor Johan Duflou (13 February 2019) pp 30, 45; Transcript of the Inquiry, 21 March 2019 T278.4-6 (Professor Hilton).

191 3 April 2003 T 142.33-35, T145.45-46, T147.23-46.

192 See Henry F Krous et al, 'Sudden Infant Death Syndrome and Unclassified Sudden Infant Deaths: A Definitional and Diagnostic Approach' (2004) 114 *Pediatrics* 234.

105. Taking into account all the medical evidence available to me, there remains no identified natural (including genetic) cause of Caleb's death. Death from unnatural causes cannot be excluded. Most medical experts considered that the death could have been the result of an asphyxiating event. No medical expert has excluded asphyxia or smothering as a possible cause of Caleb's death.Patrick

Patrick

Patrick's birth and life before the ALTE

106. Patrick was born at 39 weeks' gestation (full term) and normal weight on 3 June 1990.[193] According to hospital notes, he was bottle fed upon discharge on 8 June 1990.[194] Notes recorded that there was to be a sleep study on 14 June 1990 and an appointment with Dr Robert Morris, a paediatrician at the Mater Hospital, at three weeks.[195]

107. The sleep study when Patrick was one and a half weeks old was conducted given Caleb's death, to investigate Patrick for possible SIDS-related problems. He was admitted to hospital for the investigations.[196] The study was carried out by Dr David Cooper, formerly Head Paediatrician at the Paediatric Respiratory and Sleep Service at the John Hunter Hospital.

108. Dr Cooper gave evidence at trial and a prepared statement dated 6 December 1999.[197] At trial, Dr Cooper described studies in 1990 and 1992 as being "simplistic", using "primitive" equipment, and said that techniques had become much more sophisticated since.[198] For example, at the time, the doctors did not differentiate between central and obstructive apnoea: central apnoea being where the brain fails to send a message to the respiratory system to breathe and obstructive apnoea referring to some obstruction meaning the air is not able to get in.[199]

109. Dr Cooper said that the results of Patrick's tests were entirely normal.[200] Dr Ian Wilkinson, paediatric neurologist, also gave evidence at trial of Patrick's investigations.[201] He also said that the results of Patrick's tests were within normal limits.[202]

110. On 18 October 1990, when Patrick was four months and 15 days old, he suffered an ALTE. This is discussed further below.

111. Dr Christopher Marley, a general practitioner, saw Patrick on several occasions during Patrick's lifetime.[203] In his statement of 9 March 1999, he stated that he saw no sign of neglect on Patrick.[204] In his evidence at trial he said that Ms Folbigg impressed him as a caring and concerned parent.[205] He saw Patrick five times for routine childhood illnesses and injections, and said that

> *Patrick was no different from many other children. We saw him for minor respiratory infections. He did have a major neurological problem, but that was mainly dealt with by his paediatrician and neurologist. That was the epilepsy and cortical blindness.*[206]

193 Exhibit H, Forensic pathology tender bundle, Birth certificate of Patrick (3 June 1990).
194 Exhibit H, Forensic pathology tender bundle, Neonatal record of Patrick (8 June 1990) p 1.
195 Exhibit H, Forensic pathology tender bundle, Neonatal record of Patrick (8 June 1990) p 1.
196 10 April 2003 T512.32-46.
197 Exhibit H, Forensic pathology tender bundle, Statement of Dr David Cooper (6 December 1999) p 1; 14 April 2003 T585.25-615.6.
198 14 April 2003 T586.6-18.
199 14 April 2003 T586.12-32.
200 14 April 2003 T587.39-588.3; Exhibit H, Forensic pathology tender bundle, Statement of Dr David Cooper (6 December 1999) p 2.
201 Exhibit H, Forensic pathology tender bundle, Statements of Dr Ian Wilkinson (12 March 1999 and 8 October 1999).
202 10 April 2003 T512.35-55.
203 Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Marley (9 March 1999).
204 Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Marley (9 March 1999) [5].
205 11 April 2003 T538.46-48.
206 11 April 2003 T539.10-17.

112. Dr Marley's reference to the major neurological problem was to Patrick's symptoms and disorders following the ALTE. Otherwise, Patrick was "progressing well and growing well."[207] He never required antibiotics.[208]

Patrick's ALTE

Ambulance call-out and reports

113. Mr Folbigg gave evidence at trial that on the occasion of Patrick's ALTE on 18 October 1990, he was awoken by a "blood-curdling scream".[209] He ran down to Patrick's bedroom and saw Ms Folbigg standing at the end of the cot, screaming.[210] He noted the covers were down towards the end of the bed, and Patrick looked like he was asleep. Mr Folbigg grabbed him out of his bed and screamed at Ms Folbigg to call the ambulance. He heard a little noise and thought he was breathing. Patrick was warm and pink. Mr Folbigg started CPR.[211]

114. Ms Folbigg was recorded as saying that Patrick had been coughing at 3:00am, and she saw him then. She was alerted again at 4:30am because she heard Patrick gasping.[212] She noted that he was blue around the lips, lifeless and floppy, and was making minimal respiratory effort.[213] She stated that CPR was not performed and soon after this Patrick gave a high pitched cry.[214]

115. Mr David Hopkins and Mr Lance Yorke were ambulance officers who attended in response to Patrick's ALTE, arriving at the Folbigg home 4:41am.[215] Patrick's clothing was moved to expose his chest and Mr Hopkins saw that he appeared to be having respiratory difficulties. Patrick was pale around the face and listless. He exhibited tracheal tug and intercostal recession, that is, he was in respiratory distress and was attempting to breathe but it was very laboured.[216]

116. Mr Hopkins and Mr Yorke immediately transported Patrick to the Mater Hospital.[217] Mr Hopkins applied oxygen therapy en route[218] and Patrick's level of consciousness rose although his respiratory effect remained impaired.[219] They arrived at the hospital at 4:52am.[220]

117. Mr Hopkins and Mr Yorke recorded on the ambulance report that Ms Folbigg had told them that Patrick had had a cold for two days but was normally happy and dynamic.[221] The ambulance report recorded that Patrick's skin temperature was hot.[222]

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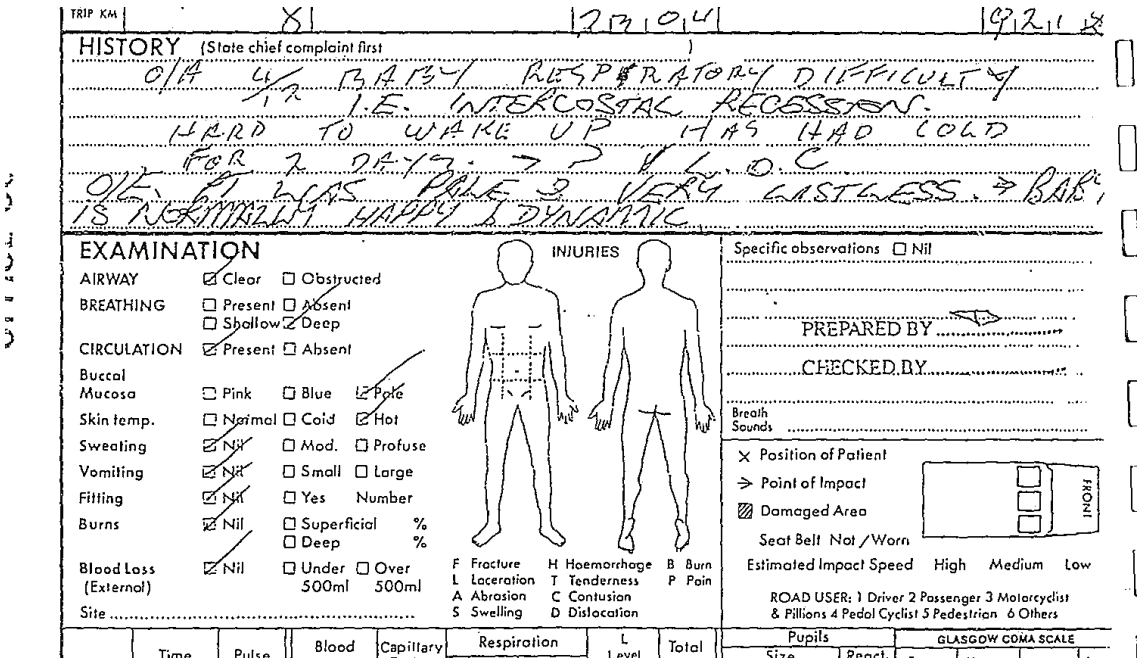
207 11 April 2003 T539.29-31.
208 11 April 2003 T539.48-52.
209 2 April 2003 T109.41-44.
210 2 April 2003 T109.44-49.
211 2 April 2003 T110.7-39.
212 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 2.
213 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 2.
214 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 2; 9 April 2003 T447.21-32.
215 Exhibit H, Forensic pathology tender bundle, Statement of David Hopkins (1 October 1999) p 3.
216 Exhibit H, Forensic pathology tender bundle, Statement of David Hopkins (1 October 1999) p 4; 9 April 2003 T436.14.
217 Exhibit H, Forensic pathology tender bundle, Statement of David Hopkins (1 October 1999) p 4.
218 Exhibit H, Forensic pathology tender bundle, Statement of David Hopkins (1 October 1999) p 4; 9 April 2003 T436.48-50.
219 9 April 2003 T436.52-56.
220 Exhibit H, Forensic pathology tender bundle, Statement of David Hopkins (1 October 1999) p 4; 9 April 2003 T436.58-437.1.
221 Exhibit S, Section of Patrick's medical records, p 526; 9 April 2003 T437.22-48, T438.19-21.
222 Exhibit S, Section of Patrick's medical records, p 526; 9 April 2003 T438.23-34.


TRIP KM 81 12/10/41 19/2/11

HISTORY (State chief complaint first)

O/A 4½ BABY RESPIRATORY DIFFICULTY
I.E. INTERCOSTAL RECESSION.
HARD TO WAKE UP HAS HAD COLD
FOR 2 DAYS. → V.O.C
O/E PT LOOKS PALE & VERY LISTLESS. → BABY
IS NORMALLY HAPPY & DYNAMIC

EXAMINATION

AIRWAY	☑ Clear	☐ Obstructed	
BREATHING	☐ Present	☐ Absent	
	☐ Shallow	☑ Deep	
CIRCULATION	☑ Present	☐ Absent	
Buccal Mucosa	☐ Pink	☐ Blue	☑ Pale
Skin temp.	☐ Normal	☐ Cold	☑ Hot
Sweating	☑ Nil	☐ Mod.	☐ Profuse
Vomiting	☑ Nil	☐ Small	☐ Large
Fitting	☑ Nil	☐ Yes	Number
Burns	☑ Nil	☐ Superficial	%
		☐ Deep	%
Blood Loss (External)	☑ Nil	☐ Under 500ml	☐ Over 500ml

Site .....

INJURIES

F Fracture L Laceration A Abrasion S Swelling
H Haemorrhage T Tenderness C Contusion D Dislocation
B Burn P Pain

Specific observations ☐ Nil

PREPARED BY .....

CHECKED BY .....

Breath Sounds .....

× Position of Patient
→ Point of Impact
▨ Damaged Area
FRONT
Seat Belt Not / Worn
Estimated Impact Speed High Medium Low

ROAD USER: 1 Driver 2 Passenger 3 Motorcyclist & Pillions 4 Pedal Cyclist 5 Pedestrian 6 Others

Time	Pulse	Blood	Capillary	Respiration	L Level	Total	Pupils Size	Pupils React.	GLASGOW COMA SCALE

Figure 5: Ambulance report dated 18 October 1990: Exhibit S in the Inquiry, p 526

118. A hospital note upon Patrick's admission recorded that he had been "snuffly" for the past three days with dry coughs and some vomits post bottle feeds, but had otherwise been well and behaving normally with no fevers.[223]

Medical investigation immediately following the ALTE

119. Dr Joseph Dezordi, neonatal fellow, examined Patrick at the Mater Hospital on 18 October 1990.[224] He gave a statement dated 17 March 2000 and oral evidence at trial.

120. When Patrick came in, Dr Dezordi "saw a lethargic, cyanosed infant, who was responsive only to painful stimuli."[225] In his oral evidence, he described Patrick as being hypoxic on arrival; blue, lethargic, with no fever.[226]

121. On initial examination, Patrick had an oxygen saturation level of 88 per cent.[227] Dr Dezordi proceeded to treat him with oxygen administered by a Hudson mask, and after about 15 minutes Patrick became more alert, and remained pink, even when oxygen in high concentration was not being administered. Dr Dezordi therefore concluded that Patrick's condition was not likely to be due to a respiratory problem.[228]

223 Exhibit S, Section of Patrick's medical records, p 533.
224 9 April 2003 T446.25-447.13; Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) [4].
225 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (7 March 2000) p 2.
226 9 April 2003 T446.46-447.13; T452.8-13.
227 Exhibit S, Section of Patrick's medical records, p 534.
228 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 2.

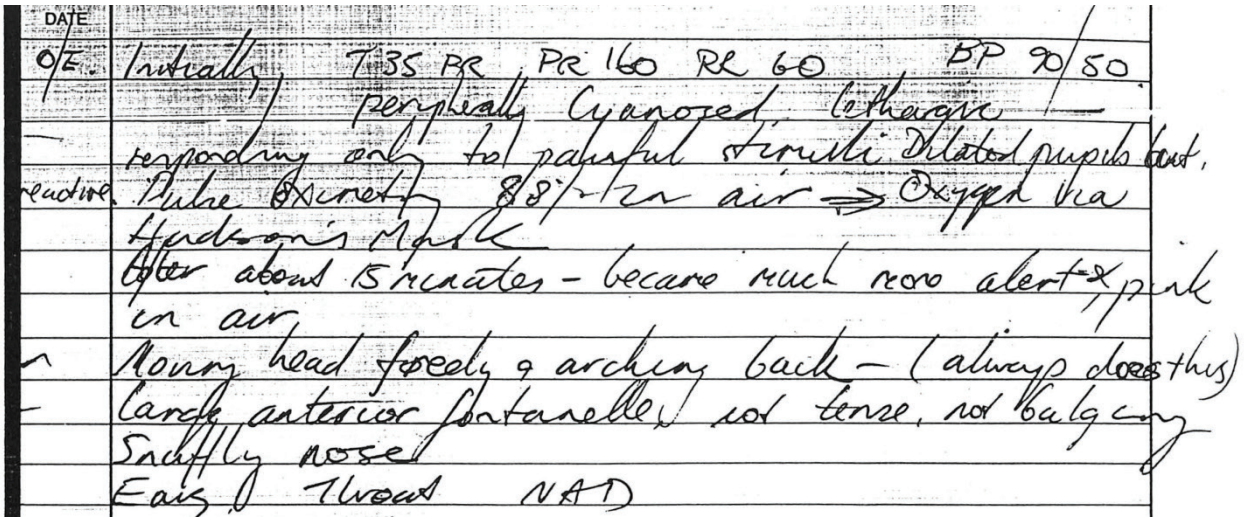

DATE
O/E. Initially T 35 RR PR 160 RR 60 BP 90/50
Peripherally Cyanosed, lethargic —
responding only to painful stimuli. Dilated pupils but,
reactive. Pulse Oxim 88% in air ⇒ Oxygen via
Hudson's Mask
After about 5 minutes - became much more alert & pink
on air
Mouny head floppy & arching back — (always does this)
Large anterior fontanelle, not tense, not bulging
Snuffly nose
Ears, Throat NAD

Figure 6: Mater Hospital medical notes recorded by Dr Dezordi on examination of Patrick on 18 October 1990: Exhibit H in the Inquiry, p 82

122. Dr Dezordi also gave oral evidence before the jury that because of "fairly rapid normalisation of [Patrick's] colour and oxygen saturation level", he concluded that "I was not dealing primarily with pathology involving his lungs, chest and possibly his airways" (such as pneumonia or bronchiolitis).[229] He said that Patrick appeared to respond to oxygen treatment, although it was possible he was getting better spontaneously.[230] Medical staff were able to measure the oxygen content in his blood with saturation monitors and Patrick maintained saturation levels.[231]

123. Dr Dezordi conducted a detailed examination of Patrick, which he said was generally unremarkable at this stage.[232] Patrick was appropriately grown.[233] He was arching his back; this was non-specific although it could indicate that Patrick may have been cerebrally irritated, in turn indicating inflammation of the brain, meningitis or other pathology involving the brain.[234] However, Patrick did not have sufficient signs at the time for Dr Dezordi to pursue a diagnosis of meningitis.[235]

124. There were no signs to suggest other serious illness, or of trauma or injuries.[236] There were also no signs of upper airway obstruction or aspiration which might have accounted for the history Ms Folbigg gave Dr Dezordi, that Patrick had been gasping and that he was barely breathing.[237]

229 9 April 2003 T448.2-41.
230 9 April 2003 T448.2-3.
231 9 April 2003 T448.15-18.
232 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 2.
233 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 2; 9 April 2003 T448.58.
234 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 3; 9 April 2003 T482.49-483.4, T449.1-6.
235 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 3; 9 April 2003 T450.9-16.
236 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (7 March 2000) p 3; 9 April 2003 T450.5-7.
237 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) pp 2-3; 9 April 2003 T449.11-18.

125. By 6:00am on 18 October 1990, preliminary test results were available. The blood tests showed no abnormality.[238] However, a urine test returned a high level of glucose, which was unexpected and which Dr Dezordi said in his oral evidence before the jury tended to suggest "a fairly catastrophic event, such as an asphyxiating event, or a prolonged seizure."[239] By "asphyxiating event" he meant "any event, that leads to obstruction of air into the lungs and impairment of oxygen levels in the blood and to the brain."[240] At the time he received the results, Dr Dezordi thought it could have possibly been caused by a seizure of some kind, or an episode of prolonged hypoxia.[241]

126. During this early period in hospital, Patrick vomited three times but had no respiratory difficulty with the vomiting.[242]

127. On 19 October 1990, hospital progress notes recorded Patrick as seeming "okay" and "fine in the morning".[243] However, around 2:00pm he had a high temperature and was vomiting, and in the afternoon he was "not a very well boy".[244]

128. At 9:00pm that night, Patrick had a convulsion fit, with physical shaking. Overnight through to 20 October 1990, he had approximately 15 fits involving stiffening of the neck, eyes open and glazed with a fixed stare.[245] The longest fit was one minute and 10 seconds, the shortest was 30 seconds.[246] He was given Valium and an anti-fitting drug which seemed to work.[247]

129. Dr Dezordi saw Patrick at 6:00am on 20 October 1990.[248] Dr Dezordi noted the fitting, and that Patrick's eyes were deviated to the right hand side.[249] He gave evidence at trial of Patrick having recurrent seizures in the two days after he was admitted, and subsequently.[250]

130. Dr Dezordi gave evidence that virological tests did not support a diagnosis of bronchiolitis (compared with an official report that a chest x-ray showed signs which could be due to bronchiolitis).[251] Patrick was found to be neurologically normal, although the examinations were arguably not complete because his pupils were not dilated to investigate for retinal haemorrhages.[252] Meningitis, septicaemia and meningococcal were not present.[253]

238 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 3; 9 April 2003 T450.20-25.
239 9 April 2003 T449.38-55; Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (7 March 2000) p 2.
240 9 April 2003 T449.57-450.3.
241 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 3.
242 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 3; 9 April 2003 T451.15-23.
243 Exhibit S, Section of Patrick's medical records, p 437.
244 Exhibit S, Section of Patrick's medical records, p 437.
245 Exhibit S, Section of Patrick's medical records, p 439.
246 Exhibit S, Section of Patrick's medical records, p 439.
247 Exhibit S, Section of Patrick's medical records, p 439.
248 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 4.
249 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 4.
250 9 April 2003 T466.46-49.
251 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (7 March 2000) p 3; 10 April 2003 T451.37-43, T505.7-21.
252 9 April 2003 T450.27-451.13.
253 9 April 2003 T449.40-45, T450.20-25.

131. By 23 October 1990, Patrick's progress was recorded as slow with occasional fitting.[254] Dr Dezordi thought that the seizures were focal (localised), and so ordered a CT scan, which was conducted that day by Dr Man Kit Lai, a staff specialist radiologist at the Mater Hospital.[255] Dr Lai's report indicated some brain abnormalities in the occipital lobes (at the back), and temporal lobes (at the side).[256]

Exams : CB3 on 23-Oct-1990 at 11

CT BRAIN (pre and post contrast scans)

In the pre contrast scant there is a decrease in attenuation seen in both occipital lobes, temporal lobe and left frontal lobe. The grey/white matter differentiation is lost. Ventricular system not dilated. No haemorrhage seen. Minimal widening of the peripheral cerebral sulci is seen in the frontal and the parietal lobes.

Post contrast scan with thin cuts over the posterior cranial fossa and temporal lobe shows the hypodense areas involving both posterior parts of the temporal lobes and occipital lobes. Abnormal enhancement demonstrated. The intra-cranial vessels are well enhanced. No abnormal fluid collection seen.

IMPRESSION

The picture is compatible with encephalitis involving both temporal lobes, occipital lobes and left frontal lobe. Herpes encephalitis has to be considered.

Figure 7: Extract of report of CT scan on 23 October 1990 by Dr Lai: Exhibit H in the Inquiry, p 72

132. In his statement, Dr Dezordi stated that it was "not really clear what the cause of these unusual CT scan findings were".[257] He recorded in his notes at the time that they demonstrated some pathological process involving the occipital and temporal lobes of the brain.[258]

133. A first EEG of Patrick's brain was taken on 18 October 1990 and a second on 22 October 1990.[259] In oral evidence, Dr Wilkinson, paediatric neurologist, explained that an EEG measures electrical waves in the brain to see the normality of electrical activity rhythm for continuous functioning of the brain, and changes consistent with any epileptic process.[260] The result of Patrick's first EEG indicated normal functioning and thus, Dr Dezordi said, an absence of encephalitis.[261] The second EEG also did not disclose a pattern suggestive of herpes simplex encephalitis.[262]

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254 Exhibit S, Section of Patrick's medical records, p 439.

255 9 April 2003 T467.2-4; Exhibit H, Forensic pathology tender bundle, Statement of Dr Man Kit Lai (11 February 2000).

256 9 April 2003 T467.52-468.6; 10 April 2003 T497.1-19; Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (7 March 2000) p 4, Statement of Dr Man Kit Lai (11 February 2000) p 3.

257 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 4.

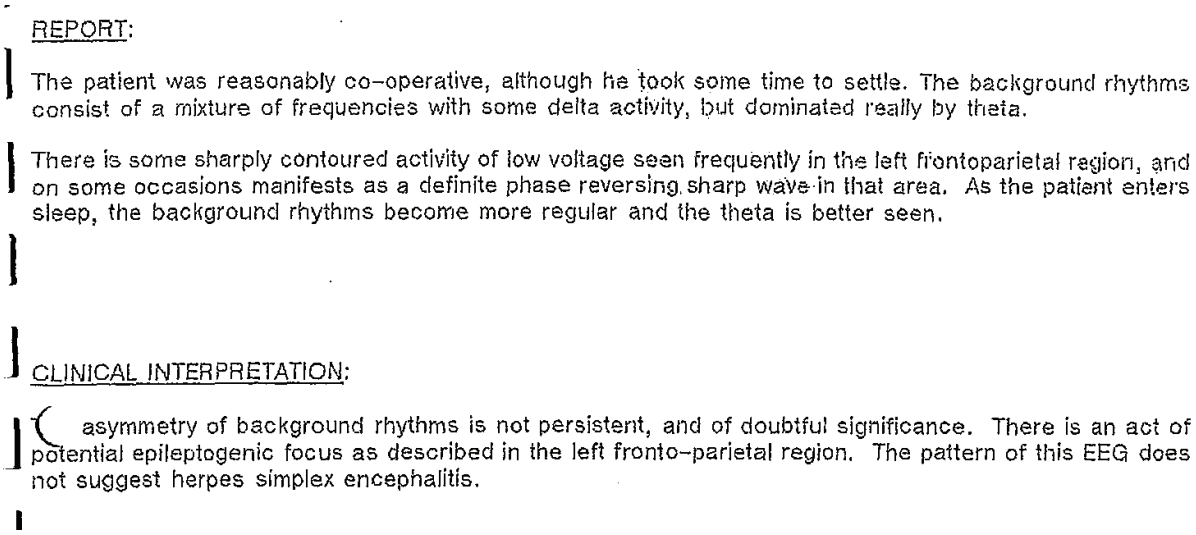
258 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 4.

259 Exhibit S, Section of Patrick's medical records, p 525; Exhibit S, Section of Patrick's medical records, p 613.

260 10 April 2003 T508.36-40.

261 9 April 2003 T457.42-458.46, see 10 April 2003 T507.53-56 (Dr Wilkinson); Exhibit S, Section of Patrick's medical records, p 525.

262 Exhibit S, Section of Patrick's medical records, p 613.



REPORT:

The patient was reasonably co-operative, although he took some time to settle. The background rhythms consist of a mixture of frequencies with some delta activity, but dominated really by theta.

There is some sharply contoured activity of low voltage seen frequently in the left frontoparietal region, and on some occasions manifests as a definite phase reversing sharp wave in that area. As the patient enters sleep, the background rhythms become more regular and the theta is better seen.

CLINICAL INTERPRETATION:

(asymmetry of background rhythms is not persistent, and of doubtful significance. There is an act of potential epileptogenic focus as described in the left fronto-parietal region. The pattern of this EEG does not suggest herpes simplex encephalitis.

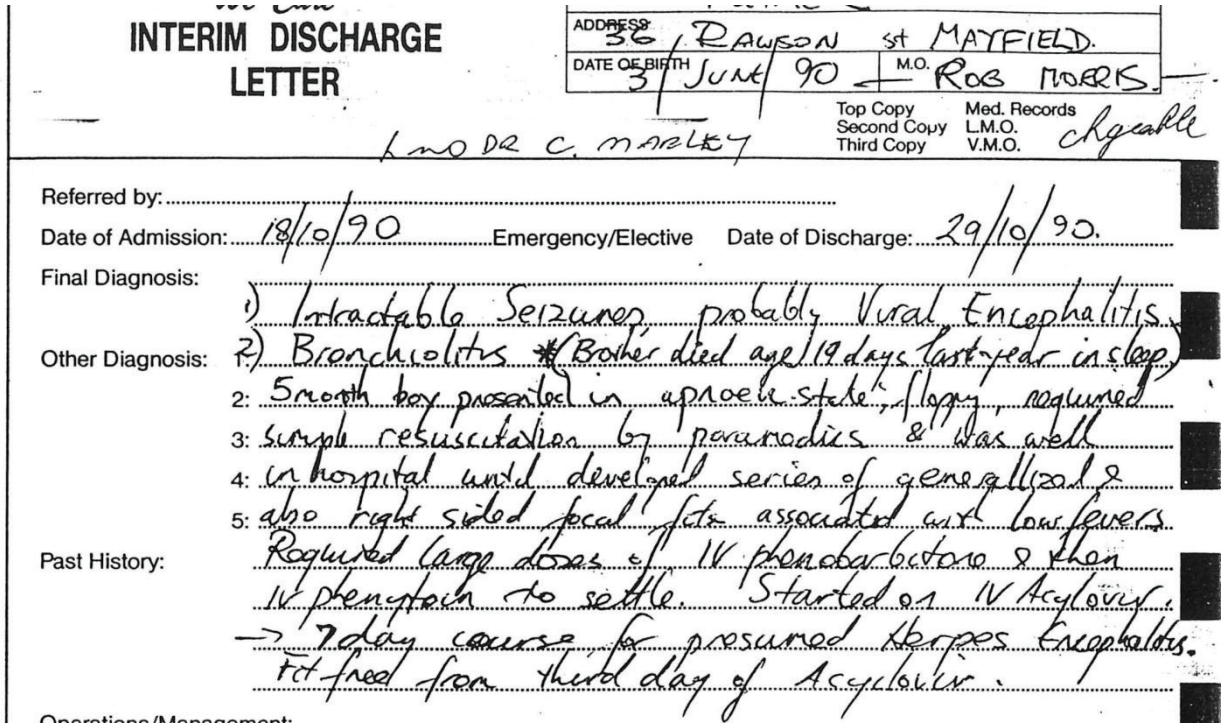
Figure 8: Extract of the second EEG report dated 22 October 1990: Exhibit S in the Inquiry, p 613

134. Dr Wilkinson saw Patrick between 18 and 20 October 1990. Dr Wilkinson gave evidence at trial about the investigations, including the CT scans and EEG tests.[263] In a reporting letter, he described Patrick as presenting with what initially sounded like apnoea but that subsequently in the ward he demonstrated clearly that he was having seizures.[264] On examination Dr Wilkinson could not find any neurological problem.[265] The CT scan, which he said was "[t]he most worrying thing", showed symmetrical areas of hypodensity in occipital regions posteriorly, changes which suggested the possibility of a metabolic disorder although herpes encephalitis could not be ruled out absolutely.[266] While Patrick had been febrile (feverish) at various times, he had no white cells in his spinal fluid and only three red cells.[267] Dr Wilkinson wrote, that "[w]e have chosen to treat as though he might have Herpes Simplex encephalitis, with Acylclovir, but his fits have been quite resistant to treatment".[268]

135. At the time of Patrick's admission, Dr Dezordi considered that herpes encephalitis was possible, although with "strong doubts" given the CT scan and also a normal lumbar puncture.[269] In his oral evidence at trial, Dr Dezordi explained that while a normal lumbar puncture does not exclude herpes, it is very rare; in light of normal EEG, lumbar puncture and tests for viruses, he said he was left with unknown causes of Patrick's seizures.[270]

136. Dr Dezordi prepared Patrick's discharge report on 29 October 1990, recording as "diagnosis", "intractable seizures, probably viral encephalitis" and also "bronchiolitis" and "brother died aged 19 days in sleep."[271]

263 10 April 2003 T507.25-508.11.
264 Exhibit H, Forensic pathology tender bundle, Letter from Dr Ian Wilkinson to Dr Robert Morris (30 October 1990).
265 Exhibit H, Forensic pathology tender bundle, Letter from Dr Ian Wilkinson to Dr Robert Morris (30 October 1990).
266 Exhibit H, Forensic pathology tender bundle, Letter from Dr Ian Wilkinson to Dr Robert Morris (30 October 1990).
267 Exhibit H, Forensic pathology tender bundle, Letter from Dr Ian Wilkinson to Dr Robert Morris (30 October 1990).
268 Exhibit H, Forensic pathology tender bundle, Letter from Dr Ian Wilkinson to Dr Robert Morris (30 October 1990).
269 9 April 2003 T468.21-26; 10 April 2003 T484.53-58.
270 9 April 2003 T468.51-469.38.
271 Exhibit H, Forensic pathology tender bundle, Interim discharge letter of Dr Christopher Marley (29 October 1990); 10 April 2003 T484.48.51.



INTERIM DISCHARGE LETTER

ADDRESS: 36 RAWSON st MAYFIELD.
DATE OF BIRTH: 3/JUNE/90 M.O. ROB MORRIS.
Top Copy Med. Records
Second Copy L.M.O.
Third Copy V.M.O.
Lnoor C. MARLEY chgeable

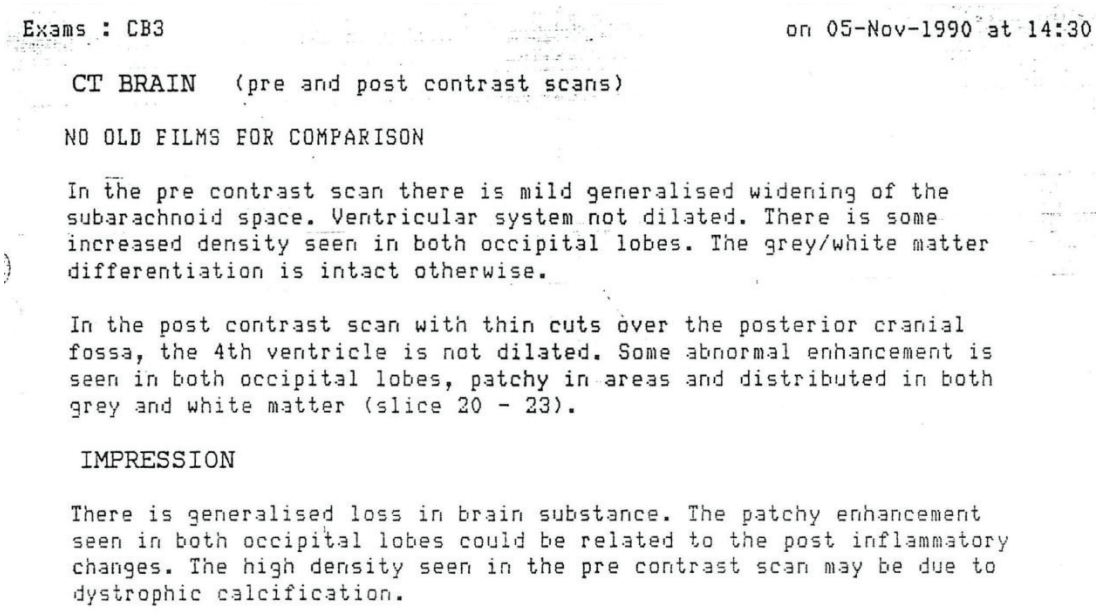
Referred by:
Date of Admission: 18/10/90 Emergency/Elective Date of Discharge: 29/10/90.
Final Diagnosis:
1) Intractable Seizures probably Viral Encephalitis.
Other Diagnosis: 2) Bronchiolitis *(Brother died age 19 days last year in sleep)
2: 5month boy presented in apnoeic state, floppy, required
3: simple resuscitation by paramedics & was well
4: in hospital until developed series of generalized &
5: also right sided focal fits associated with low fevers.
Past History: Required large doses of IV phenobarbitone & then IV phenytoin to settle. Started on IV Acyclovir. → 7day course for presumed Herpes Encephalitis. Ceased from third day of Acyclovir.
Operations/Management:

Figure 9: Extract from Mater Hospital interim discharge letter completed by Dr Dezordi, dated 29 October 1990: Exhibit H in the Inquiry, p 80

137. In his trial evidence, Dr Dezordi said that the use of "probably" for viral encephalitis was to describe a "working possibility" – he had nothing else to explain Patrick's presentation medically, so it was a possibility.[272] However, Dr Dezordi was never convinced that Patrick had encephalitis.[273] The normal lumbar punctures caused him to doubt the encephalitis theory altogether.[274] He recorded on the discharge, "marked serial deterioration in three EEGs suggests progressive encephalopathic process", and gave evidence that this was a very ambiguous term, which showed the doctors did not know what was going on.[275] He said that the term was not the same as encephalitis, and he was not thinking herpes encephalitis.[276] He said the damage to Patrick's brain "definitely" could have been caused by asphyxiation.[277]

138. Patrick presented again at hospital on 4 November 1990, with a seizure.[278] Dr Dezordi organised a repeat CT scan for 5 November 1990, which was again conducted by Dr Lai. This scan demonstrated abnormalities already seen on the previous scan but they seemed to have worsened, with deterioration and damage at the back of Patrick's brain.[279]

272 10 April 2003 T484.22-40.
273 10 April 2003 T484.57-58.
274 10 April 2003 T488.30-38.
275 10 April 2003 T489.3-42, T499.23-51; Exhibit H, Forensic pathology tender bundle, Interim discharge letter of Dr Christopher Marley (29 October 1990).
276 10 April 2003 T487.6-15.
277 10 April 2003 T499.57-500.19.
278 Exhibit S, Section of Patrick's medical records, p 650.
279 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 4; 9 April 2003 T469.43-55, 10 April 2003 T487.6-15.



Exams : CB3 on 05-Nov-1990 at 14:30

CT BRAIN (pre and post contrast scans)

NO OLD FILMS FOR COMPARISON

In the pre contrast scan there is mild generalised widening of the subarachnoid space. Ventricular system not dilated. There is some increased density seen in both occipital lobes. The grey/white matter differentiation is intact otherwise.

In the post contrast scan with thin cuts over the posterior cranial fossa, the 4th ventricle is not dilated. Some abnormal enhancement is seen in both occipital lobes, patchy in areas and distributed in both grey and white matter (slice 20 - 23).

IMPRESSION

There is generalised loss in brain substance. The patchy enhancement seen in both occipital lobes could be related to the post inflammatory changes. The high density seen in the pre contrast scan may be due to dystrophic calcification.

Figure 10: Extract of report of CT scan on 5 Nov 1990 by Dr Lai: Exhibit H in the Inquiry, p 73

139. Dr Lai gave evidence at trial. He said that when he prepared the reports of the scans, he believed Patrick was suffering from encephalitis, possibly due to herpes simplex.[280] In his statement, Dr Lai stated that he believed that at the time he was searching for the most likely diagnosis and this should always be considered until excluded – this was part of his usual work practice.[281]

140. However, the cause of this "loss of brain substance" was not clear to Dr Dezordi or to any of the medical staff.[282] Following many further tests and consultations with colleagues, Dr Dezordi was unable to find a specific explanation for the abnormality.[283]

141. A third EEG on 5 November 1990 also showed abnormalities, which Dr Dezordi said would not be necessarily pathognomonic of encephalitis – it could just be a process of epilepsy that is untreated or unresolved, but could possibly also be due to herpes encephalitis.[284] He said the report did not necessarily leave open a possibility that Patrick had encephalitis, because an encephalopathic process is much broader than herpes encephalitis itself – i.e. could be, for example, due to an ongoing infection, metabolic disease, or brain damage due to hypoxia.[285] While the third EEG report was less inconsistent with encephalitis than was the previous EEG report, by 5 November 1990 any diagnosis of herpes encephalitis would be somewhat tenuous, given normal lumbar punctures (a far more powerful test than an EEG) in the interim since Patrick's presentation.[286]

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280 Exhibit H, Forensic pathology tender bundle, Statement of Dr Man Kit Lai (11 February 2000) [6].

281 Exhibit H, Forensic pathology tender bundle, Statement of Dr Man Kit Lai (11 February 2000) [6].

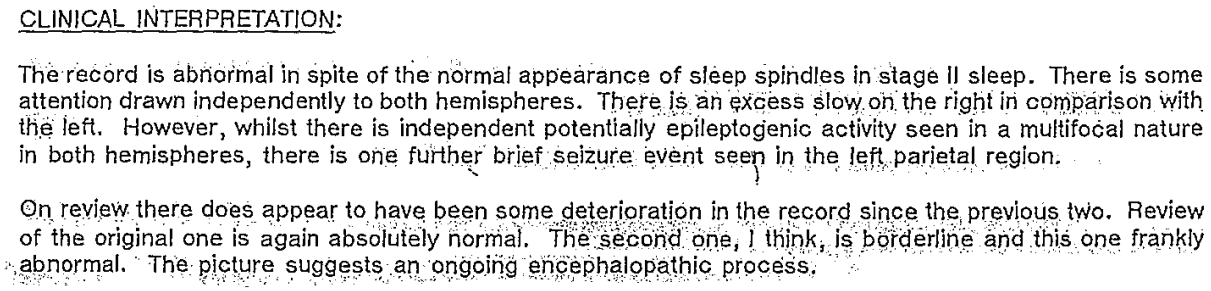
282 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 4, CT scan report of Patrick (5 November 1990).

283 9 April 2003 T470.5-22, T470.44-49.

284 9 April 2003 T460.11-31.

285 9 April 2003 T460.33-41. An encephalopathic process is any diffuse disease of the brain that alters brain function or structure: National Institute of Neurological Disorders and Strokes, 'Encephalopathy', *Encephalopathy Information Page* (Web Page, 27 March 2019) <https://www.ninds.nih.gov/Disorders/All-Disorders/Encephalopathy-Information-Page>.

286 9 April 2003 T460.49-53, T461.1-11.



CLINICAL INTERPRETATION:

The record is abnormal in spite of the normal appearance of sleep spindles in stage II sleep. There is some attention drawn independently to both hemispheres. There is an excess slow on the right in comparison with the left. However, whilst there is independent potentially epileptogenic activity seen in a multifocal nature in both hemispheres, there is one further brief seizure event seen in the left parietal region.

On review there does appear to have been some deterioration in the record since the previous two. Review of the original one is again absolutely normal. The second one, I think, is borderline and this one frankly abnormal. The picture suggests an ongoing encephalopathic process.

Figure 11: Extract from EEG report dated 5 November 1990: Exhibit S in the Inquiry, p 682

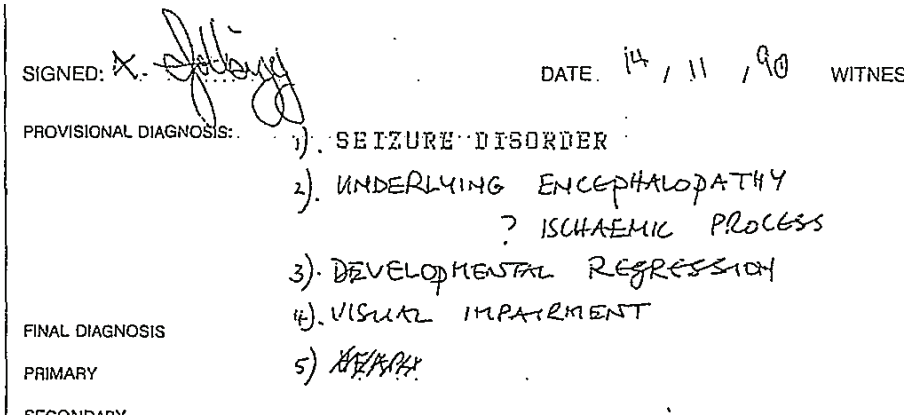
142. Dr Wilkinson gave evidence that after the first EEG, subsequent EEGs showed increasing abnormalities and progressive epileptogenic change.[287] He said he thought the CT scan showed localised changes in the occipital lobes (back part of the brain) whereas the EEGs demonstrated changes in multiple areas.[288]

143. At trial, Dr Dezordi was asked about whether an encephalopathic process included damage to the brain which could be caused from a hypoxic event, such as where the brain has been starved of oxygen.[289] He agreed, and also with the propositions that asphyxiation can cause a hypoxic event and that encephalopathic process would include asphyxiation.[290]

144. Dr Dezordi discussed Patrick's case with Dr Wilkinson and at Dr Wilkinson's request, the CT scans were sent to Professor Merl DeSilva, an expert radiologist at the Children's Hospital at Camperdown.[291] Professor DeSilva said that the changes in the CT scans were not classical of encephalitis.[292] Desperate to find an explanation, Dr Dezordi also looked at rare medical diseases.[293]

145. On 14 November 1990, Patrick was admitted again to the Mater Hospital following generalised seizures, and with a virus.[294] By then, Patrick had lost the ability to fix on a face and to follow, and seemed to only respond to bright lights.[295] He had further seizures in hospital before he was discharged on 22 November 1990.[296]

287 10 April 2003 T507.53-56.
288 10 April 2003 T508.46-51.
289 10 April 2003 T499.23-500.3.
290 10 April 2003 T500.5-19.
291 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 4; 9 April 2003 T470.15-42.
292 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) p 5.
293 9 April 2003 T470.44--49; Exhibit H, Forensic pathology tender bundle, Statement of Dr Dezordi (17 March 2000) History, examination and progress notes dated 18 October 1990.
294 Exhibit S, Section of Patrick's medical records, pp 714, 747, 772.
295 Exhibit S, Section of Patrick's medical records, p 747.
296 Exhibit S, Section of Patrick's medical records, pp 714, 722, 772.


SIGNED: DATE. 14 / 11 / 90 WITNES

PROVISIONAL DIAGNOSIS:
1). SEIZURE DISORDER
2). UNDERLYING ENCEPHALOPATHY
? ISCHAEMIC PROCESS
3). DEVELOPMENTAL REGRESSION
4). VISUAL IMPAIRMENT
5)

FINAL DIAGNOSIS
PRIMARY
SECONDARY

Figure 12: Extract from Mater Hospital discharge form dated 22 November 1990: Exhibit S in the Inquiry, p 774

146. Because Patrick had a sibling who had died, all the tests available in 1990 were conducted when Patrick was admitted following the ALTE, to look for inherited diseases that might have brought about neurological abnormalities. Dr Wilkinson described the system of testing that the doctors did as "extremely exhaustive", looking at a number of markers in the body for abnormalities of biochemical pathways, including chemicals in the urine, a rectal biopsy and staining of white cells to look for occlusions.[297] No abnormalities were found.[298] The doctors never had any absolute explanation for how Patrick suffered the damage to his brain.[299]

Patrick's death

147. Patrick died on 13 February 1991. Mr Folbigg gave evidence that at 10:00am that morning he received a phone call at work from Ms Folbigg, who screamed down the phone "It's happened again" and "I need you. Come home".[300] He drove home quickly and upon running into the house saw Ms Newitt (his sister) and Ms Folbigg there. He raced into Patrick's room and saw he was lying in his cot. He scooped Patrick up, put him on the lounge and commenced CPR. Patrick was floppy, warm, with blue lips. The ambulance officers then took over.[301]

148. Ms Kathleen Coyle, Mr Russell Mullins, and Mr Murray Hetherington, ambulance officers, attended the Folbigg house at 10:10am.[302] Ms Coyle stated that she went through the lounge room and into another room on her left side, where she saw a man who she thought was attempting CPR on Patrick. Ms Coyle performed heart compressions, and one of the other officers performed ventilation on Patrick for a very short time.[303] Ms Coyle checked Patrick's vital signs and from her notes it appears that he was normal/warm to touch and slightly blue around the lips.[304] Ms Coyle's notes also recorded shallow breathing, however, this was contradicted in the respiration rate having been recorded as "nil".[305] Ms Coyle considered that she would never have recorded breathing as being present and shallow if she did not note it that day.[306]

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297 10 April 2003 T509.4-43.

298 10 April 2003 T509.41-46; Exhibit H, Forensic pathology tender bundle, Letter from Dr Ian Wilkinson to Dr Christopher Marley (30 November 1990).

299 10 April 2003 T509.48-50.

300 2 April 2003 T115.3-17.

301 2 April 2003 T116.24-27.

302 Exhibit H, Forensic pathology tender bundle, Statement of Kathleen Coyle (6 September 1999) p 1; 9 April 2003 T439.34-56.

303 Exhibit H, Forensic pathology tender bundle, Statement of Kathleen Coyle (6 September 1999) p 2; 9 April 2003 T440.7-55.

304 Exhibit H, Forensic pathology tender bundle, Statement of Kathleen Coyle (6 September 1999) p 2; 9 April 2003 T441.1-10.

305 Exhibit H, Forensic pathology tender bundle, Statement of Kathleen Coyle (6 September 1999) p 2; 9 April 2003 T442.23-26.

306 Exhibit H, Forensic pathology tender bundle, Statement of Kathleen Coyle (6 September 1999) p 2; 9 April 2003 T442.28-443.1.

149. Ambulance notes, annexed to Ms Coyle's statement and signed by her and Mr Mullins, recorded that Patrick had a previous history of apnoeic episodes and epilepsy, that his skin colour was pink, he was peripherally cyanosed, his skin temperature was warm, and he had nil vital signs.[307]

150. Mr Hetherington stated that he knelt down to examine Patrick in the house, where Mr Folbigg appeared to be performing CPR. Mr Hetherington found that Patrick had no pulse and was not breathing, and there was no question he was in cardiac arrest.[308] Mr Hetherington picked Patrick up and commenced CPR and took him to the Mater Hospital.[309] Mr Hetherington said he could categorically state that Patrick was not breathing when he examined him.[310]

151. Mr Mullins stated that Mr Hetherington commenced CPR on Patrick at the house, and Patrick appeared not to be responding.[311] Mr Mullins believed there was never any breathing noted when the officers arrived.[312]

152. Dr Wilkinson saw Patrick while resuscitation attempts were being performed at the Mater Hospital in Newcastle, during which it became clear to him that Patrick had died.[313] At trial, Dr Wilkinson said that Patrick's body was warm, indicating the death was fairly recent.[314] Dr Christopher Walker was the Director of the Emergency Department at the Mater Hospital when Patrick arrived.[315] He pronounced Patrick's death at 10:40am.[316] He gave evidence at trial that the death was following an out of hospital cardiac arrest, no cause for which was found.[317]

Autopsy reports

153. The post-mortem of Patrick was conducted by Dr Gurpreet Singh-Khaira, histopathologist who had experience conducting post-mortem examinations, and Dr Jan Bishop, anatomical pathologist. Two reports were produced, one dated 14 February 1991[318] and a final report dated 2 September 1991.[319]

Interim autopsy report

154. The report dated 14 February 1991 recorded that the time and date of death was 1040 hours on 13 February 1991, and the time and date of the post-mortem was 1230 hours on 13 February 1991.[320]

155. The diagnoses were recorded as:

> *Clinical Diagnosis*
>
> *1. Encephalopathic disorder leading to intractable seizures. The underlying cause of encephalopathy not determined on investigation.*
>
> *2. Asystolic cardiac arrest at home leading to death.*

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307 Exhibit H, Forensic pathology tender bundle, Ambulance report Q037 (13 February 1991).
308 Exhibit H, Forensic pathology tender bundle, Statement of Murray Hetherington (6 September 1999) p 2.
309 Exhibit H, Forensic pathology tender bundle, Statement of Murray Hetherington (6 September 1999) p 2.
310 Exhibit H, Forensic pathology tender bundle, Statement of Murray Hetherington (6 September 1999) p 3.
311 Exhibit H, Forensic pathology tender bundle, Statement of Anthony Mullins (1 October 1999) p 2.
312 Exhibit H, Forensic pathology tender bundle, Statement of Anthony Mullins (1 October 1999) p 2.
313 10 April 2003 T510.48-55; Exhibit H, Forensic pathology tender bundle, Statement of Dr Ian Wilkinson (8 October 1999) [4].
314 10 April 2003 T511.17-20.
315 9 April 2003 T472.1-13; Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Walker (18 January 2000) [5].
316 9 April 2003 T473.45-50; Exhibit H, Forensic pathology tender bundle, Report of Professor Robert Ouvrier (28 October 2002) p 4.
317 9 April 2003 T474.19-25; Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Walker (18 January 2000) [7].
318 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991).
319 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Patrick (2 September 1991).
320 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991) p 1.

Macroscopic Diagnosis

1. Normally formed male infant of approximately eight months of age.

2. Brain and spinal cord fixed for later dissection.

3. Hepatic congestion.

4. Congested postero-basal dependant segments – both lungs.

5. Enlarged thymus.[321]

156. The clinical history recorded in the report stated that the presenting complaint was that Patrick was brought into casualty at 1020 hours by paramedics on 13 February 1991 "after an asystolic cardiac arrest at home".[322] ECG on arrival in casualty showed asystole. Subsequent resuscitation efforts were unsuccessful and Patrick was pronounced dead at 1040 hours.[323]

157. Patrick's past medical history was recorded, including details of a viral illness suffered by Ms Folbigg at 26 weeks, Patrick's birth, the sleep studies, and the ALTE. In relation to the ALTE, the report recorded that after resuscitation, Patrick remained well until he developed generalised and also right sided focal fits which were associated with a low grade fever. The fits were thought to be secondary to herpes encephalitis and were treated with Acyclovir and large doses of Phenobarbitone and Phenytoin. Cardiac monitoring was normal.[324]

158. The report recounted investigations which were conducted into the cause of the ALTE. It summarised as follows:

CSF: Biochemical and cytology studies were normal. Herpes culture was negative.

Serum herpes IgM: Normal.

US scan of the brain and kidneys: Normal.

Cranial CT scan: Showed hypodense areas in the temporal and occipital lobes secondary to viral encephalitis ? demyelination disorder.

E.E.G.: Showed left frontal lobe epileptogenic foci.

Chest Xray: Showed features consistent with bronchiolitis.

Naso pharyngeal aspirate: Culture for viruses and viral antigens were negative.

Urine metabolic screen: Was negative for methylmalonic acid. Urinary organic amino acid profile, urinary amino acid pattern showed no abnormality. Urinary lactic acid was within normal range.

Serum lactate, ammonia, calcium, magnesium and glucose: Were all normal.

Rectal biopsy: Showed no neuronal inclusion bodies.

Leucocyte inclusions: Were normal.

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321 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991) p 1.
322 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991) p 1.
323 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991) p 1.
324 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991) p 2.

> *Blood metabolic screen: was negative for GM1 and GM2 gangliosidoses, and MLD; Gaucher's Krabbe's and Niemann-Pick diseases; Mannosidosis, Fucosidosis, Mucolipidoses II and III, and Mucopolysaccharidosis VII.*
>
> *Plasma screen for very long chain fatty acids and Phytanic acid was negative for ALD/AMN, Refsum's disease, Zellweger's and other generalized peroxisomopathies.*
>
> *Mucopolysaccharide screen was negative.*
>
> *Plasma carnitine values were normal.*[325]

159. The forensic pathologists observed in the report that the fits following Patrick's presentation on 18 October 1990 were stabilised with anticonvulsants and Patrick was discharged with a diagnosis of intractable seizures, probably viral encephalitis and bronchiolitis. They noted his history over the subsequent months:
    a. his presentation on 4 November 1990 with prolonged seizures resembling an oculogyric crisis and which resolved spontaneously after 90 minutes;
    b. that he had bilateral conjunctivitis and an upper respiratory tract infection ("URTI") on that presentation;
    c. the repeat CT scan showing further decrease in brain substance; the repeat EEG showing multifocal epileptogenic foci;
    d. that a further CT scan on 14 November 1990 showing "? Occipital ischaemic area with clinical visual impairment (probably cortical blindness) and developmental regression"; and
    e. a further admission on 23 December 1990 with an oculogyric crisis secondary to past encephalitic basal ganglia problem, which was provoked by a viral illness.[326]

Key findings on external examination

160. The external examination showed the body to be that of a normally formed and well-nourished male child weighing 8.57 kilograms, head circumference 44cm, crown rump length 53cm, crown heel length 77cm, and foot length 10cm. There was no external abnormality.[327]

Key findings on internal examination

161. Both lungs were congested in their posterior basal dependant segments.[328]

162. The thymus weighed 30 grams (average was 10 grams +/- 2 grams). It was enlarged.[329]

163. Investigations on post-mortem tissue included questioning Dr Bale, histopathologist at the Royal Alexandra Hospital for Children, concerning investigation of a possible cardiac conduction defect. The report records that as previous ECG monitoring showed no abnormality and arrhythmias were never noted clinically, "this was thought to be very unlikely."[330] However, tissue was kept for subsequent dissection if requested.

Final autopsy report

164. In the final report dated 2 September 1991, the clinical diagnosis of the cause of death was unchanged from the initial report:

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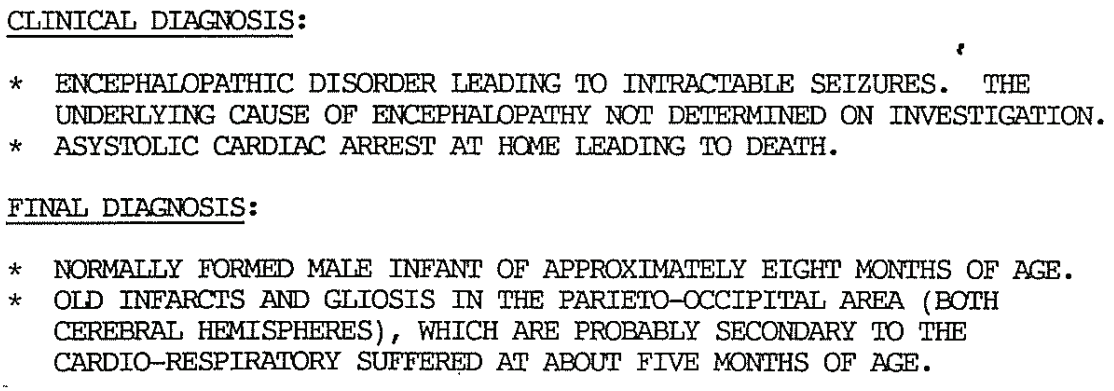
325 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991) p 2.
326 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991) p 3.
327 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991) p 3.
328 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991) p 5.
329 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991) p 5.
330 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991) p 5.



CLINICAL DIAGNOSIS:

* ENCEPHALOPATHIC DISORDER LEADING TO INTRACTABLE SEIZURES. THE UNDERLYING CAUSE OF ENCEPHALOPATHY NOT DETERMINED ON INVESTIGATION.
* ASYSTOLIC CARDIAC ARREST AT HOME LEADING TO DEATH.

FINAL DIAGNOSIS:

* NORMALLY FORMED MALE INFANT OF APPROXIMATELY EIGHT MONTHS OF AGE.
* OLD INFARCTS AND GLIOSIS IN THE PARIETO-OCCIPITAL AREA (BOTH CEREBRAL HEMISPHERES), WHICH ARE PROBABLY SECONDARY TO THE CARDIO-RESPIRATORY SUFFERED AT ABOUT FIVE MONTHS OF AGE.

Figure 13: Extract from final autopsy report in relation to Patrick: Exhibit H in the Inquiry, p 46

Macroscopic report

165. This report focused upon macroscopic findings of the brain. The brain weighed 750 grams after fixation. The gyri of both occipital lobes (visual cortex) were shrunken, thinner and more undulated than normal and the sulci were widened.[331]

166. On section, the cortical grey matter of the visual cortex in both hemispheres was thinner than normal and showed cystic degeneration. The cysts measured 1-2 mm in diameter and were present in a linear pattern at the junction of grey and white matter. Underlying white matter was firmer than normal and appeared to be expanded. Similar areas of firm white matter were present in the left front and both parietal lobes.[332]

Microscopic report

167. Again, significant findings were made in relation to Patrick's brain upon microscopic examination. They were recorded in a report dated 24 June 1991 by Dr Alex Kan from the Histopathology Department of the Royal Alexandra Hospital for Children who conducted the brain examination.

168. Dr Kan's examination showed no evidence of any neuronal storage disease or leukodystrophy.[333] The major changes were old infarcts and gliosis mostly in the form of old laminar necrosis which, in keeping with the macroscopic finding, was most severe in the parieto-occipital area.[334] The only spongy change was seen in the gliotic (proliferation or hypertrophy of glial cells) cortical scars and the subjacent white matter, in the old infarcts.[335] The cerebellar cortex was unaffected, which ruled out Canavan's disease.[336]

169. In the deeper parts of the cerebrum and in the cerebellar and brain stem nuclei there were neurones showing simple atrophy, which could have resulted from Patrick's epileptic seizures.[337]

331 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Patrick (2 September 1991) p 1.
332 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Patrick (2 September 1991) pp 1-2.
333 Exhibit H, Forensic pathology tender bundle, Letter from Dr Alex Kan to Dr Jan Bishop and Dr Gurpreet Singh-Khaira (24 June 1991).
334 Exhibit H, Forensic pathology tender bundle, Letter from Dr Alex Kan to Dr Jan Bishop and Dr Gurpreet Singh-Khaira (24 June 1991).
335 Exhibit H, Forensic pathology tender bundle, Letter from Dr Alex Kan to Dr Jan Bishop and Dr Gurpreet Singh-Khaira (24 June 1991).
336 Exhibit H, Forensic pathology tender bundle, Letter from Dr Alex Kan to Dr Jan Bishop and Dr Gurpreet Singh-Khaira (24 June 1991).
337 Exhibit H, Forensic pathology tender bundle, Letter from Dr Alex Kan to Dr Jan Bishop and Dr Gurpreet Singh-Khaira (24 June 1991).

170. There was a slight lymphocytic infiltrate in the leptomeninges (the inner two meninges), which was in addition to the small amount of residual haemopoiesis, normal in the age group.[338] It could be non-specific and related to the cortical infarcts, or "related to the treated encephalitis (? assumed or proven)."[339]

171. Dr Kan concluded that:

> *I believe that the small amount of linear cortical calcification in the occipital region is just part of the laminar cortical necrosis. I can see no suggestive features of toxoplasmosis or cytomegalovirus infection, and the distribution of the lesions is unusual for herpes simplex encephalitis and they certainly appear far more likely to be the result of the episode of cardio-respiratory arrest this baby suffered at about 5 months of age.*[340]

172. Otherwise, on microscopic examination, the lungs showed no significant abnormality apart from small foci of alveolar collapse in the periphery of the lung.[341]

173. Microscope slides of the heart, skeletal muscle, liver, spleen, thymus, pancreas, kidneys, thyroid, adrenal glands, testes, and intestine showed no abnormality other than autolysis.

174. The histology report for Patrick stated that the culture for viruses and viral antigens was negative in Patrick's nasopharyngeal aspirate.[342] However, his "post mortem blood cultures grew mixed cocci and bacilli identified as E.coli, Enterococcus faecolis and Enterococcus avium."[343] It was recorded by the forensic pathologist that:

> *these findings are not significant and probably reflect contamination. Post mortem lung tissue cultures were negative for organisms. Post mortem lung tissue cultures for viruses and mycoplasma were negative.*[344]

175. On 19 September 1991, Dr Wilkinson wrote to Mr and Ms Folbigg, stating that he had received Patrick's final autopsy report and that the examination of the brain did not suggest any inherited disorder – the changes were of a type that could occur after seizures, and encephalitis, or interference with oxygen supply.[345] Further, there was no evidence that any future children would have any definite neurological problem.[346]

Death Certificate

176. The death certificate issued by Dr Wilkinson in respect of Patrick's death recorded, as cause of death and duration of last illness:

> *(A) Asphyxia due to airway obstruction, 1 hour*
>
> *(B) Epileptic fits, 4 months.*[347]

338 Exhibit H, Forensic pathology tender bundle, Letter from Dr Alex Kan to Dr Jan Bishop and Dr Gurpreet Singh-Khaira (24 June 1991).
339 Exhibit H, Forensic pathology tender bundle, Letter from Dr Alex Kan to Dr Jan Bishop and Dr Gurpreet Singh-Khaira (24 June 1991).
340 Exhibit H, Forensic pathology tender bundle, Letter from Dr Alex Kan to Dr Jan Bishop and Dr Gurpreet Singh-Khaira (24 June 1991).
341 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Patrick (2 September 1991) p 2.
342 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991) p 2.
343 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Patrick (2 September 1991).
344 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Patrick (2 September 1991).
345 Exhibit E, trial Exhibit 6, Letter from Dr Ian Wilkinson to Craig and Kathleen Folbigg (19 September 1991).
346 Exhibit E, trial Exhibit 6, Letter from Dr Ian Wilkinson to Craig and Kathleen Folbigg (19 September 1991).
347 Exhibit H, Forensic pathology tender bundle, Death certificate of Patrick (13 February 1991).

Evidence at the time of trial

Opinions on cause of Patrick's ALTE

177. Asked at trial what he concluded, with his additional years of experience, caused Patrick's seizures, Dr Dezordi said that he had seen quite a lot of babies with asphyxia and hypoxia, and with meningitis and encephalitis, and he had no doubt that "the whole scenario… is most consistent with some catastrophic event that caused the lack of oxygen to the child's brain on the morning of October 18."[348] He did not find any medical cause for that event.[349]

178. In his evidence at trial, Dr Wilkinson "absolutely" excluded that Patrick ever had encephalitis.[350] Dr Wilkinson summarised, as the reasons that he excluded encephalitis, the three EEGs; lumbar puncture; Dr Kan's report; the timing and nature of the seizures; the loss of visual function; calcium changes shown on the 5 November 1990 CT scan; lack of fever on admission; and an antibodies test specific for herpes encephalitis.[351] He gave evidence that two further opinions about the 5 November 1990 scan were obtained: Dr John Bear, a senior paediatric radiologist, considered encephalitis a possibility but not a classic case; Professor Merl DeSilva, the most senior radiologist at Royal Alexander Hospital for Children, did not believe it to be in keeping with encephalitis.[352]

179. Dr Wilkinson gave evidence before the jury that in relation to the ALTE, it was quite possible that an epileptic seizure had caused asphyxiation by resulting in obstruction of Patrick's airway, or had caused a cardiac arrest and cerebral anoxia (lack of oxygen to the brain).[353]

180. Dr Wilkinson gave evidence that he considered "absolutely" that damage to Patrick's brain was consistent with Patrick having suffered a catastrophic asphyxiating event from unknown causes, explaining that it is very typical for a child suffering asphyxial damage to the brain to develop progressive change within the brain which produces seizures down the track.[354]

181. Having considered records and also relying on his knowledge of the tests conducted in relation to Patrick, Dr Wilkinson was of the view that the direct cause of Patrick's ALTE was a catastrophic asphyxiating event itself of an unknown cause.[355] He explained "asphyxia" to mean:

> *a situation where the end result is that the blood cannot deliver oxygen to the tissues and that may be as a result of a number of issues. It would be as a result of just obstructing the passage of air and oxygen into the lungs, it can be other situations.*[356]

182. At trial, Dr Cala gave evidence that he considered that it was "a possibility" that Patrick's ALTE was the result of him being deliberately smothered.[357] It was "a possibility" that it was the result of an acute catastrophic asphyxiating event of unknown causes.[358]

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348 10 April 2003 T505.23-42.
349 10 April 2003 T505.44-46.
350 10 April 2003 T517.15-19, T519.31-35; 23 April 2003 T859.57-860.1, T876.8-11.
351 23 April 2003 T872.44-876.11.
352 23 April 2003 T882.37-883.4.
353 10 April 2003 T511.46-512.8.
354 10 April 2003 T509.52-510.8; 23 April 2003 T874.50-875.1.
355 10 April 2003 T514.18-24; 23 April 2003 T876.15.
356 23 April 2003 T876.18-25.
357 16 April 2003 T747.2-9.
358 16 April 2003 T747.11-14.

183. Dr Beal opined that it was "[m]ost unlikely" that the ALTE resulted from an epileptic fit.[359] She considered the likely cause of the ALTE was an acute asphyxial event of undetermined origin (and, as noted below, the death was from same cause).[360] Dr Beal stated that "[i]mposed suffocation was likely to have caused this event".[361]

184. Under cross-examination, she agreed that she relied on other experts to exclude epilepsy and encephalitis in relation to the ALTE, but said she found the possibility that the ALTE was the first epileptic fit to be "extremely unlikely because epilepsy starting at that age usually shows elements, always shows some other cause."[362]

185. Dr Ophoven stated that no natural disease or process had been identified to explain the ALTE, "nor was there a recurrence of an acute life threatening event observed by anyone except his mother."[363] In relation to the ALTE, Dr Ophoven concluded that in her opinion, "the cause of Patrick's cardio-respiratory arrest is the same process that killed him" (that is, suffocation, discussed below).[364]

186. Professor Herdson opined that Patrick's ALTE was consistent with him having been deliberately or accidentally smothered.[365] The cause of the asphyxiating event that led to the ALTE could not be determined, but there are not many causes in a child of Patrick's age.[366] While epilepsy could cause a seizure leading to such an event, Professor Herdson would have expected a history of epilepsy.[367] In his transcribed answers to the model questions, Professor Herdson said "yes" to the question of whether Patrick's ALTE was consistent with deliberate smothering, and to whether it was a result of an acute catastrophic asphyxiating event.[368]

187. Professor Berry gave evidence that he thought it very unlikely that a first epileptic fit led to the severe brain damage suffered by Patrick as a consequence of the ALTE.[369] The damage to Patrick's brain, laminar cortical necrosis (at the back of the brain, which caused blindness and so on) appeared to have been consequent on the first episode.[370] Also, the blood protein and sugar in urine found shortly after Patrick's first admission was an indication of severe hypoxic injury at the time.[371]

188. In his report, referring to Patrick's ALTE, Professor Berry opined that such "nearmiss events" resulting in brain damage are a cause for concern because the window of opportunity to find the child and effect resuscitation is probably only a few minutes, which raises the question whether the person who found the baby may have been present when the collapse occurred.[372] In the transcribed answers to model questions during the trial, Professor Berry said that whether the ALTE was consistent with deliberate suffocation was a question for a clinician, but the findings in the brain at post-mortem were entirely consistent with that having been caused by a hypoxic episode.[373]

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359 5 May 2003 T1139.36-50.

360 5 May 2003 T1138.52-1140.2, T1147.35-36.

361 Responses to Crown model questions – Dr Beal, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.

362 5 May 2003 T1146.45-1147.25.

363 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 6.

364 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 10.

365 1 May 2003 T1035.35-38.

366 1 May 2003 T1042.47-1043.11.

367 1 May 2003 T1043.13-16, T1047.43-53.

368 Responses to Crown model questions – Professor Herdson, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.

369 1 May 2003 T1061.20-33, T1072.3-16.

370 1 May 2003 T1072.25-57.

371 1 May 2003 T1072.52-57.

372 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 25.

373 Responses to Crown model questions – Professor Berry, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.

189. Professor Byard opined that an epileptic seizure could not be excluded on the pathology, although it was very unusual to have this degree of damage from a first epileptic episode.[374] He considered that an epileptic episode could have resulted in a hypoxic event, which could possibly have caused the extent of the brain damage.[375]

190. In oral evidence, consistently with his first report, Professor Byard said that it was very difficult on the pathology to say what may have happened to cause Patrick's ALTE.[376] It was most likely caused by an asphyxiating event of unknown causes.[377] It was possible that Patrick was accidentally or deliberately suffocated, as the pathology findings were unspecific.[378]

191. Professor Robert Ouvrier, paediatric neurologist, gave to investigating police a report containing his opinions in relation to Patrick's ALTE and the subsequent effects upon Patrick's brain.[379] Professor Ouvrier was provided with records by police but had not seen x-rays or other pathological material.[380] He was aware that Patrick was one of four siblings who had died unexpectedly in infancy.[381] He did not give evidence at trial.

192. In his report, which was not tendered as an exhibit at trial, Professor Ouvrier concluded that the most plausible explanation for Patrick's series of events was an acute asphyxial event on 18 October 1990, which could have been a "near miss SIDS" or could have been due to deliberate suffocation.[382] Patrick appeared to have been normal at birth and well until the time of his admission upon the ALTE.[383] The clinical history and findings on that occasion, coupled with the early onset of seizures, which became intractable, "would be in keeping with an encephalopathy, due most likely… to an asphyxia episode."[384] The pattern of delayed seizures was common in ALTEs of whatever cause.[385] Subsequent evolution, with episodic tonic upgaze (upward deviation of the eyes), seizures and decrease in visual attention, and also brain atrophy seen on the second CT scan, would have been consistent with brain damage suffered during the initial event.[386] There was no supportive evidence of an underlying metabolic or degenerative disease of the brain.[387]

Opinions on cause of Patrick's death

193. Dr Wilkinson gave a statement describing his examination of Patrick during the resuscitation attempts at hospital on 13 February 1991.[388] He stated:

> [Patrick's] *appearance at the time was consistent with a patient who had suffered asphyxiation. At that time I knew that Patrick had suffered from epilepsy in the past and felt that on this occasion he could have experienced an epileptic fit which had resulted in obstruction of his airways, asphyxia with consequent cerebral anoxia and subsequent death. At that moment there appeared to be no suspicious circumstances.*[389]

374 7 May 2003 T1209.51-52, T1212.35-45, T1237.32-38, T1254.54-57.
375 7 May 2003 T1210.26-52, T1212.35-45, T1254.54-57.
376 7 May 2003 T1209.43-46, T1211.26-35; Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 5.
377 7 May 2003 T1237.57-1238.1.
378 7 May 2003 T1209.58-1210.7.
379 Exhibit H, Forensic pathology tender bundle, Report of Professor Robert Ouvrier (28 October 2002) p 2.
380 Exhibit H, Forensic pathology tender bundle, Report of Professor Robert Ouvrier (28 October 2002) p 2.
381 Exhibit H, Forensic pathology tender bundle, Report of Professor Robert Ouvrier (28 October 2002) p 2.
382 Exhibit H, Forensic pathology tender bundle, Report of Professor Robert Ouvrier (28 October 2002) p 5.
383 Exhibit H, Forensic pathology tender bundle, Report of Professor Robert Ouvrier (28 October 2002) p 4.
384 Exhibit H, Forensic pathology tender bundle, Report of Professor Robert Ouvrier (28 October 2002) p 4.
385 Exhibit H, Forensic pathology tender bundle, Report of Professor Robert Ouvrier (28 October 2002) p 4.
386 Exhibit H, Forensic pathology tender bundle, Report of Professor Robert Ouvrier (28 October 2002) p 5.
387 Exhibit H, Forensic pathology tender bundle, Report of Professor Robert Ouvrier (28 October 2002) p 5.
388 Exhibit H, Forensic pathology tender bundle, Statement of Dr Ian Wilkinson (8 October 1999) [5].
389 Exhibit H, Forensic pathology tender bundle, Statement of Dr Ian Wilkinson (8 October 1999) [5].

194. He believed that smothering could have caused asphyxiation at the time of Patrick's death.[390] Hypoxia could be caused by smothering.[391]

195. In evidence, Dr Wilkinson stated that the death certificate listed epileptic fits as having given rise to asphyxia, in the absence of other medical findings.[392] At the time, he considered that it was quite possible that an epileptic seizure, itself caused by encephalopathic disorder, had caused asphyxiation or had caused a cardiac arrest and cerebral anoxia (lack of oxygen to the brain).[393]

196. However, the post-mortem report radically changed Dr Wilkinson's mind in relation to epileptic fits being a possible cause.[394] The report showed no evidence of things that might be associated with asphyxiation from an epileptic seizure such as vomit, tongue obstruction or swollen airways.[395] At trial, Dr Wilkinson said he considered it to be possible that epileptic fits led to the asphyxiation that caused Patrick's death, but was no longer of the conviction that this was the cause.[396] In his statement, given after Sarah and Laura died, Dr Wilkinson stated while he was satisfied with his diagnosis at the time of Patrick's death, he had doubt in his mind, stating that:

> *I still believe that Patrick could have been asphyxiated but I have doubts that it was as a result of an epileptic fit. I must stress that I cannot positively rule out that an epileptic fit did cause the asphyxiation. Other causes of asphyxia must now be considered in light of the other deaths in the family.*[397]

197. Dr Beal said she would defer to Dr Kan (recorded as "Dr Cala" in the trial transcript, apparently in error), and while accepting the death could have been the result of seizure disorder, would have diagnosed "undetermined".[398] She said it was "certainly" consistent with deliberate smothering, and as a result of an acute catastrophic asphyxiating event of unknown causes.[399]

198. Professor Herdson would have attributed the death "at least" as undetermined, because the history of the ALTE with subsequent abnormalities would be most unusual for a death to be due to "so-called" SIDS.[400] He would not have diagnosed SIDS as having caused Patrick's death, due to Patrick's catastrophic medical history.[401] In this, his opinion at trial and in his report appears inconsistent with an answer recorded as having been given by him to the model questions, which was that he would have diagnosed SIDS.[402] This record would appear to contain an error. Professor Herdson opined that the death was consistent with having been caused by smothering.[403] As noted, he also opined that a seizure could have caused the death but it was highly unlikely.[404] He concurred with analyses by Professor Berry and Dr Ophoven.[405]

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390 10 April 2003 T514.51-54, T516.46-49.

391 23 April 2003 T883.13-14.

392 10 April 2003 T512.10-14; Exhibit H, Forensic pathology tender bundle, Medical certificate of Patrick's cause of death (14 February 1991).

393 10 April 2003 T511.46-512.8; 23 April 2003 T863.9-35, T865.1-8, T881.57-882.3. Dr Wilkinson explained encephalopathic disorder to be a disorder in which there is some abnormality within the brain.

394 23 April 2003 T865.5-8; Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991).

395 10 April 2003 T511.38-44; 23 April 2003 T865.25-33, T872.1-16, T881.13-34; Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991).

396 10 April 2003 T517.1-5; 23 April 2003.

397 Exhibit H, Forensic pathology tender bundle, Statement of Dr Ian Wilkinson (8 October 1999) [7].

398 5 May 2003 T1147.30-46; Responses to Crown model questions – Dr Beal, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.

399 Responses to Crown model questions – Dr Beal, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.

400 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Herdson (17 January 2002) p 2.

401 1 May 2003 T1035.46-1036.2.

402 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Herdson (17 January 2002) p 2.

403 1 May 2003 T1036.4-6.

404 1 May 2003 T1044.1-10, T1048.31-52.

405 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Herdson (17 January 2002) p 2.

199. In answers to the model questions, Professor Berry would have diagnosed Patrick's death in isolation as "not ascertained", ascribing it to brain damage following an unexplained collapse, and also noting his mother found him on both occasions. The death was consistent with deliberate smothering.[406] Professor Berry said that children with severe epilepsy do die suddenly.[407] He said that Patrick's death was probably attributable to the first event, but possibly a later asphyxiating event.[408] Professor Berry gave evidence that suffocation causing the kind of severe brain damage suffered by Patrick would take a few minutes.[409]

200. In Professor Byard's first report, because Patrick's autopsy findings could not be taken in isolation, he stated he would list Patrick's death as "undetermined, cannot exclude epilepsy."[410] In isolation, he would have attributed death to epilepsy against a background of possible encephalitis.[411] He deferred to Drs Dezordi and Wilkinson in relation to encephalitis. There was no finding or symptom that could amount to proof of suffocation in the ALTE or the death.[412]

201. In oral evidence, Professor Byard explained that when a person dies from epilepsy, the pathological findings are very non-specific.[413] People who die of epilepsy do not always have external signs to indicate there had been a fit (such as unusual hand position).[414] Whilst unusual, it was possible to understand Patrick's death in terms of seizure against a background of seizure disorder, such a seizure having caused a catastrophic asphyxiating event.[415]

202. In his first report, Professor Byard noted that the frequency of sudden unexpected death in epilepsy ("SUDEP") in children is unknown, however, in general epileptic populations estimates have ranged from one in 200 to one in 680 patients.[416] A typical SUDEP case is of an epileptic child, often with mental retardation, found dead in bed with minimal external or internal findings.[417] The association with sleep most likely related to a reduction in seizure threshold and increase in epileptic discharges.[418]

203. Professor Busuttil opined that Patrick's death should not have been attributed to SIDS, nor to asphyxia in the absence of typical asphyxial signs at autopsy.[419] No features of mechanical asphyxia (e.g. petechial haemorrhages) were recorded.[420]

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406 Responses to Crown model questions – Professor Berry, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.

407 Responses to Crown model questions – Professor Berry, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.

408 Responses to Crown model questions – Professor Berry, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.

409 1 May 2003 T1061.1-18.

410 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 7.

411 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 5.

412 7 May 2003 T1215.21-30.

413 7 May 2003 T1214.6-32.

414 7 May 2003 T1255.20-25, T1255.50-58.

415 7 May 2003 T1214.41-1215.3.

416 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 5.

417 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 5.

418 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 5.

419 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 11.

420 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 11.

204. Professor Busuttil considered that Patrick's brain condition could have given rise to serious convulsions causing death.[421] Given his medical history, an epileptic fit could have resulted in sudden damage to the vital brain stem with cessation of breathing and heart function, or a secondary effect on the heart.[422] This would not have produced changes which could specifically be identified at autopsy, except perhaps by damage to Patrick's lips or tongue in the course of the convulsion.[423] While blood levels of anticonvulsants at autopsy were within therapeutic range, this did not mean convulsions were fully controlled.[424]

205. In oral evidence at trial, Dr Singh-Khaira agreed that, essentially, he was not able to find a cause of Patrick's death.[425] In the absence of other causes, a catastrophic asphyxiating event could have been a cause.[426] At the time of the post-mortem, he was aware of the previous death of Caleb, and he looked for signs of manual asphyxia, such as petechia and changes in the airways but did not find any.[427] He saw nothing in the brain that could account for Patrick's death such as new injury or damage or deterioration.[428] Dr Singh-Khaira did not exclude the possibility that a seizure led to asphyxiation and ultimately cardiac arrest.[429]

206. Dr Singh-Khaira and Dr Bishop excluded infective disorders, metabolic disorders that they could think of, and genetic disorders – none of them were a positive indicator as to cause of death.[430]

207. Dr Cala would have given the cause of Patrick's death as "undetermined".[431]

208. Dr Beal considered that the ALTE and death probably had the same cause; the likely cause of the ALTE was an acute asphyxial event of undetermined origin and the death was most likely caused by some unexplained asphyxiating event which was "most certain[ly]" consistent with deliberate suffocation.[432]

209. In her first report, Dr Ophoven opined to "a reasonable degree of medical certainty" that the cause of Patrick's death should be listed as suffocation, and the manner of death as homicide.[433] She stated that Patrick's brain damage was consistent with a hypoxic episode (synonymous in this case with asphyxia) and that it "unfortunately heralds the fatal event in retrospect."[434] Facts that led to her conclusion included:

    a. the autopsy failed to identify any known natural disease or disease process that could explain the death. Patrick was growing and developing normally for his age and circumstance and, despite his handicaps, was advancing well;

    b. the autopsy findings were consistent with death by suffocation;

    c. Patrick's death was not consistent with a seizure or the presence of a seizure disorder;

    d. Patrick was in the care of Ms Folbigg at his time of death, and she was the last person to see him alive (as were all of the children);

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421 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 11.
422 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) pp 10-11.
423 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 11.
424 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 11.
425 11 April 2003 T560.56-58.
426 11 April 2003 T560.43-48.
427 11 April 2003 T561.7-49.
428 11 April 2003 T560.36-41.
429 11 April 2003 T562.50-563.7.
430 11 April 2003 T559.46-52.
431 16 April 2003 T747.16-21.
432 5 May 2003 T1138.52-1140.2.
433 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 6.
434 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 6.

e. there was an absence of risk factors commonly recognised in the epidemiology of SIDS, no history of infant apnoea or significant breathing problems, no evidence of hyperthermia, no recent history of illness; and

f. none of the deaths could be attributed to SIDS.

210. Professor Ouvrier opined that the "final event appears to have been a further asphyxial episode without clear explanation."[435] The pathological findings at autopsy would have been consistent with damage due to a serious hypoxic event suffered at the time of the ALTE, but he could not exclude the possibility that the findings could have been caused by shaking or trauma, which can sometimes cause apnoea.[436]

211. Dr Hawker, consultant paediatric cardiologist, examined "Medi Traces" from Patrick's medical records and electrocardiograph recordings.[437] He could not interpret the Medi Trace records because of their poor quality.[438] The electrocardiographs for Patrick showed no abnormalities (qualified in that the electrocardiographs for Patrick were not complete).

Evidence in the Inquiry

SIDS risk factors

212. In her report, Professor Horne, infant sleep and SIDS specialist, referred to the following "potential protective factors for SIDS" in respect of Patrick prior to his ALTE: Patrick was born at term at a normal weight, was found supine with his face uncovered and in his own bed, was vaccinated and used a dummy. She referred to an increased risk in mothers younger than 26 years at the time of the baby's birth (Ms Folbigg was 22 when Patrick was born) and paternal smoking.[439]

213. Taking the above into account, Professor Horne gave evidence in the Inquiry that she considered Patrick to be at a low risk for an ALTE.[440] Professor Elder noted that if Caleb's death presented a risk to Patrick, this was the reason that investigations were undertaken in relation to Patrick, and these were in turn shown to be normal.[441]

214. Because of the damage Patrick suffered after the ALTE, it was not possible to apply SIDS risk factors to Patrick at the time of his death.[442] However, Professor Elder noted that he did not have significant factors for sudden infant death – he was not in an unsafe position when he was found and his mother did not smoke.[443]

Encephalitis

215. Professor Cordner accepted that he was not in any position to take issue with Dr Wilkinson's evidence in relation to herpes simplex encephalitis and that it is most unlikely that this was ever present.[444] No forensic pathologist in the Inquiry challenged the proposition that encephalitis was excluded.[445]

216. Professor Blackwell, in oral evidence, agreed that although herpes simplex encephalitis was targeted in Patrick's initial treatment following the ALTE, encephalitis was ultimately excluded.[446]

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435 Exhibit H, Forensic pathology tender bundle, Report of Professor Robert Ouvrier (28 October 2002) p 5.
436 Exhibit H, Forensic pathology tender bundle, Report of Professor Robert Ouvrier (28 October 2002) p 5.
437 Exhibit H, Forensic pathology tender bundle, Statement of Dr Richard Hawker (6 March 2003) p 2.
438 Exhibit H, Forensic pathology tender bundle, Statement of Dr Richard Hawker (6 March 2003) p 2.
439 Exhibit J, Expert report of Professor Rosemary Horne (10 February 2019) pp 2-3.
440 Transcript of the Inquiry, 18 March 2019 T36.2-10.
441 Transcript of the Inquiry, 18 March 2019 T37.8-19.
442 Transcript of the Inquiry, 18 March 2019 T37.21-36.
443 Transcript of the Inquiry, 18 March 2019 T37.1.
444 Exhibit Q, Report of Professor Stephen Cordner (undated) p 67.
445 Transcript of the Inquiry, 21 March 2019 T278.40-279.3.
446 Transcript of the Inquiry, 22 March T340.16-21.

217. Compatibly with this evidence, Professor Monique Ryan, paediatric neurologist and Director of Neurology at the Royal Children's Hospital in Victoria, noted that the cerebral spinal fluid and CT findings, and later the post-mortem examination, were not consistent in Patrick's case with an encephalitis affecting the basal ganglia.[447] Associate Professor Michael Fahey, paediatric neurologist, clinical geneticist and Director of Neurology at the Monash Children's Hospital in Victoria, agreed.[448] Evidence of the expert neurologists on other features of clinical findings in relation to Patrick is discussed in detail below.

Opinions on cause of Patrick's ALTE – forensic pathology

218. All of the forensic pathologists who gave evidence in the Inquiry opined that the cause of the ALTE was unknown or unexplained.[449]

219. Professor Cordner referred to Patrick's ALTE as being thought of as "SIDS/SUDI-like in character", on the basis that a quarter to a half of ALTEs are not explained and that these "belong in the same basket as SIDS/SUDI".[450] Professor Pollanen referred to the finding of old laminar necrosis in the cerebral cortex and history, and stated that this was a "straightforward clinicopathologic correlation that can explain death" (i.e. SUDEP).[451] He classified Patrick's death as a "Class 3"(see [62] above).

220. Dr Cala also stated that many ALTEs are never satisfactorily explained, and there are many underlying causes.[452] The underlying cause for Patrick's ALTE was never known, and but for the ALTE it appeared that he was normal and progressing well.[453] Dr Cala stated that while he could not definitely prove smothering, he was concerned that smothering could explain the ALTE and also Patrick's sudden death.[454] He noted that soon after the ALTE, Patrick exhibited signs of respiratory difficulties, and that this is a most unusual finding following a seizure – the postictal period usually results in intense drowsiness and a period of sleep.[455] If the airway is not compromised in some way, there should be no evidence after a seizure of respiratory difficulty.[456]

221. Professor Hilton, however, opined that to take Patrick's constellation of central nervous system disabilities, structural and/or functional, as necessarily being caused by non-natural events could not be justified, and to ascribe responsibility for Patrick's brain damage to "epilepsy", or the converse, must remain a moot point.[457]

222. Professor Duflou did not identify a specific cause of Patrick's ALTE. He referred to normal investigations of Patrick after birth. In that regard he noted a barium swallow which suggested uncoordinated swallowing. Dr Cala referred to the same test as showing no gastro oesophageal reflux.[458] Professor Duflou also referred to, as relevant to Patrick's ALTE but not identifying as a specific cause, possible bronchiolitis when Patrick was admitted with the ALTE; the discharge diagnosis (see above at [136]); deterioration of Patrick's neurological function after his discharge; and that there are a number of differential diagnoses of an ALTE such as reflex causes.[459]

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447 Exhibit AJ, Report of Professor Monique Ryan (15 March 2019) p 15.

448 Exhibit AK, Report of Associate Professor Michael Fahey (30 March 2019) p 15.

449 Transcript of the Inquiry, 20 March 2019 T146.1-T147.23; Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 41; Exhibit Q, Report of Professor Stephen Cordner (undated) pp 60, 90.

450 Exhibit Q, Report of Professor Stephen Cordner (undated) pp 32-33.

451 Exhibit C, Report of Professor Michael Pollanen (1 June 2015) p 5.

452 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 10.

453 Exhibit M, Report of Dr Allan Cala (26 November 2018) pp 7, 10; Transcript of the Inquiry, 20 March 2019 T152.13-30.

454 Exhibit M, Report of Dr Allan Cala (26 November 2018) pp 10-11; Transcript of the Inquiry, 20 March 2019 T165.23-24.

455 The post-ictal period is "the abnormal condition occurring between the end of an epileptic seizure and return to baseline condition": Robert S Fisher and Jerome J Engel Jr, 'Definition of the Postictal State: When Does it Start and End?' (2010) 19(2) *Epilepsy & Behaviour* 100, 100.

456 Exhibit M, Report of Dr Allan Cala (26 November 2018) pp 10-11.

457 Exhibit O, Report of Professor John Hilton (22 January 2019) p 2.

458 Exhibit L, Report of Johan Duflou (13 February 2019) p 30; Exhibit M, Report of Dr Allan Cala (26 November 2018) p 10.

459 Exhibit L, Report of Johan Duflou (13 February 2019) pp 30-32.

Opinions on cause of Patrick's ALTE – neurology

223. Specialist paediatric neurology evidence was commissioned and received in the Inquiry in view of Patrick's symptoms and presentation related to the ALTE. First, Professor Ryan was briefed by Ms Folbigg's legal representatives, to give an opinion in relation to Patrick's ALTE and also his death. At the time of preparing her report Professor Ryan had received some but not all of the clinical records concerning Patrick. Secondly, Associate Professor Fahey was engaged by the Inquiry.

224. On the basis of material available to her, Professor Ryan opined that she was

> *not convinced that Patrick's clinical history is consistent with him having neurologic deficits resulting from a single hypoxic-ischaemic episode on October 18, 1990.*[460]

225. In summary, and discussed in further detail below, the basis for this opinion was variability in Patrick's presentation from the time he presented at the hospital.

226. This opinion was significant. If Patrick did not experience a single hypoxicischaemic episode (brain cell damage due to lack of oxygen from restricted blood flow) on 18 October 1990, it would follow that his presentation afterwards was not consistent with him having been the subject of an asphyxiating event on that date.

227. Professor Ryan identified alternative diagnoses potentially causative of Patrick's ALTE, and death, referring to a number of conditions possibly associated with epilepsy and fluctuating neurologic symptoms which were not excluded by previous testing.

Alternative genetic diagnoses – Patrick's ALTE and death

228. The alternative diagnoses identified by Professor Ryan as potentially causative of Patrick's ALTE, and death, included disorders of creatine metabolism, alternating hemiplegia of childhood, neurotransmitter disorders and genetic channelopathies causing infantile encephalopathies and cardiac arrhythmias.[461] She stated in her report that further testing for these conditions would be best accomplished by whole genome sequencing ("WGS").[462] Importantly, in offering an opinion as to these potentially causative alternative diagnoses, Professor Ryan did not distinguish between known or recognised genetic disorders which could be identified through WGS, and as yet unknown or unrecognised genetic disorders which could not.

229. The WGS that was undertaken by the Inquiry is set out in detail in Chapter 7. Specifically in relation to potential genetic causes of Patrick's presentation, Associate Professor Fahey provided to Dr Buckley a list of 204 genes known to be associated with abnormal creatine metabolism, alternating hemiplegia of childhood, neurotransmitter disorders and genetic channelopathies causing infantile encephalopathies and cardiac arrhythmias. Associate Professor Fahey's list took into account the conditions mentioned in Professor Ryan's report, as well as other relevant genetic variants.[463]

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460 Transcript of the Inquiry, 17 April 2019 T585.11-17; Exhibit AJ, Report of Professor Monique Ryan (15 March 2019) p 14.

461 Exhibit AJ, Report of Professor Monique Ryan (15 March 2019) pp 13, 15; Transcript of the Inquiry, 17 April 2019 T584.39.

462 Exhibit AJ, Report of Professor Monique Ryan (15 March 2019) pp 13, 15; Transcript of the Inquiry, 17 April 2019 T583.1-8.

463 Exhibit AK, Report of Associate Professor Michael Fahey (30 March 2019) p 4; Transcript of the Inquiry, 17 April 2019 T588.15-30.

230. No relevant pathogenic genetic mutation was found. When taken together with the hypothesis-free analysis undertaken by the Sydney genetics team, Associate Professor Fahey considered that the genetic investigations in relation to Patrick, his siblings and mother for an alternative diagnosis for a single hypoxic episode as raised by Professor Ryan were "comprehensive".[464] He opined that all recognised genetic conditions are now excluded as the cause of Patrick's ALTE and death.[465]

231. Professor Ryan agreed with the scope of the testing undertaken, and with Associate Professor Fahey's conclusion as to the results.[466] However, as an example of a "potential alternative" unidentified genetic cause for Patrick's presentation on 18 October 1990 and his subsequent course and associated findings, Professor Ryan referred to children with a clinical presentation consistent with Dravet syndrome, but without the recognised associated genetic mutation (SCN1A). Dravet syndrome involves prolonged seizures in the context of fever (especially significant fever according to Professor Ryan),[467] resulting in hypoxicischaemic brain injuries of the sort seen on Patrick's brain post-mortem.[468]

232. Associate Professor Fahey accepted the possibility that in the future a genetic cause of Patrick's presentation, unrecognised as at April 2019, may become recognised.[469] He agreed that "the seizure threshold is lowered by having a fever".[470] He also agreed there are other as yet unrecognised genetic causes associated with the Dravet syndrome clinical presentation.[471] He emphasised however, most significantly, that the presentation necessarily involved being hypoxic on presentation, which was not otherwise accepted by Professor Ryan, and also that such presentation typically presents with fitting movements of the body, which were not observed in Patrick on 18 October 1990.[472]

Single hypoxic-ischaemic episode or degenerative condition

233. Notwithstanding the results of the genetic testing, there was a deal of evidence in the Inquiry, primarily centred around expert neurological interpretation of clinical and post-mortem presentations and findings, concerning whether Patrick suffered a degenerative neurological condition to explain the ALTE and his symptoms afterwards.

234. As stated above, underpinning Professor Ryan's opinion doubting whether Patrick's presentation on 18 October 1990 was consistent with a single hypoxic-ischaemic episode was evidence of variability in his presentation from that time and also that she did not accept that beforehand he was developing typically and otherwise well.[473]

Patrick's health before the ALTE

235. In relation to Patrick's health before the ALTE, Professor Ryan pointed specifically to him having had torticollis (the head turned and tucked down towards a shoulder),[474] which she accepted can be a benign phenomenon, and to Ms Folbigg's description to medical staff at the hospital of Patrick having always tended to arch his back at times. Professor Ryan said she did not know what to make of those factors, but they suggested a possibility that he was not entirely normal prior to 18 October 1990.[475]

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464 Exhibit AK, Report of Associate Professor Michael Fahey (30 March 2019) pp 4, 16; Transcript of the Inquiry, 17 April 2019 T589.28-39.
465 Exhibit AK, Report of Associate Professor Michael Fahey (30 March 2019) pp 4, 16; Transcript of the Inquiry, 17 April 2019 T588.24.
466 Transcript of the Inquiry, 17 April 2019 T583.6-43.
467 Transcript of the Inquiry, 17 April 2019 T604.5-19, T604.47-605.1.
468 Transcript of the Inquiry, 17 April 2019 T604.11-13.
469 Transcript of the Inquiry, 17 April 2019 T589.16-590.10.
470 Transcript of the Inquiry, 17 April 2019 T605.5-6.
471 Transcript of the Inquiry, 17 April 2019 T605.7-8.
472 Transcript of the Inquiry, 17 April 2019 T604.24-30, T605.14-24.
473 Transcript of the Inquiry, 17 April 2019 T586.20-46.
474 Transcript of the Inquiry, 17 April 2019 T259.27-29.
475 Transcript of the Inquiry, 17 April 2019 T586.40-46.

236. Within their area of expertise, both Dr Cala and Professor Hilton observed in their oral evidence that there was no indication of a degenerative neurological condition developing prior to 18 October 1990.[476] It was put to them that they would not speculate whether Patrick's encephalopathic disorder developed on or prior to that date. Dr Cala said there was, from medical records and ambulance reports, no information that Patrick was anything other than a well and normal child leading up to whatever caused the ALTE.[477] There did not appear to him be any evidence of a chronic degenerative condition other than the infarcts and gliosis that were evident on brain examination after Patrick's death, which were not part of a chronic degenerative process.[478] Dr Cala said that if a chronic neurological condition triggered some epileptiform type disorder, he would expect to see some pathological sign for the underlying degenerative condition on the EEGs (after the ALTE).[479]

237. No other expert took issue with the proposition that Patrick was a healthy and normally developing baby prior to the ALTE. Dr Colley observed that torticollis is a not uncommon condition which is "often quite benign" and on its own does not make a diagnosis of a neurogenetic condition. She also observed that back arching is a common behaviour in healthy children when irritable. She opined it was hard in retrospect to know whether this was really relevant, and it would be more relevant if a treating practitioner had recorded prior to 18 October 1990 that Patrick was back arching.[480]

Patrick's presentation after the ALTE

238. The greater proportion of the evidence on this topic focussed on Patrick's presentation on and after 18 October 1990 and the findings on clinical investigations and post-mortem.

239. Dr Cala observed that Patrick was extensively investigated and no cause of the ALTE was found.[481] He thought a degenerative neurological condition was highly unlikely, but would defer to a paediatric neurologist.[482] He did not know whether the ALTE could have been caused by an epileptic fit.[483] Professor Duflou said the absence of a specific indicator for epilepsy on examination of the brain did not exclude the possibility.[484] Further, in about 25 per cent of ALTEs, the cause is never identified.[485] All the pathologists agreed that the ALTE could have been caused by a great number of disorders, including neurological disorders.[486] Professor Cordner did not rule out the possibility of an epileptic seizure causing the ALTE, but deferred to a paediatric neurologist.[487]

240. Features of Patrick's presentation at the Mater Hospital are discussed above from [119]. Further observations that were relevant to Professor Ryan's opinion, include that:

    a. when Patrick was first brought to the Mater Hospital, he was pale and lethargic, but had some back arching. He was hypothermic, tachycardic (rapid heartbeat) and tachypnoeic (rapid breathing);

    b. on the same day, however, a head ultrasound and EEG were normal; and

    c. within a few hours of admission Patrick was described in nursing notes as feeding well.[488]

476 Transcript of the Inquiry, 21 March 2019 T269.47-270.19.
477 Transcript of the Inquiry, 21 March 2019 T268.31-42.
478 Transcript of the Inquiry, 21 March 2019 T269.3-9.
479 Transcript of the Inquiry, 21 March 2019 T269.18-35.
480 Transcript of the Inquiry, 17 April 2019 T591.35-50.
481 Transcript of the Inquiry, 21 March 2019 T278.35-38.
482 Transcript of the Inquiry, 21 March 2019 T269.47-270.9.
483 Transcript of the Inquiry, 21 March 2019 T270.39.
484 Transcript of the Inquiry, 21 March 2019 T270.41-43.
485 Transcript of the Inquiry, 21 March 2019 T279.42-43.
486 Transcript of the Inquiry, 21 March 2019 T262.41-263.6.
487 Transcript of the Inquiry, 21 March 2019 T271.7-19.
488 Exhibit AJ, Report of Professor Monique Ryan (15 March 2019) p 14.

241. Professor Ryan stated that had Patrick sustained a severe hypoxic-ischaemic insult on the morning of 18 October 1990 – one sufficiently severe to cause the changes seen on his subsequent imaging and his post-mortem examination – it was difficult to imagine that he would have been able to feed well that day, and that his EEG could have been entirely normal.[489]

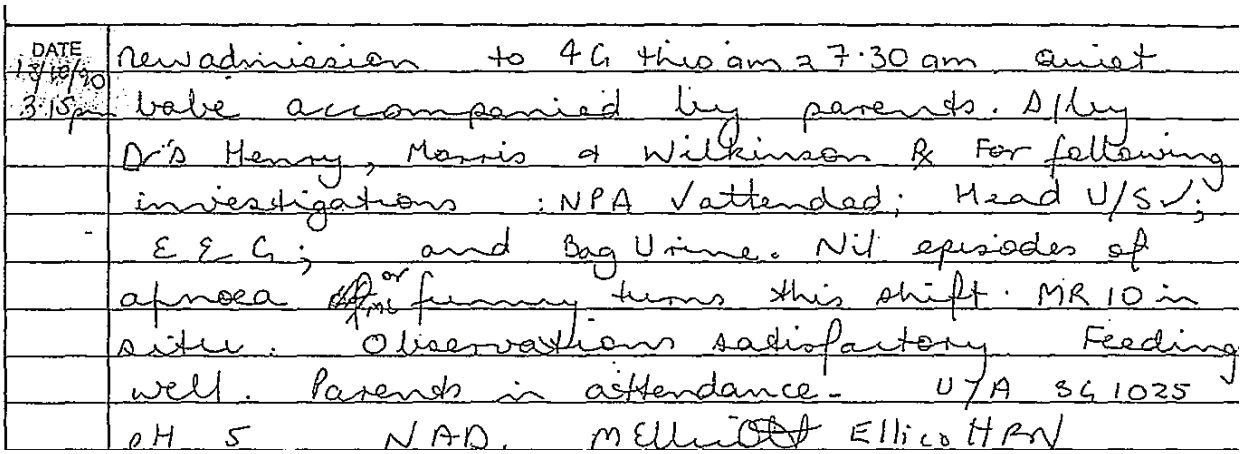
DATE
18/10/90
3.15pm
New admission to 4G this am at 7.30 am quiet
babe accompanied by parents. D/by
Dr's Henry, Morris & Wilkinson Rx For following
investigations: NPA ✓attended; Head U/S ✓;
E E G; and Bag Urine. Nil episodes of
apnoea or funny turns this shift. MR 10 in
situ. Observations satisfactory. Feeding
well. Parents in attendance. U/A SG 1025
pH 5 NAD. M Elliott Ellico HRN

Figure 14: Clinical note, Mater Hospital, 18 October 1990: Exhibit S in the Inquiry, p 531

242. In relation to Patrick's clinical presentations after 18 October 1990, in summary Professor Ryan also stated that:

a. A repeat EEG on 22 October 1990 showed no slowing of the background, which might be expected with a hypoxic encephalopathy, but did show left frontal focal changes. Dr Wilkinson reported that he could not find any neurological problem relative to that admission. During the admission, despite the development of a refractory seizure disorder, Patrick was not presenting like a child who had sustained a significant hypoxic-ischaemic insult.

b. Patrick's admission on 4 November 1990 may have been due to a seizure but might also have been an oculogyric crisis (in which there is tonic gaze deviation for long periods), an episode consisting of tonic upward eye deviation lasting an hour without other features of an epileptic seizure. Oculogyric crises are rare but can reflect an underlying genetic disorder.

c. A physiotherapy assessment on 14 January 1991 documented no fixed severe abnormalities of tone or reflexes such as would be expected after a significant hypoxic-ischaemic brain injury. The major finding was visual loss, and isolated visual loss is not common after hypoxic brain injury. Less than one month later, a physiotherapy review felt Patrick's vision to be much better, suggesting a fluctuating picture, potentially more consistent with a metabolic or other encephalopathy rather than a fixed neurologic deficit related to a static hypoxic-ischaemic injury sustained some months earlier.[490]

243. According to Associate Professor Fahey, the variability in Patrick's presentation warranted consideration.[491] However, in contrast to Professor Ryan, he concluded that Patrick's presentation and his pathology at post-mortem were consistent with a severe hypoxic event on 18 October 1990.[492] He noted that no alternative diagnosis was found.[493]

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489 Exhibit AJ, Report of Professor Monique Ryan (15 March 2019) p 14; Transcript of the Inquiry, 17 April 2019 T603.11-28.

490 Exhibit AJ, Report of Professor Monique Ryan (15 March 2019) pp 14-15.

491 Exhibit AK, Report of Associate Professor Michael Fahey (30 March 2019) p 12.

492 Transcript of the Inquiry, 17 April 2019 T595.27-31,T593.48-594.12.

493 Transcript of the Inquiry, 17 April 2019 T595.27-31.

244. Associate Professor Fahey considered Professor Ryan's report but in his own report referred to further material which he considered to be relevant, summarised as follows:[494]

    a. Patrick's oxygen saturation of 88 per cent on presentation at the Mater Hospital, and that Patrick was poorly responsive to painful stimuli and had glycosuria;

    b. Dr Wilkinson's oral evidence at trial that it was:

> [Q]*uite common in asphyxiation to find that there's effectively a honeymoon period that the child is brought in and there is a period of hours or days when there seems to be recovery and no major neurological problem, and subsequently they develop particularly seizures.*[495]

    c. Dr Dezordi's oral evidence at trial that:

> *objectively, there is no doubt that Patrick was hypoxic when he came in, because the blood saturation tests proved that. There was no question that he was hypoxic.*[496]

245. Professor Ryan was not briefed with Dr Wilkinson's or Dr Dezordi's oral evidence from the trial when she prepared her report but was provided with these prior to giving evidence in the Inquiry.[497]

246. Associate Professor Fahey and Dr Colley agreed that Patrick was hypoxic on the basis of the 88 per cent oxygen saturation reading.[498] Professor Ryan, however, did not accept this because an oximetry probe, as was used, can "misread" blood oxygen levels and also because the reading was "low but not terribly low".[499]

247. In oral evidence in the Inquiry on this point, Associate Professor Fahey referred additionally to:

    a. the ambulance officer's observations that Patrick had poor respiratory effect, with a reduced drive to take breaths. Associate Professor Fahey said that this can signify the cause being related to the brain rather than anywhere else, such as a cardiac condition or obstruction of the airway;[500] and

    b. literature concerning presentation of children with hypoxia, identifying other instances of seizures beginning after initial presentation followed by "a striking interval of near normality before neurological deterioration" with an evolution of the seizure disorder in some instances over days.[501]

248. Associate Professor Fahey gave evidence in the Inquiry that he was "satisfied from [the literature] that this was a possibility after hypoxia and that it had been reported, in fact remarkably similar to how Patrick presented."[502] He also noted that the changes seen in Patrick's brain on the CT scans proximate to his presentation on 18 October 1990, most likely represented hypoxia-ischaemia changes, given what was observed at post-mortem.[503] Professor Kirk, in his oral evidence, agreed that the post-mortem pathology was consistent with a hypoxic event.[504]

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494 Exhibit AK, Report of Associate Professor Michael Fahey (30 March 2019) p 8; Transcript of the Inquiry, 17 April 2019 T598.14-18.
495 23 April 2003 T874.53-58.
496 9 April 2003 T452.10-22.
497 Exhibit AJ, Report of Professor Monique Ryan (15 March 2019) Letter of instruction.
498 Transcript of the Inquiry, 17 April 2019 T592.5-29.
499 Transcript of the Inquiry, 17 April 2019 T585.34-T586.2.
500 Transcript of the Inquiry, 17 April 2019 T592.18-25, T602.44-48.
501 Exhibit AK, Report of Associate Professor Michael Fahey (30 March 2019) p 13; Transcript of the Inquiry, 17 April 2019 T593.13-15.
502 Transcript of the Inquiry, 17 April 2019 T593.21-22.
503 Exhibit AK, Report of Associate Professor Michael Fahey (30 March 2019) p 15; Transcript of the Inquiry, 17 April 2019 T590.34-591.5.
504 Transcript of the Inquiry, 17 April 2019 T600.49-50.

249. Associate Professor Fahey explained further, that he found it "very difficult to... walk away from" the post-mortem pathology findings of brain damage, which indicated there had been ischaemia (reduced blood flow) at some stage, and there having been a sentinel event on 18 October 1990 with emerging CT changes from that time. Associate Professor Fahey considered there was no potential for brain abnormality other than ischaemic damage.[505] Further, Patrick had had no other subsequent seizures associated with a period of hypoxia.[506]

250. Associate Professor Fahey affirmed Dr Cala's reservations about attributing Patrick's ALTE, and subsequent death, to a degenerative neurological condition.[507] He did not accept that Patrick had a "deteriorating" or "progressive" condition, preferring the term "evolving" and saying that "we're not finding anything suggestive, either pathologically, biochemically, or genomically, that suggests that he had a, an underlying progressive disease."[508] Associate Professor Fahey also noted that the changes seen in Patrick's brain pathology were "old… not… active, not changes where the cells would deteriorate."[509]

251. Professor Ryan accepted as a possibility, but not as a reasonable possibility, that Patrick did have a single hypoxic-ischaemic episode on 18 October 1990.[510] Even after having received the geneticists' reports, Patrick's additional clinical information and hearing Associate Professor's Fahey's, Dr Colley's and Professor Kirk's opinions about Patrick's hypoxic presentation and his post-mortem brain pathology, Professor Ryan maintained there was nothing that caused her to change her opinion "at all".[511] In adhering to her original opinion, Professor Ryan relied on the following matters, in addition to Patrick's variability of presentation:[512]

    a. the possibility of an alternative diagnosis of an unknown genetic cause, as yet unrecognised by the field of genetics;[513]

    b. a suggestion, by reference to a paper to which Associate Professor Fahey referred, that when children or adults have a severe hypoxic-ischaemic injury, there is evidence of other organ injury after the fact, such as kidney failure;[514] and

    c. disagreement with a suggestion that with ALTEs and epilepsy there always has to be a family history, referring to instances in the literature of children presenting with a first presentation seizure and sustaining a significant hypoxic-ischaemic insult.[515] (Associate Professor Fahey in his report referred to literature which made the point that all the infants in a study with both ALTE and epilepsy had a family history.)[516]

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505 Transcript of the Inquiry, 17 April 2019 T603.45-50.

506 Transcript of the Inquiry, 17 April 2019 T606.14-15.

507 Transcript of the Inquiry, 17 April 2019 T595.47-50.

508 Transcript of the Inquiry, 17 April 2019 T608.29-46.

509 Transcript of the Inquiry, 17 April 2019 T608.44-45.

510 Transcript of the Inquiry, 17 April 2019 T587.12-21.

511 Transcript of the Inquiry, 17 April 2019 T587.17-21; T597.34-598.5.

512 Transcript of the Inquiry, 17 April 2019 T587.35-41.

513 Transcript of the Inquiry, 17 April 2019 T584.44-47; T587.17-21.

514 Transcript of the Inquiry, 17 April 2019 T599.17-600.16; J E Constantinou et al 'Hypoxic-Ischaemic Encephalopathy after Near Miss Sudden Infant Death Syndrome (1989) 64 *Archives of Disease in Childhood* 703.

515 Transcript of the Inquiry, 17 April 2019 T597.41-598.1.

516 Exhibit AK, Report of Associate Professor Michael Fahey (30 March 2019) p 13, citing J L *Bonkowsky et al, 'Seizures in Children Following an Apparent Life-Threatening Event' (2009) 24(6) Journal of Child Neurology 709 and F J DiMario, 'Apparent Life-Threatening Events: So What Happens Next?' (2008) 122(1) Pediatrics 190; Transcript of the Inquiry, 17 April 2019 T593.24-32.*

252. In relation to the suggestion of organ injury ordinarily following severe hypoxicischaemic injury, Associate Professor Fahey noted that the paper to which he referred had an inclusion criteria of "or" – not "and" – in respect of organ failure.[517] Additionally, the paper included "people just like Patrick".[518] Professor Kirk did associate organ damage with hypoxic events, but this was based on his experience with newborn babies, as there are special circumstances at the time of birth that do not apply to four month old children.[519]

253. In respect of Professor Ryan's possible alternative diagnosis of unrecognised first epileptic seizure on presentation,[520] Associate Professor Fahey additionally observed that if this was the case, the first seizure was very different from any other seizure that Patrick presented with throughout his life, saying that Patrick "never showed that semiology again, the seizures seemed to be stiffening seizures or soaking seizures, or even eye-rolling seizures potentially later."[521] That would make the first event different from the other events which, Associate Professor Fahey said, he would consider unusual.[522]

254. Dr Colley said that in her opinion it is relevant that in the Folbigg family, there were three other children who also died young without any evidence of epilepsy or seizure, which was inconsistent with a genetic epileptic encephalopathy in the family.[523] She said prior to the ALTE, there was no evidence of a myopathy clinically at all (in relation to any of the children).[524] She said none of the children had a phenotype of a syndrome or a condition or an illness that was diagnosable before each catastrophic event (in Patrick's case, the ALTE).[525]

Opinions on cause of Patrick's death – forensic pathology

255. In the Inquiry, none of the forensic pathologists disagreed that Patrick had an encephalopathic disorder at the time of death, Dr Cala noting that that would be the suggested diagnosis from treating doctors as a guide for the pathologists conducting the autopsy.[526]

256. Professor Cordner and Professor Duflou each attributed Patrick's death to the consequences of the encephalopathic disorder, with Professor Cordner attributing it to epileptic seizures with no evidence of the underlying cause of the encephalopathic disorder, and Professor Duflou more directly attributing it to the encephalopathy brought on by the ALTE.[527]

257. Professor Cordner stated it to be "a non-controversial, ordinary thought" that Patrick's death was from delayed effects of the ALTE:[528]

> *To cut a long story short, considered alone, this death is reasonably thought of as being due to the epileptic consequences of the brain damage following the ALTE. This approach still leaves the ALTE unexplained*.[529]

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517 Transcript of the Inquiry, 17 April 2019 T600.11-12.
518 Transcript of the Inquiry, 17 April 2019 T600.11-15.
519 Transcript of the Inquiry, 17 April 2019 T601.1-8.
520 Transcript of the Inquiry, 17 April 2019 T597.45-49.
521 Transcript of the Inquiry, 17 April 2019 T593.38-46.
522 Transcript of the Inquiry, 17 April 2019 T593.45-46.
523 Transcript of the Inquiry, 17 April 2019 T600.25-28.
524 Transcript of the Inquiry, 15 April 2019 T442.31-34.
525 Transcript of the Inquiry, 16 April 2019 T503.14-17.
526 Transcript of the Inquiry, 21 March 2019 T267.41-268.11.
527 Transcript of the Inquiry, 20 March 2019 T162.32-39; 21 March 2019 T268.1 (Professor Duflou); 20 March 2019 T160.39-40, T161.19, T161.21-27, T162.4 (Professor Cordner); Exhibit L, Report of Professor Johann Duflou (13 February 2019) p 32.
528 Exhibit Q, Report of Professor Stephen Cordner (undated) p 82.
529 Exhibit Q, Report of Professor Stephen Cordner (undated) p 60, n 60.

258. Professor Cordner accepted that it is most unlikely that encephalitis was ever present, although he noted that at the time of Patrick's death, Dr Wilkinson was of the view that Patrick's death was due to his epilepsy, in turn a consequence of brain damage from the ALTE.[530] He analysed Dr Wilkinson's evidence at trial, and concluded inter alia that insofar as it related to understanding the cause of Patrick's ALTE and death, it was confusing and potentially misleading.[531] Professor Cordner also referred to the seizures resulting in admissions on 4 and 14 November 1990 never having been alleged to be other than the natural consequences of Patrick's underlying encephalopathy – there was, he stated, strong internal evidence of the fatal potential of the associated seizure disorder.[532]

259. In this regard, in his peer review of Professor Cordner's report, Professor Pollanen described Patrick's death as a straightforward clinicopathologic correlation between old laminar necrosis (cell death in the cerebral cortex) in the cerebral cortex and the history of a seizure disorder, which could explain the death as SUDEP.[533]

260. In his report, Professor Duflou stated that Patrick's cause of death was the consequences of hypoxic-ischaemic encephalopathy brought on by an ALTE of unascertained cause.[534] He referred to the severe brain pathology upon death, typical of one or more episodes of hypoxia-ischaemia sometime in the past.[535] No definite cause was identified "although again there were concerns in relation to prior encephalitis".[536] Professor Duflou noted that on the day of his death, Patrick had a fever and had a cardiorespiratory arrest; he also referred to autopsy findings of severe brain damage in the form of laminar necrosis, gliosis and a leptomeningeal chronic lymphocytic infiltrate.[537]

261. Professor Duflou considered that epilepsy was possibly the cause of Patrick's death, noting that in the majority of epilepsy-related deaths, the brain is normal with the exception of possible changes consequent on the epilepsy.[538] Professor Duflou referred to general difficulties diagnosing death due to epilepsy, especially where the death is sudden and unexpected, and concluded that it is an area of evolving scientific investigation with familial and many ill-defined conditions likely playing a role in death.[539] He noted that to a very large extent, the event is:

> *unwitnessed and not independently verifiable, and unless there has been brain damage which is either the cause of the epilepsy or is a consequence of seizures, with or without episodes of hypoxia/ischaemia to the brain, even a very detailed neuropathological examination of the brain can be entirely negative.*[540]

Professor Duflou emphasised the complexity, with many suggested links and interactions between the brain and heart in epilepsy.[541] Professor Hilton described the cause of death as part of an epileptic-type illness.[542]

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530 Exhibit Q, Report of Professor Stephen Cordner (undated) pp 60, 67.
531 Exhibit Q, Report of Professor Stephen Cordner (undated) pp 60-67.
532 Exhibit Q, Report of Professor Stephen Cordner (undated) p 67.
533 Exhibit C, Report of Professor Michael Pollanen (1 June 2015) p 5.
534 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 32; Transcript of the Inquiry, 20 March 2019 T162.32-35.
535 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 32.
536 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 32.
537 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 30; Transcript of the Inquiry.
538 Transcript of the Inquiry, 20 March 2019 T162.32-39; 21 March 2019 T267.45-268.1, T270.41-48.
539 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 40.
540 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 40.
541 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 40.
542 Transcript of the Inquiry, 20 March 2019 T164.13-22.

262. Dr Cala remained unconvinced that epilepsy caused Patrick's death and would not give epilepsy as a cause.[543] As noted above, he stated that although he could not definitely prove smothering had occurred, he was concerned that smothering could explain Patrick's ALTE and his sudden death.[544] He noted that many tests were performed both ante- and post-mortem, with no satisfying diagnosis ever reached.[545]

263. Dr Cala also noted that in 1989-90, a death of a second child from natural causes following sudden death of first child, in Australia, would be considered most unusual, and is of concern.[546]

Opinions on cause of Patrick's death – neurology

264. The neurology experts disagreed on whether at the time of his death Patrick clearly had encephalopathy. Professor Ryan had no doubt, although also said that she thought it was difficult to be entirely sure whether or not Patrick had a progressive neurological condition.[547]

265. Associate Professor Fahey, however, did not accept that Patrick had a progressive encephalopathic condition.[548] He would not use the word "progressive", as it implies something which is deteriorating and changing; he said that this was not known in Patrick's case. Rather, Associate Professor Fahey's opinion was that Patrick had an insult, and there were evolving changes as a result of it.[549] There were no signs of a deteriorating condition on the brain pathology; what was seen was, rather, old ischaemic changes and not active changes involving cell deterioration.[550]

266. After receiving the genetics results, Associate Professor Fahey concluded that the testing at the time of Patrick's ALTE and death, and the genomic testing conducted in the Inquiry, excluded any recognised conditions associated with genetic epilepsies, encephalopathy, cardiac arrhythmias or sudden death, including the alternative potential diagnoses identified by Professor Ryan.[551]

267. Duncan and Byard (2018) note that the entity SUDEP usually occurs during sleep as an unwitnessed event and young individuals are mostly found in the prone position.[552] While it is a well-known complication of any seizure disorder, the rates in childhood are reported as 1.1-4.3/10,000 patient years.[553] Like SIDS, SUDEP is a diagnosis of exclusion after detailed post-mortem examination reveals no anatomical or toxicological cause of death in an individual with a known history of epilepsy. Further:

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543 Transcript of the Inquiry, 22 March 2019 T281.20-22; Exhibit M, Report of Dr Allan Cala (26 November 2018) p 10.

544 Exhibit M, Report of Dr Allan Cala (26 November 2018) pp 10-11; Transcript of the Inquiry, 20 March 2019 T165.14-24.

545 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 10.

546 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 10, citing American Academy of Pediatrics, 'Distinguishing Sudden Infant Death Syndrome from Child Abuse Fatalities' (2006) 118(1) *Pediatrics 421.*

547 Transcript of the Inquiry, 17 April 2019 T608.11-14, T609.11-12.

548 Transcript of the Inquiry, 17 April 2019T608.29-31.

549 Transcript of the Inquiry, 17 April 2019 T608.29-34.

550 Transcript of the Inquiry, 17 April 2019 T608.39-46.

551 Transcript of the Inquiry, 17 April 2019 T589.34-590.5; Exhibit AK, Report of Professor Michael Fahey (30 March 2019) p 4.

552 Exhibit D, Heather E Jeffery, 'Future Directions in Sudden Unexpected Death in Infancy Research' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 283, 292, citing J A Liebenthal et al, 'Association of Prone Position with Sudden Unexpected Death in Epilepsy' (2015) 84 *Neurology* 703.

553 Exhibit D, Victoria A Bryant and Neil J Sebire, 'Natural Diseases Causing Sudden Death in Infancy and Early Childhood' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 539, 565, citing C M Milroy, 'Sudden Unexpected Death in Epilepsy in Childhood' (2011) 7(4) *Forensic Science, Medicine and Pathology* 336.

> [s]*tructural brain lesions may be identified as the underlying cause of epilepsy, such as cortical malformations, hippocampal sclerosis, cerebral atrophy, and hydrocephalus, but often there is no obvious pathology identified. A number of mechanisms have been proposed, including cardiac arrhythmias and central apnea.*[554]

268. In his report, Associate Professor Fahey also cited research on the features and incidence of SUDEP.[555] He noted that the definition of SUDEP is that it occurs in children older than one year old and with normal autopsy examination and as a diagnosis of exclusion. He observed that SUDEP is a rare condition, and:

> *affects 1 in 4,500 children with epilepsy per year. It is more likely in those with long-standing epilepsy, with frequent generalised tonic-clonic seizures despite multiple medications and in those who had a young age of onset. In childhood, it is also associated with specific genetic changes, particularly those of sodium, potassium and calcium ion channels and in those who have apnoea (stop breathing) during the epileptic events. As above, pathogenic changes within these genes were not identified. I note that Patrick's last documented event consisting of tonic up gaze was on the 22nd December 1990 before his death on the 13th February 1991.*[556]

Time of death

269. Professor Duflou noted that the time of Patrick's death was not assessed, but he was described as having a normal skin temperature by ambulance officers.[557] Professor Duflou stated in his report that given that the only physical description provided was that the body was warm to touch, it is entirely possible for Patrick to have died at any time from when Mr Folbigg went to work until the time at which the ambulance officers arrived, with a later time more likely than an earlier time.[558]

270. In oral evidence in the Inquiry, Professor Duflou said that he did not have a specific time that Mr Folbigg had gone to work and accepted that not knowing this rendered his statement as to the earlier time somewhat meaningless.[559] He agreed that the time of Patrick's death could not be determined with any degree of certainty.[560]

Counsel assisting's submissions on cause of Patrick's ALTE

271. Counsel assisting submitted that as with Caleb, there have been two material changes since the 2003 trial. First, genetic testing has been completed in relation to Patrick and no genetic variant which is pathogenic or likely pathogenic has been identified to account for Patrick's ALTE. Secondly, more recent research on SIDS that maternal smoking and sleeping position pose the highest risks relevantly reduces any assessment of Patrick's risk of SIDS or ALTE.[561]

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554 Exhibit D, Victoria A Bryant and Neil J Sebire, 'Natural Diseases Causing Sudden Death in Infancy and Early Childhood' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 539, 565, citing C M Milroy, 'Sudden Unexpected Death in Epilepsy in Childhood' (2011) 7(4) *Forensic Science, Medicine and Pathology* 336.

555 Exhibit AK, Report of Professor Michael Fahey (30 March 2019) p 14, citing S Shorvon and T Tomson, 'Sudden Unexpected Death in Epilepsy' (2011) 378 *Lancet* 2028.

556 Exhibit AK, Report of Professor Michael Fahey (30 March 2019) p 14, citing S Shorvon and T Tomson, 'Sudden Unexpected Death in Epilepsy' (2011) 378 *Lancet* 2028.

557 Exhibit L, Report of Professor Duflou (13 February 2019) p 30.

558 Exhibit L, Report of Professor Duflou (13 February 2019) p 30.

559 Transcript of the Inquiry, 20 March 2019 T167.1-11.

560 Transcript of the Inquiry, 20 March 2019 T167.13-23.

561 Submissions of Counsel Assisting the Inquiry (17 May 2019) Chapter 7, [69].

272. The medical experts gave broadly consistent evidence at the trial that the ALTE was most likely caused by an asphyxiating event.[562] Before the Inquiry, Professor Ryan was not "convinced" that this was the case, whereas Associate Professor Fahey expressed the opinion that Patrick's presentation was consistent with a severe hypoxic event on 18 October 1990.[563]

273. Counsel assisting submitted that I should reject Professor Ryan's opinion as to Patrick's presentation and subsequent variability, and his prior health, not being consistent with a single hypoxic episode on 18 October 1990. Counsel assisting contended that the opinion is mere conjecture and is inconsistent with the opinion evidence of multiple other relevantly qualified witnesses at the trial and in the Inquiry, the foundation for which is found in clinical records of Patrick's presentation and medical history prior to and after the ALTE.[564]

274. Counsel assisting noted that when asked in oral evidence whether the consensus opinion of the other expert witnesses affected her initial opinion, Professor Ryan responded that it did not. Further, she did not accept the uncontroversial proposition grounded in lay and medical evidence that Patrick was a healthy and normally developing baby immediately prior to the ALTE.[565]

275. In addition, Professor Ryan impliedly accepted in any event the reasonableness of the proposition that Patrick suffered a single hypoxic episode on that date by suggesting prolonged seizure in the context of fever resulting in ischaemic damage, akin to Dravet syndrome, as a potential alternative unidentified genetic cause of the ALTE.[566]

276. Counsel assisting submitted that ultimately, on the basis of the medical evidence, both clinical and expert, there is no identified natural cause of the ALTE, in the sense of something more than a "debating point possibility". In particular, evidence does not support as the cause of Patrick's ALTE a respiratory problem or neurological condition such as encephalitis including a degenerative neurological disease, or a SIDS-type event.[567]

277. Counsel assisting also submitted that the medical evidence does not exclude that the ALTE was caused by an asphyxial event including smothering. Expert opinion evidence supports an asphyxial event having occurred, with a cause other than one attributable to a respiratory or a recognised neurological condition.[568]

278. Ultimately, counsel assisting submitted that on the medical evidence in 2019 there remains no identifiable natural (including genetic) cause of Patrick's ALTE and that it occurred from unnatural causes cannot be excluded.[569]

562 Submissions of Counsel Assisting the Inquiry (17 May 2019) Chapter 7, [70].
563 Submissions of Counsel Assisting the Inquiry (17 May 2019) Chapter 7, [71].
564 Submissions of Counsel Assisting the Inquiry (17 May 2019) Chapter 7, [72].
565 Submissions of Counsel Assisting the Inquiry (17 May 2019) Chapter 7, [73].
566 Submissions of Counsel Assisting the Inquiry (17 May 2019) Chapter 7, [74].
567 Submissions of Counsel Assisting the Inquiry (17 May 2019) Chapter 7, [76].
568 Submissions of Counsel Assisting the Inquiry (17 May 2019) Chapter 7, [77].
569 Submissions of Counsel Assisting the Inquiry (17 May 2019) Chapter 7, [78].

Counsel assisting's submissions on cause of Patrick's death

279. Counsel assisting drew attention to evidence that Drs Cala and Beal and Professors Herdson and Berry all considered that the death could have been the result of an asphyxiating event.[570] Professor Ouvrier said that it appeared to be an asphyxial episode without clear explanation.[571] Professor Busuttil stated that the death should not be attributed to asphyxia, but gave no alternative, saying only that the brain condition "could" have given rise to convulsions causing death.[572] At trial, four forensic pathologists – Dr Cala and Professors Herdson, Berry and Byard – all said they would give Patrick's death as undetermined.[573] Dr Cala maintained this view in the Inquiry.[574]

280. Also, counsel assisting submitted, in the Inquiry Professors Cordner and Duflou each attributed Patrick's death to the consequences of the encephalopathic disorder he suffered, with Professor Cordner attributing it to epileptic seizures with no evidence of the underlying cause of the encephalopathic disorder, and Professor Duflou more directly attributing it to the encephalopathy brought on by the ALTE.[575] To say that encephalopathy caused the death, however, does not identify the mechanism of the final event. Professor Duflou considered that epilepsy was possibly the cause.[576] Professor Hilton described the cause of death as part of an epileptic-type illness.[577]

281. Counsel assisting also relied upon the material change in the available evidence since the 2003 trial that no genetic variant which is pathogenic or likely pathogenic has been identified to account for Patrick's death.[578] In addition, the Inquiry has enabled further consideration of the role infection may have played in Patrick's death. As will be considered in Chapter 6, counsel assisting submitted that Patrick's cause of death cannot be attributed to infection.[579]

282. Counsel assisting observed that no medical expert, at trial or in the Inquiry, has ruled out the possibility of a seizure having caused Patrick's death. Opinions have ranged from this being highly unlikely, or not excluded, or could have, to "would say" that epilepsy caused death. Accordingly, it is possible, but no more than possible, that a seizure caused his death.[580]

283. Also, most medical experts considered that the death could have been the result of an asphyxiating event. No medical expert excluded asphyxia or smothering.[581]

284. Counsel assisting submitted that Professor Duflou's opinion in his report that it was possible that Patrick died any time from when Mr Folbigg went to work, should not be accepted.[582]

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570 16 April 2003 T747.28-31 (Dr Cala); 5 May 2003 T1139.52-1140.2 (Dr Beal); 1 May 2003 T1036.4-11 (Professor Herdson) T1061.53-1062.6 (Professor Berry), T1076.28-37 (Professor Herdson).

571 Exhibit H, Forensic pathology tender bundle, Statement of Dr Robert Ouvrier (28 October 2002) p 4.

572 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 10.

573 16 April 2003 T747.21 (Dr Cala); 1 May 2003 T1035.46-1036.2 (Professor Herdson) T1061.53-1062.2 (Professor Berry), T1073.52-1074.9, T1076.24-3 (Professor Herdson); Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 8.

574 Transcript of the Inquiry, 20 March 2019 T163.43-47.

575 Transcript of the Inquiry, 20 March 2019 T162.32-39 (Professor Duflou); T268.1, T160.39-40; T161.19, T161.21-27, T162.4 (Professor Cordner).

576 Transcript of the Inquiry, 20 March 2019 T162.32-39, T268.1 (Professor Duflou), T160.39-40, T161.19, T161.21-27, T162.4 (Professor Cordner).

577 Transcript of the Inquiry, 20 March 2019 T164.13-22.

578 Submissions of Counsel Assisting the Inquiry (17 May 2019) Chapter 7, [96].

579 Submissions of Counsel Assisting the Inquiry (17 May 2019) Chapter 7, [97].

580 Submissions of Counsel Assisting the Inquiry (17 May 2019) Chapter 7, [98].

581 Submissions of Counsel Assisting the Inquiry (17 May 2019) Chapter 7, [99].

582 Submissions of Counsel Assisting the Inquiry (17 May 2019) Chapter 7, [95].

Ms Folbigg's submissions on cause of Patrick's ALTE and death

285. Ms Folbigg submitted that Patrick's ALTE was likely caused by the same condition that caused his death.[583]

Evidence at trial

286. Ms Folbigg submitted that at the trial there was limited consideration of any alternative causes of encephalopathy that may explain Patrick's ALTE and death, and no evidence to address issues of immunology and infection, or any progressive neurological condition or congenital or other disorder.[584] This was despite it being accepted by Dr Kan that separately from encephalitis there could have been a seizure because of some abnormality in some section of the brain.[585] Re-examination by the Crown presupposed that an abnormality of the brain was structural and visible, which ignored:

> *the possibility of a congenital or other disorder giving rise to a process with respect to the disturbance of respiration, neurotransmitters or cardiac function.*[586]

287. Ms Folbigg submitted that the issue of some congenital or other disorder was not considered, let alone excluded, by the Crown.[587] She noted that clinical notes recorded back arching and torticollis, both of which can be a sign of cerebral irritation.[588] She asserted the potential cause of encephalopathy was treated simplistically by the Crown prosecutor, and that in closing the Crown overstated Dr Kan's report in saying:

> *Dr Kan's opinion excluded effective causes of death, metabolic causes of death, genetic disorders, and that changes in the brain from the past episode, the ALTE, appeared to have been caused by some event which is just a hypoxic event in the past. There was only signs of old damage to the brain, consistent with having been done four or five months earlier. Dr Kan and Dr Singh-Khaira were unable to find any cause of death.*[589]

288. Ms Folbigg suggested that the Crown reliance upon Dr Wilkinson's evidence was misplaced, because he was a paediatrician, not a neurologist.[590] Further, she submitted that Dr Dezordi's opinion, that Patrick's condition following his ALTE was not likely to be due to a respiratory condition because he remained pink even when he was not administered a high concentration of oxygen, is inconsistent with smothering.[591] The Crown prosecutor was also wrong insofar as he submitted that Patrick had stopped breathing during his ALTE.

289. Ms Folbigg also submitted that if she had lost control in a murderous rage when Patrick suffered the ALTE, she would likely have inflicted some injury on Patrick. Further, such a theory was inconsistent with Mr Folbigg's evidence that he noticed nothing untoward regarding her behaviour proximate to the event.[592]

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583 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [38].
584 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [9]-[11].
585 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [12]-[13]; 24 April 2003 T929.13-16.
586 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [16]-[17].
587 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [11].
588 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [23].
589 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [12]; 13 May 2003 T1325.12-20.
590 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [27].
591 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [40] n 14.
592 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [32], citing Exhibit E, ERISP of Kathleen Folbigg Q186, 445.

290. Ms Folbigg also submitted that the summing-up failed to refer to:

    a. the clinical finding on autopsy of encephalopathy;

    b. a progressive disease or condition; or

    c. the opinions of the emergency physicians that Patrick had suffered a cardiac arrest.[593]

Evidence before the Inquiry

291. Ms Folbigg submitted that there was evidence in the Inquiry that Patrick probably had a progressive neurological condition that caused the ALTE and his death.[594] She submitted that counsel assisting neglected to address evidence given by Professor Ryan that genetic disorders can only be identified in about one third of cases of severe neurological dysfunction.[595] She suggested that this means that in the vast majority of cases the cause is unknown, and that this undermines Associate Professor Fahey's opinion that because no pathogenic genetic mutation was found after genetic testing, all recognised genetic conditions are now excluded as the cause of Patrick's ALTE and death. Importantly, Professor Ryan gave evidence that "[t]here are other children in whom a similar clinical presentation is seen for which a genetic cause cannot be found."[596] She noted that a genetic cause had not been identified by a urine screen.[597]

292. Ms Folbigg referred to the notes of back arching and torticollis, noting that Dr Colley may be correct that they are common symptoms. She submitted that Patrick was seriously unwell on 18 October 1990, and Associate Professor Fahey would not commit to him being normal and healthy beforehand.[598]

293. In respect of Hunter syndrome, Ms Folbigg submitted that although Professor Kirk thought it was highly unlikely to have caused Patrick's death, his opinion was qualified by a lack of further testing and did not exclude a digenic trigger or a combination of a genetic cause and exogenous stressor such as infection or fever: "In other words, the Hunter Syndrome of itself may not have been pathogenic… but in combination with other things, it could have become pathogenic."[599]

294. A study by Constantinou et al described "at least one, and often many organ systems"[600] as showing hypoxic derangement following a hypoxic event or insult which Ms Folbigg submitted contradicts Associate Professor Fahey's opinion that the study had inclusion criteria that did not require failure of multiple organs. The study is consistent with Professor Ryan's views that it is more likely that, had Patrick suffered hypoxic insult, there would be hypoxic derangement in other organ systems, which there was not.[601]

295. Ms Folbigg noted that Associate Professor Fahey did not exclude the possibility that the ALTE may have been caused by a seizure.[602] Professors Hilton, Duflou and Cordner agreed.[603]

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593 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [34], citing 19 May 2003 T65-66.

594 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [10].

595 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [44] n 19.

596 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick [96], citing Transcript of the Inquiry, 17 April 2019 T604.15-17.

597 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [7].

598 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [41].

599 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [49] n 23.

600 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [58], citing J E Constantinou et al, 'Hypxic-Ischaemic Encephalopathy After Near Miss Sudden Infant Death Syndrome' (1989) 64(5) *Archives of Diseases in Childhood* 703, 706.

601 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [60] n 38.

602 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [69] n 45.

603 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [70] n 46.

296. Ms Folbigg submitted that Professor Ryan undertook a retrospective analysis of Patrick's ALTE while Professor Fahey appears to have undertaken a prospective analysis by assuming the ALTE was as a consequence of a hypoxic insult and then drawn inferences from that assumption. Associate Professor Fahey ignored the deteriorating and inconsistent presentation of Patrick post 18 October 1990, which is inconsistent with a hypoxic event.[604]

297. She submitted that that the submission by counsel assisting that Professor Ryan's opinion should be rejected is without basis. Associate Professor Fahey did not dismiss Professor Ryan's opinions, nor vice versa.[605]

Conclusions

298. Ms Folbigg submitted that the treating medical practitioners on 18 October 1990 considered Patrick's condition was not hypoxia, as otherwise there would have been a different clinical management strategy revealed in the clinical records.[606]

299. She also submitted that the progression of cerebral symptoms confirms the back arching noted in the records is likely caused by cerebral irritation.

300. In Ms Folbigg's submission, the Crown case that the ALTE was caused by smothering is open to considerable doubt in light of the evidence of Professor Ryan that the progression of symptoms did not follow the pattern that would be expected from hypoxia.[607]

301. She further submitted that Patrick was showing clinical signs of infection on the night before his death and that infection can cause cardiac arrhythmia.[608] She noted that Dr Walker opined that Patrick had sustained a cardiac arrest prior to his arrival in hospital. She submitted that this issue was not addressed at trial and is an alternative natural cause of death.[609]

302. Ms Folbigg asserted that counsel assisting's submission that "[t]he medical experts gave broadly consistent evidence at the trial that the ALTE was most likely caused by an asphyxiating event" is misleading. She said neither Associate Professor Fahey nor Professor Ryan gave evidence that the ALTE was most likely caused by asphyxiation.[610]

303. She concluded that the ALTE could have been caused by an epileptic seizure or a progressive brain disorder, and the death could have been caused by arrhythmia, an epileptic fit, a progressive brain disorder or infection.

Professor Hilton's submissions on cause of Patrick's death

304. In his submissions in response, Professor Hilton said that Ms Folbigg's submissions fairly reflected his opinion as to the possible cause of death of Patrick.[611]

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604 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [71]-[72] nn 48-49.
605 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [68] n 44.
606 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [64] nn 40-41.
607 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [90] n 59.
608 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [77].
609 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [77]-[79].
610 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [100] nn 66-68.
611 Submissions of Professor Hilton to the Inquiry (18 June 2019).

Findings: Patrick's ALTE and death

ALTE

305. In relation to Patrick's ALTE, having regard to all of the medical evidence set out above, I find as follows.

306. There have been two material changes since the 2003 trial in relation to diagnosis of any medical cause of Patrick's ALTE. First, genetic testing has been completed in relation to Patrick and no genetic variant which is pathogenic or likely pathogenic has been identified to account for the ALTE. Secondly, more recent research on SIDS that maternal smoking and sleeping position pose the highest risks relevantly reduces any assessment of Patrick's risk of SIDS or ALTE.

307. Medical evidence in the Inquiry, including evidence adduced at trial, and including both clinical and expert evidence, did not identify any natural cause of Patrick's ALTE as a reasonable possibility. However, on the basis of evidence in the Inquiry, the following conditions are excluded as a possible cause of the ALTE:

    a. encephalitis; and

    b. genetic conditions which were the subject of testing in the Inquiry (as set out in Chapter 7);

308. Having regard to the medical (including genetic) evidence in the Inquiry the following conditions are not excluded as having caused the ALTE, however I find that it is not reasonably possible that any of them caused the ALTE:

    a. epilepsy, or an initial seizure;

    b. an unidentified genetic or metabolic condition;

    c. an unidentified degenerative brain condition or neurological disease or condition;

    d. infection or virus (including particularly pneumonia, meningitis, septicaemia, meningococcal and bronchiolitis) other than encephalitis; and

    e. a SIDS-type event.

309. In making these findings, I refer particularly to the following evidence:

    a. the findings within normal limits following sleep studies soon after Patrick's birth;

    b. the absence of evidence that Patrick was otherwise than growing normally and was healthy prior to the ALTE;

    c. Dr Dezordi's evidence at trial that the scenario was most consistent with some catastrophic event causing lack of oxygen on 18 October 1990;

    d. Dr Wilkinson's evidence at trial of examinations and tests of Patrick following the ALTE, of findings upon EEGs and CT scans, and his opinion that the changes within Patrick's brain and development of seizures were typical of asphyxial damage;

    e. Dr Lai's evidence at trial concerning the two CT scans of Patrick's brain;

    f. Dr Kan's findings and evidence at trial of post-mortem examination of changes in Patrick's brain. In this regard I have considered Ms Folbigg's submissions on Dr Kan's evidence. Under cross-examination Dr Kan did not exclude the possibility of a seizure, nor encephalitis, having caused damage to Patrick's brain found at autopsy.[612] He also agreed it was possible his brain could have swollen as a result of oxygen deprivation which led to a seizure disorder.[613] His evidence in this regard was one part of the evidence as to possible causes of the ALTE;

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612 24 April 2003 T929.37-55.

613 24 April 2003 T931.15-25.

g. forensic pathology evidence at trial and in the Inquiry. This includes acknowledgement by Professors Hilton, Duflou and Cordner in the Inquiry that the ALTE could have been caused by an epileptic seizure;

h. neurology evidence in the Inquiry, to which I refer further below; and

i. genetic evidence in the Inquiry (and to the extent of evidence of genetic tests conducted after the ALTE, and to genetic evidence at trial).

310. I have closely considered Professor Ryan's opinion (and accept Ms Folbigg's submission that she was well qualified to give it)[614] that Patrick's presentation at the time of and after the ALTE, and prior health, were not consistent with a single hypoxic episode on 18 October 1990. However, for the following reasons, I have determined to prefer Associate Professor Fahey's opinion, which supports a single such event.

311. First, both Associate Professor Fahey and Dr Wilkinson had qualifications and experience to give opinion evidence on the same subject matter. Contrary to a submission made by Ms Folbigg, Dr Wilkinson was a paediatric neurologist.[615] His opinions were tested at trial and were supported by his correspondence contemporaneous with his investigations of Patrick after the ALTE. In this regard, I have also taken into account Professor Cordner's discussion in his report of Dr Wilkinson's evidence and opinion that the cause of Patrick's ALTE was unknown.

312. Secondly, from extensive clinical notes concerning Patrick, Professor Ryan relied upon two notes of previous back arching and torticollis in support of her opinion. I agree with counsel assisting that it should have been uncontroversial that on the available evidence, Patrick was healthy before the ALTE. No other medical expert, including Associate Professor Fahey, attributed such weight to those two discrete notations in the volume of Patrick's clinical records.

313. Thirdly, in her oral evidence Professor Ryan was inexplicably unprepared, or at least gave this appearance, to either give serious consideration to the consensus of other medical opinions which were inconsistent with her own, or to accept the weight of records and evidence indicating that Patrick was healthy and developing normally prior to the ALTE. For example, she was unprepared to accept that Patrick was hypoxic on arrival at the hospital in part because an oximetry probe can misread blood oxygen levels. Professor Ryan's position added to the overall impression that she was unprepared to give reasonable consideration to the factual evidence that was available.

314. While Professor Ryan may have ultimately accepted it was "possible" that Patrick suffered a single hypoxic-ischaemic episode on 18 October 1990,[616] my concerns remain. In view of Professor Ryan's responses to these propositions, I treat her opinions with caution and I am not confident that she gave serious consideration to countervailing evidence and opinions.

315. Further, also inviting caution in taking account of her opinion that the event on 18 October 1990 may not have been isolated, Professor Ryan herself proposed that Patrick may have suffered a prolonged seizure on 18 October 1990, in the context of fever resulting in ischaemic damage, and akin to Dravet syndrome, as a potential alternative unidentified genetic cause of the ALTE.

614 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [42].

615 10 April 2003 T506.29-507.14; Exhibit H, Forensic pathology tender bundle, Statement of Dr Ian Wilkinson (8 October 1999).

616 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [42]; Transcript of the Inquiry, 17 April 2019 T585.17.

316. I find Associate Professor Fahey's opinion evidence in the Inquiry, and the reasons he gave for holding them, to be more consistent, and more consistently grounded, in the available evidence of Patrick's history and presentations. None of the experts gave a conclusive explanation for why there was a delay in the onset of Patrick's seizures and why the initial EEG was normal compared with deterioration seen later. However, Associate Professor Fahey's opinion appears to me to be supported by clinical and forensic pathology evidence (including Dr Kan's findings), and while demonstrating some variation in reasoning, was overall compatible with Dr Wilkinson's evidence that delay in onset was consistent with – indeed, Dr Wilkinson said it was typical after asphyxia damage for progressive change and seizures produced down the track – an initial event. Professor Ouvrier's report contained similar opinions.

317. Ms Folbigg's submissions underscored evidence given by Professor Ryan that genetic disorders can only be identified in about one third of cases of severe neurological dysfunction, and also pointed to qualifications on Professor Kirk's opinion. As set out more fully in Chapter 7 of this report, the genetic testing undertaken by the Inquiry was exhaustive on current resources and knowledge. In view of all of the evidence, which includes evidence of extensive searches for a medical or genetic explanation in 1990 and in the Inquiry, there is no evidence of a reasonable possibility of some as yet unidentified genetic trigger amounting to a reasonable hypothesis of a genetic cause of Patrick's ALTE (or death).

318. Dr Dezordi, paediatrician, gave evidence that he found no evidence of trauma or injury when Patrick presented after the ALTE.[617] Ms Folbigg submitted that if, as on the Crown case, she had lost control in a murderous rage she would "likely have inflicted some injury". I do not accept this proposition. There is no evidence that in her irritation (set out in Chapter 8) she was "likely" to inflict detectable injury on the children.

319. Ms Folbigg also contended that Dr Dezordi's opinion was inconsistent with smothering because Patrick remained pink even without high concentration of oxygen. Dr Dezordi gave evidence at trial that when Patrick was brought in by ambulance, he was blue (not pink) and lethargic.[618] His colour normalised fairly rapidly – a respiratory condition would have impaired exchange of oxygen across the airways into the bloodstream.[619] I do not accept Ms Folbigg's submission as to the inconsistency of Dr Dezordi's evidence.

320. It is reasonably possible that the ALTE was caused by an asphyxiating event. Dr Wilkinson was definite that this could have occurred.[620] Dr Dezordi had seen quite a lot of babies with asphyxia and hypoxia and had no doubt the whole scenario was most consistent with a catastrophic event causing lack of oxygen, without any medical cause found.[621] It is also significant that the injury to Patrick's brain and the injury to his sight are known sequelae of a lack of oxygen to the brain.[622]

321. I find that expert opinion evidence supports a single asphyxial event having occurred on 18 October 1990, with a cause other than one attributable to a respiratory or a recognised neurological condition. For clarity, by "asphyxial", I mean an event leading to obstruction of his airways. Further, medical evidence does not exclude that deliberate smothering of Patrick caused the ALTE.

Death

322. In relation to Patrick's death I find as follows.

323. Material changes in the available evidence since the 2003 trial in relation to Patrick's death are first, that no genetic variant which is pathogenic or likely pathogenic has been identified to account for Patrick's death, and secondly, there is now evidence of the role that infection may have played in Patrick's death.

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617 9 April 2003 T450.5-7.
618 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [40]; 9 April 2003 T446.46-48.
619 9 April 2003 T448.25-37.
620 10 April 2003 T499.57-500.19.
621 10 April 2003 T505.23-46.
622 1 May 2003 T1042.54-56.

324. The medical evidence in the Inquiry, which includes the medical evidence given at trial, does not establish as a reasonable possibility any natural cause of Patrick's death.

325. The evidence in the Inquiry does not support the possibility of some congenital disorder, progressive neurological condition or other similar disorder having caused or contributed to Patrick's death. I do not accept Ms Folbigg's submissions to that effect, noting that while Dr Kan's opinion at trial excluding metabolic causes and genetic disorders remains relevant, it is vastly overtaken by fresh evidence produced by genetic investigations by the Inquiry.

326. It is possible, on the basis of forensic pathology opinions in the Inquiry, that Patrick's death was attributable to encephalopathy in his brain. While this identifies a possible cause, it does not explain how the encephalopathy caused death. On the basis of the opinions of Professors Duflou, Hilton and Cordner, and no relevant medical expert either at trial or in the Inquiry having ruled out the possibility of a seizure, it is possible that the encephalopathy caused a seizure, which caused death. I note, however, evidence in the Inquiry of the rarity of SUDEP.

327. Medical evidence establishes that it is reasonably possible that Patrick's death was caused by an asphyxiating event, by which I mean an event leading to obstruction of his airways, and which in context was some obstruction from a cause other than a seizure.

328. On medical evidence at trial, Patrick's death could have been the result of an asphyxiating event. Dr Singh-Khaira, Dr Cala, Dr Beal, Professor Herdson, Professor Berry, and Professor Ouvrier all gave evidence or recorded opinions to this effect.[623] Dr Wilkinson was of the view that an asphyxiating event directly caused Patrick's death, with the cause of the event itself unknown.[624] I have taken into account Professor Busuttil's opinion that the death should not be attributed to asphyxia, saying that the brain condition "could" have given rise to convulsions causing death.[625]

329. On the new forensic pathology evidence in the Inquiry, Dr Cala maintained his concern that Patrick had been smothered and also his opinion that the cause of Patrick's death was undetermined. Neither Professor Cordner nor Professor Duflou excluded smothering.

330. For reasons set out in Chapter 6, Patrick's death cannot be attributed to infection, and I do not accept Ms Folbigg's submissions to the effect that his death could have been caused by a cardiac arrhythmia, in turn caused by an infection. I consider that proposition to be mere conjecture, made on thin evidence of Dr Walker's (emergency physician) initial opinion when Patrick arrived in hospital, and otherwise essentially implausible.

331. I do not place any weight on Professor Duflou's opinion that Patrick could have died any time after Mr Folbigg went to work on 13 February 1991, in the absence of evidence of the time that Mr Folbigg went to work.

332. I find on the available medical evidence that it is possible that the cause of Patrick's death on 13 February 1991 was a seizure or similar event related to encephalopathy in his brain. I find further on the available medical evidence that it is reasonably possible that his death was caused by an asphyxiating event, meaning an event leading to obstruction of his airways and which includes deliberate smothering. The answer to the question of which of these it was lies not in the medical evidence in relation to Patrick, but in a consideration of a number of different aspects of the evidence in this case.Sarah

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623 11 April 2003 T560.43-48 (Dr Singh-Khaira); 16 April 2003 T747.28-31 (Dr Cala); 28 April 2003 T990.52-56 (Dr Beal); 1 May 2003 T1036.8-11 (Professor Herdson), T1076.29-37 (Professor Berry); Exhibit H, Forensic pathology tender bundle, Report of Professor Robert Ouvrier (28 October 2002) p 5.

624 10 April 2003 T514.18-24; 23 April 2003 T876.15.

625 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 10.

Sarah

Sarah's birth and medical history

333. Sarah was born at 39 weeks' gestation (full term) on 14 October 1992.[626] She weighed 3,020 grams at birth and was discharged the same day.[627] She was bottle feeding on discharge.[628]

334. Sarah had a sleep study carried out when she was three weeks of age.[629] Dr Cooper, respiratory and sleep physician, supervised the study at the John Hunter Hospital, which involved a pneumogram with oximetry (oxygen saturation) performed on 5 November 1992.[630] The oximetry was normal, and there were very few sleep apnoeas recorded and some periodic breathing detected; however, the results were judged to be normal.[631] Dr Cooper opined that Sarah did not exhibit signs of a respiratory control problem.[632]

335. At trial, Dr Cooper gave evidence that the sleep study showed a very small handful of apnoeas which were normal for infants of that age.[633] He could not tell whether the apnoeas were central or obstructive, but in an infant of her age they were "very likely" to be central.[634] The apnoeas that he observed in Sarah, particularly with his experience at the time of trial, "goes along with a normal study for age in a child at this age."[635] His conclusion was that "certainly, the sleep study conducted on Sarah was normal for her age."[636]

336. On 16 November 1992, Dr Cooper wrote to a paediatrician, Dr Henry, in relation to Sarah, referring to "quite long hyperventilation [rapid or deep breathing], hypoapnoea [shallow breathing] event, one of them about 40 seconds."[637] Under cross-examination at trial, Dr Cooper said that at the time, "a number of us, me included" were concerned.[638] However, there were no follow up steps taken, and Dr Cooper said that if he had been very worried, steps would have been taken. He said that he was "really under these circumstances identifying some minor changes to a colleague and leaving them in his hands."[639] It was a shallow breathing pattern, but in the absence of oxygen saturation changes, this was recognised as "just perfectly normal and part of sort of life's evolution in the first year."[640]

337. Dr Marley, a general practitioner, saw Sarah four times during her life for routine reasons such as vaccinations.[641] She appeared to him to be a normal healthy infant.[642]

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626 Exhibit H, Forensic pathology tender bundle, Perinatal record of Sarah (14 October 1992).
627 Exhibit H, Forensic pathology tender bundle, Perinatal record of Sarah (14 October 1992).
628 Exhibit H, Forensic pathology tender bundle, Perinatal record of Sarah (14 October 1992).
629 Exhibit H, Forensic pathology tender bundle, Statement of Dr David Cooper (6 December 1999) [10], Sleep study report of Sarah.
630 Exhibit H, Forensic pathology tender bundle, Statement of Dr David Cooper (6 December 1999) [10].
631 Exhibit H, Forensic pathology tender bundle, Statement of Dr David Cooper (6 December 1999) [10].
632 Exhibit H, Forensic pathology tender bundle, Statement of Dr David Cooper (6 December 1999) [11].
633 14 April 2003 T589.6-12, T595.49-50; Exhibit H, Forensic pathology tender bundle, Statement of Dr David Cooper (6 December 1999) pp 2-3.
634 14 April 2003 T588.49-54.
635 14 April 2003 T589.6-12.
636 14 April 2003 T589.20.
637 14 April 2003 T595.52-596.34.
638 14 April 2003 T596.37-38.
639 14 April 2003 T597.21-27.
640 14 April 2003 T597.31-44.
641 11 April 2003 T540.6-25.
642 11 April 2003 T540.56.

Sarah's death

338. As noted in Chapter 3, the circumstances of Sarah's death at ten months and 16 days were disputed at trial. Relevantly, Mr Folbigg said he put her in her bed, located in the Folbiggs' room, at about 10:30 or 11:00pm, and was woken sometime after 1:10am by Ms Folbigg standing at the door of the room screaming.

339. Ms Deborah Martin, ambulance officer, attended on 30 August 1993, arriving at the Folbigg home at about 1:30am.[643] When she walked into the bedroom, she saw Mr Folbigg performing CPR on Sarah.[644] Ms Martin returned to the ambulance for equipment and when she came back she saw that Sarah was cyanosed (blue) around the mouth, and had mucus and vomit in her mouth.[645] Sarah was not breathing.[646] When she first arrived, Ms Martin was unable to determine Sarah's body temperature.[647] Ms Martin gave her adrenaline and Hartmann's fluid, then additional paramedics (Ms Louise Alderson (née Bishop), Mr Robert Foxford and Mr Rodney Avery) arrived.[648]

340. At approximately 2:10am, the officers stopped the drugs and CPR because Sarah was asystolic (no mechanical or electrical activity of the heart).[649]

341. An Ambulance Service treatment report by Ms Martin recorded that Ms Folbigg told her that Sarah had not been awake since placing her in bed "this PM", and that Sarah usually wakes at midnight, that Ms Folbigg had checked her as she had not woken, and that the parents were doing CPR on Ms Martin's arrival.[650] Ms Martin recorded that the airway was obstructed, breathing and circulation were absent, that skin temperature was both normal and cold, and that she had a small amount of vomiting.[651] Another ambulance report, by Ms Alderson and Mr Foxford, referred to the death as "?SIDS" and noted that the death was the parents' third SIDS.[652] They recorded that Sarah's airway was clear, that breathing was absent, circulation was absent, and that her skin temperature was cold.[653]

342. A report of death to the coroner (P79A form) was signed by Senior Constable Saunders and dated 30 August 1993.[654] The narrative recorded on the form noted that Sarah was the family's third natural child, and the previous two were both SIDS victims within the previous five years.[655] It noted that she had been in good health apart from a flu or cold type virus, which had been treated by Dr Marley with a prescription of flucloxacillin 125mg/5ml, prescribed on 18 August 1993 and last taken by Sarah about 26 or 27 August 1993.

643 Exhibit H, Forensic pathology tender bundle, Statement of Deborah Martin (8 October 1999) p 2; 11 April 2003 T566.58-567.9.
644 Exhibit H, Forensic pathology tender bundle, Statement of Deborah Martin (8 October 1999) p 2; 11 April 2003 T567.21-29.
645 Exhibit H, Forensic pathology tender bundle, Statement of Deborah Martin (8 October 1999) p 3; 11 April 2003 T567.39-43, T568.3-9.
646 Exhibit H, Forensic pathology tender bundle, Statement of Deborah Martin (8 October 1999) p 3; 11 April 2003 T568.11-12.
647 Exhibit H, Forensic pathology tender bundle, Statement of Deborah Martin (8 October 1999) p 6.
648 Exhibit H, Forensic pathology tender bundle, Statement of Deborah Martin (8 October 1999) p 4; 11 April 2003 T568.14-19; Exhibit H, Forensic pathology tender bundle, Statement of Louise Alderson (1 September 1999) p 2.
649 Exhibit H, Forensic pathology tender bundle, Statement of Deborah Martin (8 October 1999) p 4; 11 April 2003 T568.24-31.
650 Exhibit H, Forensic pathology tender bundle, Ambulance Report Q604 (30 August 1993).
651 Exhibit H, Forensic pathology tender bundle, Ambulance Report Q604 (30 August 1993).
652 Exhibit H, Forensic pathology tender bundle, Ambulance report Q001 (30 August 1993).
653 Exhibit H, Forensic pathology tender bundle, Ambulance report Q001 (30 August 1993).
654 Exhibit H, Forensic pathology tender bundle, Report of death to Coroner (30 August 1993).
655 Exhibit H, Forensic pathology tender bundle, Report of death to Coroner (30 August 1993).

343. The P79A stated that Sarah was put to sleep in a single bed in the parents' bedroom at about 9:00pm on 29 August 1993. She was snoring when her parents went to bed at 9:30 or 10:00pm. The form also recorded the reported circumstances of Ms Folbigg getting up at 1:30am, not hearing Sarah breathing, turning on the light and seeing that she had blue colour to the skin on her face, and a discharge from the nose.[656]

Autopsy report

344. The post-mortem examination of Sarah was carried out at 8:00am by Professor Hilton at the New South Wales Institute of Forensic Medicine. Professor Hilton prepared an autopsy report dated 25 November 1993.[657]

345. Professor Hilton concluded that the cause of Sarah's death was SIDS.[658] The pathology summary recorded:

> *1. Focal pulmonary collapse*
>
> *2. Modest pulmonary congestion and minimal oedema*
>
> *3. Occasional petechiae on pleura, epicardium and on and in thymus*
>
> *4. Congested ? haemorrhagic uvula lying anterior to the epiglottis*
>
> *5. Aspiration of gastric content (?artifactual)*[659]

Key findings on external examination

346. Professor Hilton's report recorded the body was that of a well-nourished, clean Caucasian female. There was minor abrading and drying of the lips. There were two tiny punctate abrasions present, one immediately below the lower lip on the left side, the other slightly to the left side of the midline of the chin.

347. A 1.5cm scratch was present on the anterolateral aspect of the right upper arm.

Key findings on internal examination

348. The uvula was of normal size but appeared somewhat congested/haemorrhagic on its anterior surface. When viewed at post-mortem, it was placed anterior to the epiglottis, producing an obstructive element in the airway. The epiglottis itself appeared relatively normal.

349. The lungs both showed focal areas of collapse of a geographic pattern. Occasional petechial haemorrhages were present and there was minor congestion and minimal oedema (fluid retention).

350. The heart was normal in size, shape and location. Layers of the pericardium were separate. No pericardial effusions (fluid around the heart) were present. There was a very occasional petechinum present on the epicardium. The atria were normal.

351. The thymus showed occasional petechial haemorrhages on its surface and within the substance of the gland but was normal in size shape and location.

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656 Exhibit H, Forensic pathology tender bundle, Report of death to Coroner (30 August 1993).
657 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Sarah (25 November 1993).
658 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Sarah (25 November 1993) p 5.
659 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Sarah (25 November 1993) p 5.

352. The following histology was reported:

> *Source: Lung*
>
> *Profuse Coliform*
>
> *Profuse Streptococcus, Alpha Haemolytic*
>
> *Scanty Staphyolococcus Aureus*
>
> *Source: Spleen*
>
> *Moderate Coliforms Of 3 Colonial Types.*[660]

353. In addition, it was noted that "one section of larynx shows a light mixed lymphocytic inflammatory infiltrate deep to the respiratory epithelium" and "in one section there is a light interstitial acute inflammatory infiltrate which could be seen around the occasional bronchiole".[661]

Death certificate

354. The death certificate issued in respect of Sarah's death, recorded "Sudden Infant Death Syndrome" as the cause of death.[662]

Evidence at the time of trial

Abrasions

355. The two tiny punctate abrasions below Sarah's bottom lip, noted by Professor Hilton, were "an extremely superficial injury to the outmost layer of skin of a pinpoint size", and were within a centimetre or two of the lower lip and were recent in origin.[663] They were consistent with the application of "very minor" force, either by Sarah or by someone else.[664] Professor Hilton considered these to be of little, if any, significance, even being aware of Caleb's and Patrick's deaths.[665] Under cross-examination, he explained that children at 10 months do scratch themselves, and Sarah had been the subject of resuscitative measures – there were at least two explanations for the marks.[666] He considered that a soft toy with hard bits on it would have been highly unlikely to cause the marks, and it was unlikely they were caused by a hand with a ring on it.[667]

356. The only other external finding was a 1.5cm scratch, which was recently made, on Sarah's right upper arm.[668] It was on an area of the body that Sarah could have reached with her own fingers.[669]

357. As discussed further below, the existence of these abrasions as described by Professor Hilton carried varying weight with the experts opining on the cause of Sarah's death.

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660 Exhibit H, Forensic pathology tender bundle, Microbiology reports of Sarah (13 September 1993, 21 September 1993).
661 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Sarah (25 November 1993) p 7.
662 Exhibit H, Forensic pathology tender bundle, Death certificate of Sarah (29 August 1993).
663 14 April 2003 T617.17-50, 24 April 2003 T918.14-25; Exhibit H, Forensic pathology tender bundle, Final autopsy report of Sarah (25 November 1993) p 3.
664 14 April 2003 T617.52-618.2.
665 14 April 2003 T652.13-20; 24 April 2003 T909.3-6, T913.52-915.14, T918.6-21.
666 14 April 2003 T652.15-20.
667 24 April 2003 T917.18-58.
668 14 April 2003 T616.34-37, T652.43-45; Exhibit H, Forensic pathology tender bundle, Final autopsy report of Sarah (25 November 1993) p 2.
669 14 April 2003 T652.26-31.

Internal petechial haemorrhages

358. The internal examination showed an occasional petechial haemorrhage on the lungs, minor congestion and minimal oedema, and also an occasional petechial haemorrhage on the surface of the heart and on the thymus gland.[670] Professor Hilton gave evidence under cross-examination that location of petechia in three separate organs, and changes to the lungs, taken together tended to favour a SIDS diagnosis.[671] The little haemorrhages on the lungs indicated to him the possibility of an asphyxial mode of death, meaning by an outside party or an internal form of asphyxiation.[672] He had only come across one case of reasonably conclusive deliberate suffocation that showed such haemorrhages.[673]

359. In evidence in chief at trial, Professor Hilton said that the findings of the internal examination were consistent with an asphyxial mode of death, which would include deliberate smothering and other things – there was nothing very specific about the little haemorrhages.[674] It was a finding that had been recorded in cases of smothering, and in a "limited sense" was indicative; attempts to breathe were thought to cause the very smallest blood vessels to rupture and bleed, from a negative pressure from the attempts to inhale.[675]

360. Under cross-examination on this point, Professor Hilton said that none of the typical signs or injuries that could be found when somebody is intentionally suffocated were found on Sarah, haemorrhages on the lungs not being specific for smothering (although indicating the possibility of an asphyxial mode of death).[676] Signs detectible upon autopsy may be the presence of petechia on locations outside the chest, on the eyelids, on the cheeks and on the surface of the eyes.[677] He said there may also be damage to the fraenulum, a tiny membrane inside the lip, both top and bottom; if there is force applied to the lips, the fraenulum may tear.[678] There may also be bruising on the inside of the lips from compression.[679]

Uvula

361. Professor Hilton identified that Sarah's uvula was reddened (which he said in evidence was consistent with mild infection, or with snoring).[680] Its tip was lying in front of the epiglottis, which he had never previously seen in a post-mortem examination of the 2,000-plus babies he had examined.[681] The significance of this was questionable; Professor Hilton said in oral evidence that he had grave doubts that the uvula was in that position before Sarah died.[682] He said that the presence of the inflamed uvula was a "brick in the diagnostic wall" for SIDS in Sarah's case.[683]

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670 14 April 2003 T618.53-619.12, T620.4-17; Exhibit H, Forensic pathology tender bundle, Final autopsy report of Sarah (25 November 1993) p 3.
671 14 April 2003 T653.36-45.
672 14 April 2003 T651.25-30.
673 14 April 2003 T651.50-53.
674 14 April 2003 T618.36-619.27.
675 14 April 2003 T608.46-49.
676 14 April 2003 T650.46-651.30.
677 14 April 2003 T650.53-56.
678 14 April 2003 T650.57-651.1.
679 14 April 2003 T651.1-4.
680 14 April 2003 T621.35-40, T623.4-10; 7 May 2003 T1182.36-45; Exhibit H, Forensic pathology tender bundle, Final autopsy report of Sarah (25 November 1993) p 3.
681 14 April 2003 T621.35-46, T622.45-48, T649.37-43.
682 14 April 2003 T623.12-20; 7 May 2003 T1183.6-13.
683 14 April 2003 T654.14-28.

362. Microscopic examination showed areas of inflammation consistent with mild respiratory infection, which may have helped explain the reddened uvula.[684] There was no evidence of any viral infection, but profuse growth of streptococcus might indicate a streptococcal infection which would help explain the reddening of the uvula and perhaps the light inflammation of the larynx (voice box).[685] Such inflammation would not normally be expected to have contributed significantly to death.[686] Otherwise, the findings said nothing about cause of death.[687]

363. Dr Beal noted that the inflamed and displaced uvula showed probable throat infection.[688] Professor Herdson doubted that Sarah's reddened uvula was significant in her death; it could have been caused during resuscitation.[689] Professor Berry had never heard of a child dying of a swollen uvula.[690] Professor Byard in his report referred to Professor Hilton's comments on an unusually congested uvula, and said in evidence that while he was unsure of the significance of the displaced uvula, he considered that it could not be excluded as having played a role in Sarah's death.[691] Professor Busuttil, forensic pathologist, considered that the presence of the congested uvula may have given rise to some upper airway obstruction.[692]

Opinions on cause of Sarah's death

364. As noted above, in the post-mortem, Professor Hilton concluded that SIDS was the direct cause of Sarah's death.[693] In oral evidence, he described SIDS as "not a disease", but as a cause of death of unknown origin, and that if there is an identifiable cause of death then a "diagnosis of SIDS cannot be applied."[694] This was in part based on negative findings, and in part on positive findings, with the negative findings being more important.[695]

365. Professor Hilton said that he conducted Sarah's autopsy on the basis that it was not "frankly suspicious".[696] He was aware of the previous death of Caleb and that no cause of death had been found and had been diagnosed as SIDS.[697] He was aware that Patrick died with a brain condition, which would indicate his propensity for convulsions. Caleb's death did cause him concern in relation to the SIDS for Sarah and he had that history in mind in his examination of Sarah, however, the features seen on Sarah tended to favour a SIDS diagnosis as opposed to intentional suffocation.[698] If there was any indication of intentional suffocation, Sarah's death would have been categorised as undetermined.[699]

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684 14 April 2003 T625.41-626.16; Exhibit H, Forensic pathology tender bundle, Final autopsy report of Sarah (25 November 1993) p 7.
685 14 April 2003 T627.35-628.24.
686 14 April 2003 T628.26-35.
687 14 April 2003 T627.9.
688 5 May 2003 T1142.22-23.
689 1 May 2003 T1038.23-34.
690 Responses to Crown model questions – Professor Berry, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.
691 7 May 2003 T1215.51-1216.6, T1240.47-57; Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 6.
692 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 13.
693 14 April 2003 T628.41-43; Exhibit H, Forensic pathology tender bundle, Final autopsy report of Sarah (25 November 1993) p 5.
694 14 April 2003 T628.45-57.
695 14 April 2003 T629.6-11.
696 14 April 2003 T633.29-44.
697 14 April 2003 T634.3-16. 24 April 2003 T914.6-58.
698 14 April 2003 T634.23-40 (Caleb), T653.6-13; 24 April 2003 T914.18-49 (Sarah).
699 14 April 2003 T653.30-654.13.

366. Professor Hilton agreed that circumstances that should indicate the possibility of intentional suffocation include a previous unexplained or unexpected death of one or more siblings and an ALTE of a sibling while in the care of the same person.[700] He did not agree that he ought to have diagnosed Sarah's death as "undetermined", although accepted that it may be extremely difficult at autopsy to distinguish between SIDS and asphyxiation with a soft object – the amount of force required to deliberately smother a ten-month-old with a pillow is fairly small.[701] He disagreed that Sarah had died from an acute catastrophic asphyxiating event of unknown causes, saying that on the balance of probabilities the most likely diagnosis was SIDS.[702] He agreed she died from a cessation of breathing, and that he could not say why she ceased to breathe.[703]

367. Dr Cala considered that it was not appropriate to find Sarah's death as SIDS, because of her age and the abrasions on her lower lip.[704] He would have given the cause as "undetermined".[705] He considered the post-mortem findings to be consistent with Sarah having been deliberately smothered and that they did not exclude deliberate or accidental trauma.[706] He believed it was "a possibility" that Sarah died of an acute and catastrophic asphyxiating event.[707]

368. Dr Beal gave evidence that the most likely cause of Sarah's death, in isolation, was intentional suffocation.[708] In isolation, it was appropriate to find SIDS as the cause of death, on the understanding that SIDS includes intentional suffocation. She said that Sarah died of an acute and catastrophic asphyxiating event.[709]

369. Ultimately, Dr Beal would have accepted a diagnosis of "undetermined" or SIDS.[710] Sarah was outside the usual SIDS age range and found on her back, which although non-specific, qualified a SIDS finding.[711] The findings were consistent with Sarah having died from an acute catastrophic asphyxiating event, and of having been smothered.[712]

370. In her first report, Dr Ophoven opined to "a reasonable degree of medical certainty" that Sarah did not die of SIDS because she did not fall within the age range associated with SIDS: although the classic classification of SIDS included children under one year of age, this was not the age range accepted by most forensic pathologists.[713] A sudden unexpected death, greater than six months old, was "atypical" and by one year of age, SIDS would be excluded.[714]

371. In her report, Dr Ophoven considered that Sarah's death was most consistent with suffocation, and that Sarah was the victim of "probable homicide"; the cause of death on the autopsy findings were inconsistent with SIDS but consistent with suffocation.[715] Dr Ophoven's reasons were similar to those she gave in relation to Patrick and excluding references to seizures as a cause of death.[716]

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700 14 April 2003 T648.17-34.
701 14 April 2004 T649.4-12, T653.30-34, T655.54-656.6.
702 14 April 2003 T649.14-19.
703 14 April 2003 T649.21-29.
704 16 April 2003 T748.6-25.
705 16 April 2003 T747.38, T748.4.
706 16 April 2003 T747.44-58.
707 16 April 2003 T749.4.
708 Responses to Crown model questions – Dr Beal, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C; 28 April 2003 T989.56-990.26.
709 Responses to Crown model questions – Dr Beal, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C; 28 April 2003 T989.56-990.26.
710 5 May 2003 T1142.3-13.
711 5 May 2003 T1148.47-55.
712 5 May 2003 T1142.10-13, T1142.25-32.
713 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 9.
714 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 9.
715 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) pp 8, 9.
716 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 9.

372. Professor Herdson opined that Sarah's case was close to the SIDS criteria – she was in the correct age group.[717] However, diagnosis of the cause of death was dependent upon the nature of the punctate abrasions and scratch marks noted at autopsy; if they were obvious and apparently significant, Professor Herdson would have diagnosed the cause of death as undetermined.[718] The post-mortem findings were consistent with suffocation.[719] Professor Herdson agreed that Sarah died of a sudden acute catastrophic asphyxiating event of unknown causes.[720]

373. In his report, Professor Herdson concurred with Professor Hilton that the findings in relation to Sarah, taken in isolation, could be diagnosed as SIDS, and also concurred with analyses by Professor Berry and Dr Ophoven.[721]

374. In his transcribed answers to the model questions, Professor Herdson stated that he would have diagnosed Sarah's death as SIDS.[722] Deliberate smothering could not be excluded. He considered that although SIDS was a diagnosis of exclusion, Professor Hilton had excluded trauma in isolation.[723] Again in isolation, Professor Herdson could not get past SIDS, but once he knew of two other deaths in the family, he would have to review the diagnosis. The abrasions would have alerted him to wonder, depending on the circumstances. Professor Herdson agreed that Professor Berry's histopathology and toxicologic analysis failed to give a cause for her death.[724]

375. Professor Berry said in oral evidence before the jury that one does find abrasions on children who die of SIDS, because resuscitation has been attempted.[725] Sarah was an unusual age for SIDS, but in isolation (and consistently with his opinion in his report) he would have given the cause of death as SIDS with a slight misgiving about her age.[726] In evidence he agreed that the findings were consistent with suffocation.[727] In his report he noted it was of concern that Mr Folbigg had recounted that Ms Folbigg had considerable tension on the evening that Sarah died; it appears that this contributed to Professor Berry's misgivings about SIDS as a diagnosis.[728]

376. In his transcribed answers to the model questions, Professor Berry stated that taken in isolation, Sarah's death resembled SIDS. But at 10 months of age, there was need for caution, and "Craig's account must be considered." Professor Berry would "probably say SIDS, but with misgivings." He would not want to put the family through a police investigation, and would tend to err on the side of caution. Professor Berry could not exclude deliberate or accidental suffocation of Sarah. Sarah could have died of an acute and catastrophic asphyxiating event.

377. Professor Berry reported "subsequent histological, microbiological, biochemical and toxicological examination failed to give a cause for her death."[729]

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717 1 May 2003 T1036.39-55.

718 1 May 2003 T1036.48-1037.9, T1045.45-51.

719 1 May 2003 T1038.46-48.

720 1 May 2003 T1038.53.

721 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Herdson (17 January 2002) p 2.

722 Responses to Crown model questions Professor Herdson, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.

723 Responses to Crown model questions – Professor Herdson, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.

724 Exhibit H, Forensic pathology tender bundle, Statement of Professor Peter Herdson (17 January 2002) p 2.

725 1 May 2003 T1063.40-51.

726 1 May 2003 T1063.53-1064.11; Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 24.

727 1 May 2003 T1065.42-44.

728 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 24.

729 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 25.

378. Professor Byard would have diagnosed her death as "undetermined" because of the uvula and lack of description of the death scene – putting these matters aside, the findings would be consistent with SIDS.[730] He could not exclude deliberate smothering, there being no symptoms or findings on her autopsy; neither was there any finding or symptom that could amount to proof of suffocation.[731]

379. Professor Busuttil reported that "some bacteria – especially important being *Staphylococcus aureus* – were isolated from her airways at autopsy."[732] This bacterium is "not infrequently found in SIDS".[733] He considered that Sarah's death, of the four, most approximated a typical SIDS death, although he noted that internal features associated with SIDS were not found.[734] There may have been some upper airway obstruction from the congested uvula.[735]

Evidence in the Inquiry

SIDS risk factors

380. In her report, Professor Horne referred to the following "potential protective factors for SIDS" in respect of Sarah: Sarah was born at term at a normal weight, was found supine with her face uncovered and in her own bed, was vaccinated and used a dummy (although the extent of which is unclear). She also referred to an increased risk in mothers younger than 26 years at the time of the baby's birth (Ms Folbigg was 25 when Sarah was born) and paternal smoking. However, Sarah also slept in a single bed in her parents' room which was considered to be additionally protective.[736]

381. In evidence in the Inquiry, Professor Horne observed that at 10 months, Sarah was older than is usual for SIDS.[737] She considered that a mild respiratory infection is common and half of babies who die have had a mild respiratory infection not severe enough to be attributed to the cause of death and Sarah was healthy apart from a cold. Professor Horne concluded that Sarah was at low risk for SIDS.[738] Professor Elder agreed, noting Sarah snored at times but otherwise the usual risk factors were absent, and Sarah's slightly older age.[739]

Abrasions

382. Professor Duflou described the two punctate (tiny dots) abrasions below Sarah's mouth as entirely non-specific and not in any way indicative of external obstruction of respiration (for example, it is totally normal for a 10 month old to cause minor injuries to their face in normal activities).[740] In Professor Cordner's report, he stated that no significance could be ascribed to them.[741] He noted that there were no photographs, histology, or other information about the abrasions. Further, there was no objective evidence to say whether they occurred before death, around the time of death, or after death. The marks could have been associated with resuscitation and this would be a reasonable explanation for them if they occurred around the time of death (contrary to the impression left by Dr Cala's evidence at trial).[742]

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730 7 May 2003 T1217.2-29.
731 7 May 2003 T1217.41-45, T1256.1-15.
732 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 13.
733 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 13.
734 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 13.
735 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 13.
736 Exhibit J, Expert report of Professor Rosemary Horne (10 February 2019) pp 2-3; 2 April 2003 T126.38.
737 Transcript of the Inquiry, 18 March 2019 T38.13-14.
738 Transcript of the Inquiry, 18 March 2019 T38.14-21.
739 Transcript of the Inquiry, 18 March 2019 T38.25-29.
740 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 33; Transcript of the Inquiry, 20 March 2019 T173.4-8.
741 Exhibit Q, Report of Professor Stephen Cordner (undated) p 50; Transcript of the Inquiry, 20 March 2019 T173.12-13.
742 Exhibit Q, Report of Professor Stephen Cordner (undated) p 50; Transcript of the Inquiry, 20 March 2019 T173.12-13.

383. Dr Cala noted in his report that Professor Hilton recorded apparent minor abrading and drying of the lips, normal fraenulum of the lips, two tiny punctate abrasions, and that Sarah appeared well-nourished.[743] He stated that he would question the minor abrading and drying of the lips but these may have been related to resuscitation attempts.[744]

Uvula

384. In the Inquiry, Professor Cordner considered that the uvula put Sarah in a "slightly more risky" category of infant, "a bit more vulnerable to SIDS".[745] He said that this, and histology factors – small signs of infection around some small airways, very small signs of possible inflammation in the lymph nodes draining the lungs and in the salivary glands, interesting change in the muscle fibres of the diaphragm – did not, even collectively, add to anything close to a cause of death but "sort of shows that, at some sort of level, there is abnormality".[746] He agreed that all of these factors could be accounted for by having a cold; and it may be that a cold "makes you more vulnerable to SIDS".[747] While Professor Cordner said that for him, having a preceding cold is "still part of the story of SIDS", he also referred in his report to literature that a history of a minor respiratory infection in the days leading up to death is no longer a consistent finding.[748]

385. Professor Hilton said that at autopsy the uvula was congested, red and bleeding a little, with microscopic evidence of inflammation.[749] It was not elongated.[750] The inflammation on microscopic examination was consistent with a mild respiratory infection.[751]

386. When Professor Hilton saw Sarah's uvula at autopsy, after neck organs had been removed, it overlapped the epiglottis. He said in the Inquiry that neither then, nor now, could he be certain that this was not a post-mortem artefact, although he said this was a "very real possibility".[752] He considered it was possible – "and I put it no more than that" – that Sarah's snoring was because the uvula was bouncing off the epiglottis or larynx.[753] In the Inquiry, he produced a paper identifying one case of death from a uvula overlapping an epiglottis, saying that this research was mildly supportive of this as a cause in relation to Sarah.[754]

387. Dr Cala opined that the uvula had no bearing on cause of death.[755] However, Professor Duflou cited it as a reason, together with Sarah's age, that he would ascribe her death as Category 2 SIDS. He also said that he did not know if the uvula was significant.[756]

743 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 12.
744 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 13; Transcript of the Inquiry, 20 March 2019 T172.45-46.
745 Transcript of the Inquiry, 20 March 2019 T177.37-42.
746 Transcript of the Inquiry, 20 March 2019 T178.1-7.
747 Transcript of the Inquiry, 20 March 2019 T178.15-16.
748 Exhibit Q, Report of Professor Stephen Cordner (undated) p 30; Transcript of the Inquiry, 20 March 2019 T178.24.
749 Transcript of the Inquiry, 20 March 2019 T175.42-176.28.
750 Transcript of the Inquiry, 21 March 2019 T303.14-42.
751 14 April 2003 T625.34-626.16; Exhibit H, Forensic pathology tender bundle, Final autopsy report of Sarah (25 November 1993) p 7.
752 Transcript of the Inquiry, 20 March 2019 T175.42-46; T234.38.
753 Transcript of the Inquiry, 20 March 2019 T176.7-12.
754 Transcript of the Inquiry, 20 March 2019 T175.49-176.5; 21 March 2019 T241.45-242.19; T Marom et al, 'Otolaryngological Aspects of Sudden Infant Death Syndrome' (2012) 76(3) *International Journal of Pediatric Otorhinolaryngology* 311.
755 Transcript of the Inquiry, 20 March 2019 T176.38.
756 Transcript of the Inquiry, 20 March 2019 T177.27-33.

Opinions on cause of Sarah's death

388. Professor Duflou would give Sarah's death as SIDS Category 2 on the basis of her age (i.e. greater than nine months) and the abnormality of the upper airway which he considered had the potential to obstruct the flow of air.[757] In his report he noted that SIDS Category 2 can be given where there has occurred death of siblings while in the custody of the same caregiver.[758]

389. In his peer review of Professor Cordner's report, Professor Pollanen noted that there were no positive pathologic findings, and ascribed Sarah's death as "Class 5" of the system referred to above at [131].

390. In his report, Dr Cala stated that he would give the cause of death of Sarah as "undetermined" and would not give SIDS.[759] Dr Cala's reasons were:

    a. the apparent abnormalities of the uvula were non-specific findings but were of some concern, although Dr Cala was not aware of any literature which describes elongation of a uvula with fatal consequences. In oral evidence in the Inquiry, Professor Hilton clarified that the uvula was reddened, but not elongated[760] and Dr Cala stated "I don't think it has got any bearing on the cause of death";[761]

    b. that he would question the minor abrading and drying of the lips; and

    c. the previous two deaths alone would make Dr Cala extremely cautious about Sarah's death being due to SIDS.[762]

391. Professor Cordner would give Sarah's death as SIDS Category 2, opining he considered that Sarah's death falls squarely into the definition.[763] So saying, he considered that Professor Hilton correctly anticipated the sort of approach taken by Krous et al (2004) when he gave SIDS as the cause of Sarah's death (in 1992).[764]

Time of death

392. In his report, Professor Duflou noted that Sarah's skin temperature was described by ambulance officers as either normal or cold, and the stomach contents at autopsy were described as moderate in quantity and consisting of curdled milk with or without egg white. He stated that this would suggest that Sarah died closer to the time she was put to bed by Mr Folbigg at around 9:00pm, rather than when found by Ms Folbigg at around 1:30am. He disagreed with Professor Hilton's finding at autopsy that the time of death was 1:30am.[765]

393. It appears that Professor Duflou was not provided with the evidence of Senior Constable Stephen Saunders, who was a police officer at the time of Sarah's death, and who interviewed Mr and Ms Folbigg. He was told that the parents again entered that room about 9:30 or 10:00pm, and Sarah was heard to be snoring, and that Ms Folbigg heard Sarah turn over in her sleep at about 12:00 or 12:30am.[766]

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757 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 33; Transcript of the Inquiry, 20 March 2019 T179.31-38.

758 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 33; Transcript of the Inquiry, 20 March 2019 T179.38-39.

759 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 13.

760 Transcript of the Inquiry, 21 March 2019 T303.14-42.

761 Transcript of the Inquiry, 20 March 2019 T176.38.

762 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 13.

763 Exhibit Q, Report of Professor Stephen Cordner (undated) p 83; Henry F Krous et al, 'Sudden Infant Death Syndrome and Unclassified Sudden Infant Deaths: A Definitional and Diagnostic Approach' (2004) 114 *Pediatrics* 234.

764 Exhibit Q, Report of Professor Stephen Cordner (undated) p 83.

765 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 33.

766 11 April 2003 T574.57-58.

Submissions of counsel assisting on cause of Sarah's death

394. As with Caleb, counsel assisting submitted that there have been two material changes since the 2003 trial. First, genetic testing has been completed and no genetic variant which is pathogenic or likely pathogenic has been identified to account for Sarah's death. Secondly, more recent research on SIDS that maternal smoking and sleeping position pose the highest risks relevantly lowers, in retrospect, the risk posed to Sarah of SIDS.

395. In addition, counsel assisting referred to evidence heard in the Inquiry of the role that infection may have played in Sarah's death, and submitted that Sarah's cause of death cannot be attributed to infection. This topic is considered further in Chapter 6.

396. Counsel assisting submitted that the forensic pathology evidence does not identify any natural cause of Sarah's death.

397. The only evidence of the possible role of the uvula was that it "could have" caused death or is "not excluded" as a cause. Professor Cordner and also Professor Duflou referred to it to say that she was more vulnerable to or at higher risk of SIDS, Professor Cordner referring to it alongside small signs of possible inflammation to opine that they increased Sarah's risk although did not come close to a cause of death.[767]

398. Counsel assisting noted that expert evidence both at trial and in the Inquiry as to the significance of the abrasions largely endorsed Professor Hilton's observation that the abrasions could have been due to resuscitation.

399. Counsel assisting submitted, however, that there is no evidence other than that Sarah was a low risk of SIDS, noting that SIDS itself is a rare occurrence. Relevantly, Sarah's sleep study was essentially normal. Ultimately, counsel assisting submitted, there remains no identified natural cause of Sarah's death.

400. None of the forensic pathology or medical experts at trial excluded smothering as a possible cause of Sarah's death.[768]

Ms Folbigg's submissions on cause of Sarah's death

401. Ms Folbigg submitted that Sarah's death could have been caused by obstruction caused by the uvula, laryngospasm triggered by the uvula, cardiac arrhythmia/cytokine response triggered by mild infection or bed-sharing.

402. It was contended that the first three of these arise from significant advances in medical knowledge raising a "distinct possibility of a natural death due to a number of features".[769]

403. Ms Folbigg noted that prior to her death, Sarah had been unwell for a week or so with a cold-like illness. The autopsy report identified five features that demonstrated health issues.[770]

404. In her submission, Professor Hilton changed his opinion considerably in the Inquiry compared with at trial.[771] At trial he said the presentation of Sarah's uvula at autopsy was likely a post-mortem artefact and incidental to cause of death.[772]

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767 Transcript of the Inquiry, 20 March 2019 T177.37-43, T178.1-179.3, T179.25-26.

768 16 April 2003 T747.44, T749.4 (Dr Cala); 5 May 2003 T1142.10-13, 25-32 (Dr Beal); 1 May 2003 T1038.46-48 (Professor Herdson), T1065.42-44 (Professor Berry); 7 May 2003 T1217.41-45, T1256.1-15 (Professor Byard).

769 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [20].

770 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [5].

771 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [18] n 19.

772 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [7]; 14 April 2003 T622.1-10.

It was in the correct position and, consistently with infection and abnormal movements such as in snoring, was red.[773] Two groups of germs were grown from the lung, streptococcus with profuse growth and staphylococcus in scanty proportions.[774] He considered the staphylococcus to be a contaminant.[775]

405. In the Inquiry, Professor Hilton referred to literature reporting cases of a uvula causing sudden infant death as it became stuck in or to the epiglottis.

406. In Ms Folbigg's submission, the structure of the upper respiratory tract in infants is subject to considerable change, with important new evidence on this in the literature.[776] Inflammation in Sarah's uvula could have been from repeated contact with the epiglottis; if her uvula had struck the epiglottis, it was possible it could have blocked the airway, causing death. Alternatively, trauma caused by the uvula sticking to the epiglottis (e.g. in snoring) could cause a laryngospasm in turn causing obstruction. Alternatively again, the uvula could have reflected an infective response.[777]

407. In light of evidence in the Inquiry, Ms Folbigg suggested that Dr Beal's assertion that the swollen uvula could have been at most a post-mortem artefact was incorrect.[778] Professor Hilton said it could have become swollen due to sticking or getting caught behind the epiglottis.[779]

408. Further, the connection between mild infection and SIDS was not in evidence at trial but the Inquiry has Professor Blackwell's opinion evidence that the swollen uvula was indicative of an infective process. Undermining the Crown case that Sarah was otherwise in good health, an association between mild infection and sudden death in infancy suggests that Sarah's infection was potentially fatal.[780]

409. Ms Folbigg referred to new evidence in the Inquiry that in half of SIDS deaths there is a recent history of mild viral illness, and that this had been consolidated by evidence that a mild infection can trigger a sudden arrhythmia that can cause sudden death, adduced from Professors Blackwell, Clancy and Goldwater, and in Chapter 30 of Duncan and Byard (2018).[781] In particular, Professor Blackwell opined that the swollen uvula indicated that the inflammatory response had been triggered.[782]

410. On the Crown case at trial, Professor Hilton did not think the uvula was the cause of death; Professors Byard and Berry had no experience of it causing death; and there was no recorded case in medical literature.[783] In the Inquiry, Dr Cala expressed some concern about it and Professor Hilton gave evidence to the effect that it could have caused an obstruction or triggered laryngospasm – thus resiling from his certainty at trial.[784] Overall, evidence in the Inquiry made it clear that there have been reported incidents of sudden death from a displaced uvula; there were sound otolaryngological reasons for this risk, there was also a risk of laryngospasm.[785]

411. Ms Folbigg also raised the possibility that Sarah may have accidentally suffocated from sharing Mr and Ms Folbigg's bed. Detective Senior Constable Ward, who attended on the night of Sarah's death, recorded things he was told by Senior Constable Saunders, including that:

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773 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [7]; 14 April 2003 T622.32-42, T622.51-623.28.

774 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [11]; 14 April 2003 T627.30-56.

775 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [11]; 14 April 2003 T627.58-628.14.

776 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [18(b)], citing T Marom et al, 'Otolaryngological Aspects of Sudden Infant Death Syndrome' (2012) 76(3) *International Journal of Pediatric Otorhinolaryngology* 311.

777 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [18(c)-(f)].

778 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [12].

779 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [12]; Transcript of the Inquiry, 20 March 2019 T233.43-234.32; 21 March 2019 T241.28-243.9.

780 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [14].

781 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [21].

782 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [23].

783 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [15]; see 13 May 2003 T1340.38-1341.3.

784 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [25]; see 14 April 2003 T621.32-52.

785 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [26].

> *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.*[786]

412. Ms Folbigg submitted it should be inferred that Detective Senior Constable Ward's information was accurate and accepted that his statement was clear, unambiguous and contemporaneous, whereas Mr and Ms Folbigg's contrary evidence (his at trial, and hers in her ERISP) were some years after the event. If accepted, it gives rise to a potential natural cause of death, with a risk she was overheated (supporting bacteria reproduction) and also that she was accidentally smothered.

Professor Hilton's submissions on cause of Sarah's death

413. In his submissions in response, Professor Hilton said that Ms Folbigg's submissions fairly reflected his opinion as to the possible cause of death of Sarah, with one exception: he maintained that Sarah's cause of death was SIDS and "the issue of the uvula was an alternative point of view."[787]

Findings: Sarah

414. Two material changes since the 2003 trial relevant to Sarah's death are first, that genetic testing has been completed and no genetic variant which is pathogenic or likely pathogenic has been identified to account for Sarah's death. Secondly, more recent research on SIDS that maternal smoking and sleeping position pose the highest risks relevantly lowers, in retrospect, the risk posed to Sarah of SIDS.

415. Ultimately there remains no identified natural cause of Sarah's death.

416. Forensic pathology evidence does not identify as a reasonable possibility any natural cause. Sarah's uvula having been found at autopsy to overlap the epiglottis, forensic pathology evidence is to the effect that this "could have" caused death or is "not excluded" as a cause. That is, it is possible obstruction by the uvula or because of the uvula caused Sarah's death, although "possible" is a conservative statement of the likelihood. While Professor Hilton revised his opinion in the Inquiry compared with at trial, I do not agree that his revision was as considerable as Ms Folbigg contended. His evidence in the Inquiry was that even now, he cannot be certain the uvula overlapping the epiglottis was not a post-mortem artefact. His discovery of a paper of a previous reported case was mildly supportive that it may not have been a post-mortem artefact.[788] Dr Cala agreed it might represent an artefact but in his opinion it had nothing to do with Sarah's death.[789]

417. Professors Duflou and Cordner did not add significantly to this evidence, although both opined it meant that Sarah was more vulnerable to or at higher risk of SIDS (Professor Cordner referring to it alongside small signs of possible inflammation).[790]

418. In relation to SIDS, similarly for the reasons I have recorded in relation to Caleb above at [102], particularly with low risk factors, "SIDS" (and particularly "Category 2 SIDS") is far from complete as an explanation for Sarah's death. It leaves open the possibility of an identifiable (but not identified) natural or unnatural cause. Sarah had a low risk of SIDS, noting that SIDS itself is a rare occurrence. She was outside the age range in which SIDS most commonly occurs. Her sleep study was essentially normal.

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786 Exhibit BQ, Statement of Detective Senior Constable Glen Ward (14 December 1999) p 2.
787 Submissions of Professor Hilton to the Inquiry (18 June 2019) [1].
788 Transcript of the Inquiry, 20 March 2019 T176.1-5.
789 Transcript of the Inquiry, 20 March 2019 T176.38-46.
790 Transcript of the Inquiry, 20 March 2019 T177.37-43, T178.1-179.3, T179.25-26.

419. Ms Folbigg's submission that evidence regarding the potential role of infection is new and was not adduced (except in a limited way) at trial is correct. I have recorded my general findings in relation to this evidence in Chapter 6 of this report. However, I find suggestions that infection caused Sarah's death to be implausible.

420. None of the forensic pathology or medical experts at trial or in the Inquiry excluded smothering as a possible cause of Sarah's death.[791]

421. I place no significance on the abrasions on Sarah's chin noted by Professor Hilton at autopsy. They could have been due to resuscitation or some other cause completely unrelated to her death.

422. The information in Detective Senior Constable Ward's statement, including that on which Ms Folbigg relies, is second hand hearsay from Senior Constable Saunders.

423. Senior Constable Saunders gave evidence that he did not speak with Ms Folbigg, so information he obtained about the events would have come from Mr Folbigg as well as family information and history from Mr Folbigg's brother.[792] Senior Constable Saunders was cross-examined about the accuracy of his statement and maintained that it was accurate as to what he was told; it was prepared within a few weeks of Sarah's death by reference to notes he took at the time while he was at the home.[793]

424. It was not suggested to Senior Constable Saunders during his evidence at trial that Sarah may have been in Mr and Ms Folbigg's bed, and he did not give any evidence to suggest that he had been told that.

425. Other available evidence on this point includes Mr Folbigg's oral evidence that when Ms Folbigg screamed and he woke up, Sarah was laying on her bed with no covers on her. Ms Folbigg was standing at the door with one hand on the door.[794] Again, it was not suggested to Mr Folbigg and he gave no evidence to say that Sarah had been in his bed at any stage that night.

426. Finally, Ms Folbigg herself said in the ERISP that when she got up to go to the toilet she "glanced over and saw the little lump in the bed", she "went over" to cover Sarah, and Sarah's "bed was in the corner".[795] She made no mention of Sarah having been in her bed that night, although referred to Sarah having shared the bed with Mr Folbigg on previous occasions.

427. Overall, I find the proposition to be contrary to more reliable factual evidence than that on which it is based. I do not accept it.

428. In view of evidence of when Sarah was heard or seen after she went to bed (and accepting the unreliability of opinions based on stomach content) I do not accept Professor Duflou's opinion as to the time of Sarah's death.

429. I find on the available medical evidence that it is only conjecture that Sarah's death on 30 August 1993 was naturally caused by obstruction of her airways associated with her uvula. I find further on the available medical evidence that it is reasonably possible that her death was caused by an event leading to obstruction of her airways by some other cause, which includes deliberate smothering.

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791 16 April 2003 T747.44, T749.4 (Dr Cala); 5 May 2003 T1142.10-13, 25-32 (Dr Beal); 1 May 2003 T1038.46-48 (Professor Herdson), T1065.42-44 (Professor Berry); 7 May 2003 T1217.41-45, T1256.1-15 (Professor Byard).

792 11 April 2003 T575.4-20, T576.32-577.4.

793 11 April 2003 T577.15-578.8.

794 2 April 2003 T131.35-52

795 Exhibit E, ERISP of Kathleen Folbigg Q269.

Laura

Laura's birth and medical history

430. Laura was born full term on 7 August 1997, weighing 3,260 grams.[796] She was breastfed for two weeks.[797] She was vaccinated and used a dummy.[798]

431. A year earlier in August 1996, Mr and Ms Folbigg were referred to Dr Christopher Seton, a sleep physician with SIDS expertise formerly at the Royal Alexandra Hospital for Children, and at the time of trial at the Sleep Disorders Unit at the then New Children's Hospital, Westmead.[799] Mr and Ms Folbigg saw Dr Seton for advice at that stage about the potential risk of SIDS after the three previous deaths, and then returned to him after Laura was born.[800] Dr Seton gave evidence at trial, and a statement dated 23 November 1999.

432. Soon after she was born on 19 August 1997, Laura was tested for various diseases, which returned normal results.[801] A sleep study showed mild central apnoea and no obstructive apnoea, which improved on subsequent studies and, Dr Seton stated in his statement, "was totally normal by February 1998."[802] He said in evidence at trial that central apnoea is very common in premature babies, but not in full term babies.[803]

433. Dr Seton reported on the 19 August 1997 consultation in a letter to a Dr Quentin King dated 27 August 1997.[804] In the letter, Dr Seton stated that an overnight polysomnogram was performed and reported on the mild central apnoea with no evidence of upper airway obstruction or bradycardia. He stated this was a pleasing result, even though Laura's sleep-breathing was not entirely normal. He noted that most children grow out of it. He planned to re-study Laura in five to six weeks' time. A urine metabolic screen and baseline blood tests had been performed and were all normal. Laura's physical examination was also normal with no dysmorphic features. Importantly, her oropharayngeal diameter looked normal and there was no flattening of the mid-face or smallness of the chin.

434. On 7 October 1997, Dr Seton reported that Laura had moderate central apnoea of infancy.[805] He stated there had been mild improvement since the previous study, with occasional mixed apnoeas and hypopnoeas, but "the obstructive component of sleep-breathing [was] not severe."[806] There was no bradycardic (slow heart rate) response to apnoea or hypopneoa.[807]

435. By the time of a further report by Dr Seton on 17 February 1998, Laura's sleep breathing had normalised.[808] There was no evidence of upper airway obstruction in sleep, and her sleep quality was "excellent". In further correspondence dated 30 April 1998, Dr Seton recorded that her sleep breathing remained normal.[809]

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796 Exhibit H, Forensic pathology tender bundle, Birth certificate of Laura (7 August 1997); Exhibit H, Forensic pathology tender bundle, NSW midwives data collection (11 August 1997).

797 Exhibit H, Forensic pathology tender bundle, Immunisation notes of Laura (3 February 1998); 3 April 2003 T154.40-44.

798 Exhibit H, Forensic pathology tender bundle, Immunisation notes of Laura (3 February 1998); Exhibit H, Forensic pathology tender bundle, SIDS death scene investigation checklist relating to Laura (undated); Exhibit H, Forensic pathology tender bundle, Statement of Dr Paul Innis (15 March 1999) p 1; Exhibit E, ERISP of Kathleen Folbigg Q907.

799 15 April 2003 T690.3-30.

800 Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Seton (23 November 1999) pp 2-3.

801 15 April 2003 T692.1-15; Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Seton (23 November 1999) p 2.

802 15 April 2003 T692.17-22, T693.51-54; Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Seton (23 November 1999) p 2.

803 15 April 2003 T693.3-15.

804 Exhibit CA, Letter from Dr Christopher Seton to Dr Quentin King (27 August 1997).

805 Exhibit H, Forensic pathology tender bundle, Sleep study report of Laura (7 October 1997).

806 Exhibit H, Forensic pathology tender bundle, Sleep study report of Laura (7 October 1997).

807 Exhibit H, Forensic pathology tender bundle, Sleep study report of Laura (7 October 1997).

808 Exhibit H, Forensic pathology tender bundle, Sleep study report of Laura (17 February 1998).

809 Exhibit BZ, Letter from Dr Christopher Seton to Dr David Sanders (30 April 1998).

436. Monitoring of Laura's sleeps via corometric monitor showed no serious breathing problems or heart rate problems, although she was not monitored during all of them.[810] Dr Seton noted in his statement that when he saw the Folbiggs on 30 April 1998, it became clear that the monitoring was becoming tedious due to false alarms, technical difficulty, and taking a very precautionary approach in an apparently healthy baby.[811]

437. Dr John Cash, a visiting medical officer at Singleton Hospital, examined Laura several times, including at 1:00am on 22 June 1998 when she presented with a history of a slight upper respiratory infection for several days and a croupy cough.[812] Dr Cash diagnosed an upper respiratory tract infection and mild croup.[813] On his examination, he found no signs of distress or respiratory difficulties and Laura's chest was clear, but she had mucus in her throat consistent with a cold.[814] Because of the family's history, Dr Cash admitted her for observation.[815] She did not require antibiotics.[816] Notes showed that she attended his surgery on four occasions between February and May 1998. Dr Cash did not consider this unreasonable or unusual.[817]

438. Dr Paul Innis was Laura's treating general practitioner from 14 August 1998 until February 1999.[818] Dr Innis gave evidence at trial. Over the six month period he saw Laura approximately 13 times. She appeared to be a normal, healthy child with no chronic illness.[819]

439. Leading up to 14 August 1998, Laura had flu-like symptoms for five days and presented at Dr Innis' surgery with coughing, sleep disturbance and lack of appetite but with no fever and was continuing to have wet nappies. By then she had had two previous episodes of croup.[820] She had no allergies and her immunisations were up to date. On examination her chest was clear with no signs of respiratory distress and her throat was red. Dr Innis diagnosed a viral upper respiratory tract infection and advised treatment with Panadol and fluids.[821]

440. On 19 October 1998, Laura presented with a burn on her left forearm and palm.[822] On 19 January 1999, she had a macular red rash on her shoulders, upper arms and down her arms and a red throat. Ms Folbigg told Dr Innis she had had the rash for five days. Laura was then 17 months old. She had no upper respiratory tract symptoms and was behaving normally. Dr Innis diagnosed an allergic rash and prescribed Phenergan to treat it. He advised Ms Folbigg to bring Laura back for review if the rash did not go away over the next few days.[823]

441. Laura presented again on 22 January 1999 for review of the rash. She had had fevers over the preceding few days. Her throat was red. Dr Innis diagnosed a viral rash but no additional treatment was prescribed.[824] On 5 February 1999, Laura attended Dr Innis for her 18 month immunisation. At that stage she was well. On examination her throat and ears were clear.[825]

442. Dr Innis gave evidence before the jury that Laura's death was totally unexpected and there was nothing to suggest that she was other than a well-cared-for child.[826]

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810 15 April 2003 T693.41-49.
811 Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Seton (23 November 1999) p 3; 15 April 2003 T696.38-47.
812 14 April 2003 T657.1-19.
813 Exhibit H, Forensic pathology tender bundle, Statement of Dr John Cash (9 March 1999).
814 14 April 2003 T657.21-32.
815 14 April 2003 T657.49-52.
816 14 April 2003 T657.48-52.
817 14 April 2003 T6660.1-22.
818 15 April 2003 T665.37-39; T668.51-53.
819 15 April 2003 T668.12-669.7.
820 Exhibit H, Forensic pathology tender bundle, Statement of Dr Paul Innis (15 March 1999).
821 Exhibit H, Forensic pathology tender bundle, Statement of Dr Paul Innis (15 March 1999).
822 Exhibit H, Forensic pathology tender bundle, Statement of Dr Paul Innis (15 March 1999).
823 Exhibit H, Forensic pathology tender bundle, Statement of Dr Paul Innis (15 March 1999).
824 Exhibit H, Forensic pathology tender bundle, Statement of Dr Paul Innis (15 March 1999).
825 Exhibit H, Forensic pathology tender bundle, Statement of Dr Paul Innis (15 March 1999).
826 5 April 2003 T668.18-669.7; Exhibit H, Forensic pathology tender bundle, Statement of Dr Paul Innis (15 March 1999).

Laura's death

443. Ambulance officers Mr Brian Wadsworth, Mr Harold Picton and Mr Ted Smith attended the Folbigg home at 12:14pm on 1 March 1999 in response to the callout by Ms Folbigg.[827]

444. Mr Wadsworth went straight up to Laura when he arrived. She was lying on the breakfast bar, and he found she was not breathing and had no pulse.[828] He looked inside her mouth, and did not see any blood, vomit or foreign object.[829] He performed CPR for a period, then Mr Picton took over.[830] Mr Wadsworth applied an ECG monitor at 12:17pm, which showed that Laura was in bradycardia.[831] Mr Wadsworth administered adrenalin, then Laura was taken to hospital, arriving at 12:32pm.[832]

445. Mr Wadsworth stated that when he was treating Laura, he noticed that her skin was warm to touch and cyanosis was present – she had blue colouring around the lips and face.[833] Mr Picton also stated that Laura was warm to touch.[834]

446. The Ambulance Service patient report signed by the three officers who attended recorded nil vital signs, nil pulse, pupils dilated, warm to touch, cyanosis evident.[835] The report records that Laura's airway was obstructed, and also that her skin temperature was "normal" and her buccal mucosa was blue.[836]

Report of death to the Coroner

447. After Laura's death, a death scene investigation checklist was completed, which was a pro forma form to be completed when inquiring into the circumstances of the death of a child two years and younger ("a P534").

827 Exhibit H, Forensic pathology tender bundle, Statement of Brian Wadsworth (15 September 1999) p 1; Exhibit H, Forensic pathology tender bundle, Statement of Harold Picton (15 September 1999) p 1; 15 April 2003 T699.53-700.14.

828 Exhibit H, Forensic pathology tender bundle, Statement of Brian Wadsworth (15 September 1999) p 2; 15 April 2003 T700.16-19, T700.50-54.

829 Exhibit H, Forensic pathology tender bundle, Statement of Brian Wadsworth (15 September 1999) p 2; 15 April 2003 T701.8-17.

830 Exhibit H, Forensic pathology tender bundle, Statement of Brian Wadsworth (15 September 1999) p 2; Exhibit H, Forensic pathology tender bundle, Statement of Harold Picton (15 September 1999) p 2; 15 April 2003 T701.19-21.

831 Exhibit H, Forensic pathology tender bundle, Statement of Brian Wadsworth (15 September 1999) p 2; Exhibit H, Forensic pathology tender bundle, Statement of Harold Picton (15 September 1999). Mr Picton stated that the ECG registered asystole: 15 April 2003 T701.34-36.

832 Exhibit H, Forensic pathology tender bundle, Statement of Brian Wadsworth (15 September 1999) p 2; Exhibit H, Forensic pathology tender bundle, Statement of Harold Picton (15 September 1999).

833 Exhibit H, Forensic pathology tender bundle, Statement of Brian Wadsworth (15 September 1999) p 2; 15 April 2003 T702.36-41.

834 Exhibit H, Forensic pathology tender bundle, Statement of Harold Picton (15 September 1999) p 2.

835 Exhibit H, Forensic pathology tender bundle, Ambulance report V70724 (1 March 1999).

836 Exhibit H, Forensic pathology tender bundle, Ambulance report V70724 (1 March 1999).

Who found the infant? **Natural mother** Time: **12.05pm**

How did the infant come to be found? **Noise** ☑
Specify: **Started coughing and checked 5 minutes later.**

Was any resuscitation attempted? **Parent** ☑
Describe: **CPR by parent and then ambulance**

Where was the infant when found? **Other** ☑
Describe: **Single bed**
Type of mattress: **Innerspring**
Describe:

Were there items covering the infant's head? **No** ☑
List the items:

Was the infant sleeping alone? **Yes** ☑ With whom:

Position of infant when put down: **Side** ☑

Position of infant when found: **On Back** ☑

Were there any recent changes in sleeping pattern? **No** ☑
Describe:

Was the infant found in an unusual sleeping position? **No** ☑
Describe: **Laying on back**
What clothing was the infant wearing at the time? **disposable nappy, tights and shirt**

Figure 15: Extract from Death Scene Investigation Checklist in relation to Laura: Exhibit H in the Inquiry, pp 126-127

448. The completed P534 form recorded that there had been three previous siblings who had died, and that those deaths were attributed to SIDS.[837] It also recorded that Laura had had a cold in the last 24 hours, and had been administered Demazin, 3.5ml, with the last dose on 27 February 1999.[838] She was last fed at 7:00am on 1 March 1999.[839] It also recorded that Laura had been put down on her side when she was put down to sleep, and was on her back when she was found. There was nothing covering her head.[840]

449. The P79A also gave a narrative of the circumstances under which the death had taken place.[841] That narrative included reference to the three previous deaths, and that they had been attributed to SIDS, noting that Professor Hilton had been involved with the investigation of those deaths.[842] The report also noted the administration of Demazin. It recorded that Ms Folbigg had stated that when she went in to check on Laura, having heard her cough about five minutes before, Laura was lying on her back on the bed, her face was extremely pale and she was not breathing.[843]

837 Exhibit H, Forensic pathology tender bundle, SIDS death scene investigation checklist relating to Laura (undated).
838 Exhibit H, Forensic pathology tender bundle, SIDS death scene investigation checklist relating to Laura (undated).
839 Exhibit H, Forensic pathology tender bundle, SIDS death scene investigation checklist relating to Laura (undated).
840 Exhibit H, Forensic pathology tender bundle, SIDS death scene investigation checklist relating to Laura (undated).
841 Exhibit H, Forensic pathology tender bundle, Report of death to the Coroner (1 March 1999).
842 Exhibit H, Forensic pathology tender bundle, Report of death to the Coroner (1 March 1999).
843 Exhibit H, Forensic pathology tender bundle, Report of death to the Coroner (1 March 1999).

Autopsy reports

450. Dr Cala carried out the post-mortem examination at 9:00pm on 1 March 1999.[844] Professor Hilton, as the Director of the Institute of Forensic Medicine, was involved with Dr Cala's autopsy of Laura and was present when Dr Cala conducted the autopsy.

451. At the time he conducted the autopsy of Laura, Dr Cala was aware of the death of her three siblings.[845] He was told that she had been diagnosed with central apnoea but that there was no evidence of obstructive apnoea and that she had been monitored with no significant sleep abnormalities detected, and that she had recently been unwell with cold and flu-type symptoms.[846]

Interim autopsy report

452. An interim autopsy report was completed by Dr Cala, dated 1 March 1999.[847] In that interim report, Dr Cala recorded the direct interim cause of death as "undetermined".[848] He noted that significant injuries were not present, and specimens were retained including samples of tissue and Laura's brain.[849]

Final autopsy report

453. The final autopsy report contained further detail, however, Dr Cala maintained his finding that the cause of death was "undetermined".[850] Dr Cala recorded that the time and date of death was sometime between approximately 11:00am and 12:45pm on 1 March 1999.[851]

454. Dr Cala recorded a narrative of the previous children's births, medical histories, and deaths including the ascribed causes of deaths.[852] Inter alia, he noted that Mr Folbigg had allegedly been diagnosed with Obstructive Sleep Apnoea.[853] In relation to Sarah, Dr Cala referred to letters from Dr Seton, including that Sarah had apparently been well after her birth, although she "was a very loud snorer who suffered witnessed apnoea and choking episodes during sleep. Sadly, none of this was investigated prior to her death".[854] Also, that:

> *Sarah had a very long palate and uvula. It is well known clinically that soft palates and uvulas become swollen and elongated as a secondary effect of habitual snoring.*[855]

Overview of examination

455. Dr Cala set out a narrative of his findings upon post-mortem examination in the report summary and opinion.[856]

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844 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) p 1.
845 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) pp 2-3; 15 April 2003 T706.37-40.
846 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) p 3; 15 April 2003 T707.27-41.
847 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Laura (1 March 1999).
848 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Laura (1 March 1999) p 1.
849 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Laura (1 March 1999) p 1.
850 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) p 2.
851 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) p 2.
852 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) pp 2-3.
853 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) p 3.
854 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) p 3.
855 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) p 3.
856 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) p 4.

456. In the examination he noted lividity mainly on the left side of the face. There was also dorsal lividity present. There were no significant injuries externally apart from minor bruises to the lower limbs. There were no injuries to the face or in the oral cavity. There were no petechial haemorrhages on the face or on the eyelids, and re-examination the next day also failed to show petechial haemorrhages. The neck examination was normal. Internally, there were no significant abnormalities apart from focally haemorrhagic and collapsed lungs.

457. Histological examination of tissues showed an inflammatory infiltrate in the heart, consistent with myocarditis, of probable viral origin. This accorded, Dr Cala opined, with the history of a cold/flu-like illness for several days prior to Laura's death. He stated that there were a variety of causes for myocarditis, including some viruses, bacteria, fungi, some immune-related disorders, some drugs, and several other causative agents.

458. Dr Cala also stated in the report that toxicological examination of tissues and fluids showed no drugs, alcohol or poisons. The absence of Demazin suggested that it had not been given for some time prior to Laura's death, possibly 12-24 hours or longer.

459. Dr Cala stated:

> *The death of Laura Folbigg cannot be regarded as "another SIDS"… The family history of no living children following four live births is highly unusual. Laura had metabolic blood and urine tests as an infant, as part of screening for possible inherited metabolic diseases. These tests were all normal, and this would exclude a metabolic abnormality as a cause of sudden death in Laura. Obstructive Sleep Apnoea has also been excluded as a cause of death for Laura, as there was no evidence to substantiate this diagnosis despite intensive monitoring by doctors at New Children's Hospital, Westmead.*
>
> *The diagnosis of SIDS should be made very sparingly after the age of 12 months. This diagnosis should only be made after a death scene investigation, post mortem examination, and various toxicological and microbiological cultures have failed to establish any other reasonable cause of death. Although there was an inflammatory infiltrate in the heart consistent with myocarditis, this may represent an incidental finding.*
>
> *The possibility of multiple homicides in this family has not been excluded. If homicidal acts have been committed it is most likely these acts have been in the form of deliberate smothering. Smothering, whether deliberately or accidentally inflicted, may leave no trace. There are no specific post mortem findings for smothering. It is usually performed by one person, in the absence of any witnesses. It is relatively easy for an adult to smother an infant or small child with a hand, pillow, soft toy or other similar object.*
>
> *The bodies of all the Folbigg children have been cremated.*[857]

460. A range of specimens were retained for further examination, including tissue for histology, and Laura's brain.[858]

[857] Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) p 4.

[858] Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) p 5.

External examination

461. On the external examination, there were no petechial haemorrhages on the upper or lower lids.

462. An ovoid 5x3mm brown bruise was present just medial to the left patella. An ovoid 12x10mm brown bruise was noted on the right anterior lower leg.[859]

Key findings on internal examination

463. No retinal haemorrhages were present.

464. There was no evidence of congenital heart disease.

465. The airways were normal. The left lung weighed 122 grams and the right lung 114 grams. Both were focally haemorrhagic and collapsed on section. The thymus weighed 28 grams and was normal apart from petechial haemorrhages on the anterior aspect of the suprasternal thymus gland. This part of the thymus measured approximately 15x10x10mm and projected superiorly beyond the suprasternal notch.

466. The entire body was x-rayed on 2 March 1999 – no fractures were detected. The body was also re-examined that day, with no additional significant findings.

467. Dr Cala found no injuries to Laura's face or mouth, and no petechial haemorrhages on the face or eyelids.[860] A facial dissection was carried out on 3 March 1999. Several photographs were taken. No bruises or any other injuries were detected.[861]

Microscopic examination

468. Within the myocardium was a moderately dense infiltrate of lymphocytes which had aggregated in certain areas, particularly subendocardially and along the superficial surface of the myocardium, although further sections showed large aggregates in the central area of the left ventricle. In these areas, there were large clusters of lymphocytes surrounding degenerate myocytes. Myocytolysis (damage to cardiac muscle cells) was present. No viral inclusions were seen. The appearances were of myocarditis, which was probably viral in aetiology.

469. Further blocks of heart tissue taken (2nd and 3rd cuts) confirmed the presence of aggregates of lymphocytes in a similar distribution to those in the first histological examination of the heart.

470. The spleen showed many germinal centres within white pulp areas, and a markedly increased number of lymphocytes in the red pulp. The appearances were of a probable viral infection. There was no evidence of malignancy nor any histological features to suggest any specific underlying viral infection.

471. The lungs showed an increased number of lymphocytes within the interstitium and in some alveolar spaces. There were widespread areas of haemorrhage with numerous red blood cells within alveolar spaces which also contained oedema fluid, foamy macrophages, and fibrin.

472. Focal cortical haemorrhages were present in the thymus.

473. Microscope slides of the liver, kidney, stomach, oesophagus, adrenal, salivary gland, small and large intestine, thyroid, bone-marrow, pancreas, diaphragm, skeletal muscle, and ovary were all essentially normal.[862]

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859 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) p 7.

860 15 April 2003 T708.46-52.

861 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) pp 8-9.

862 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) p 10.

Neuropathology report

474. No macroscopic abnormality was seen in the brain or in a 20cm segment of spinal cord examined.[863]

475. No microscopic abnormality was seen in the multiple sections taken of the brain.[864] Development was appropriate for age.[865]

Histology report

476. The histology report noted the following findings:

> *Source: Lung*
>
> *Profuse Post mortem contaminants.*
>
> *Profuse coliform.*[866]
>
> *Source: Spleen.*
>
> *Moderate coliforms of 2 colonial types.*
>
> *Profuse alpha haemolytic Streptococcus of 2 colonial types.*
>
> *Moderate Staphylococcus aureus.*[867]

Evidence at the time of trial

SIDS

477. At trial, consistently with his statement, Dr Seton gave evidence that Laura did not fit the profile of a high risk SIDS patient. Obstructive sleep apnoea was excluded as a risk, as were other inheritable and non-inheritable disorders – the testing of inheritable disorders known at the time to cause SIDS was exhaustive and included breathing and non-breathing disorders, with the latter including MCAD (an enzyme deficiency in the liver) and metabolic disorders.[868] He agreed that research continued around the world on the question of inherited disorders and on SIDS.[869] Dr Seton said that SIDS was highly unusual at Laura's age, but does happen, although Laura's risk of SIDS was extremely low because of the testing, monitoring, and her age when she died.[870]

478. Dr Cala gave evidence before the jury that he did not believe Laura was a SIDS case because she was 20 months old at the time of her death.[871] In addition, children who have died from SIDS are usually found the following morning after they have been fed, perhaps in the middle of the night.[872] He said that following that, a scene examination should be done, an autopsy should be held, and other testing of tissues and fluids should be carried out – if at the end of that, the clinical history is appropriate, the testing is negative and trauma is excluded, then if the child is within three to six months (but certainly no more than 12 months), then it might be SIDS.[873]

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863 Exhibit H, Forensic pathology tender bundle, Neuropathology report of Laura (13 December 1999) p 2.
864 Exhibit H, Forensic pathology tender bundle, Neuropathology report of Laura (13 December 1999) p 2.
865 Exhibit H, Forensic pathology tender bundle, Neuropathology report of Laura (13 December 1999) p 2.
866 Exhibit H, Forensic pathology tender bundle, Microbiology report of Laura (9 March 1999).
867 Exhibit H, Forensic pathology tender bundle, Microbiology report of Laura (9 March 1999).
868 15 April 2003 T695.4-16, T697.51-698.3; Statement of Dr Christopher Seton (23 November 1999) p 3.
869 15 April 2003 T698.5-16.
870 15 April 2003 T695.20-28, T698.27-32.
871 15 April 2003 T721.50-722.8.
872 15 April 2003 T722.10-36.
873 15 April 2003 T722.37-49.

479. At trial, the defence did not contend that Laura's death was a SIDS death.[874] Professor Busuttil opined that Laura's death should not be classified as SIDS.[875]

Opinions on cause of Laura's death

480. As noted above, in his examination of Laura's heart Dr Cala found nothing abnormal to the naked eye, but under microscopic examination he found inflammatory infiltrate.[876] The inflammation was moderate, but he did not believe it played any role in causing Laura's death.[877]

481. When pressed, Dr Cala said he did not exclude the possibility that the cause of Laura's death was myocarditis, but he maintained that he did not believe it to be a reasonable possibility. He acknowledged that myocarditis can cause sudden death but thought this was very unlikely with Laura.[878]

482. In his evidence, Dr Cala described the myocarditis as "quite patchy" and "rather mild", that the amount of inflammation was not particularly heavy, there was not any evidence of heart failure, the heart looked normal to the naked eye, and there was evidence of inflammation in other organs which indicated viral infection around the time of her death.[879] He attached no significance to inflammation of the subendocardial infiltrate, although he considered that clusters of lymphocytes surrounding degenerate myocites was a significant finding.[880]

483. However, Laura's heart did not exhibit things that Dr Cala said may be found in a death caused by myocarditis, such as flabbiness, a stripy appearance of the left ventricle in particular, or dilation of the chamber; nor did Dr Cala find fluid around the lungs or in the abdomen.[881] He noted that persons who have died from totally unrelated causes (such as car accidents) have been found to have this mild inflammatory infiltrate of the heart.[882]

484. On 29 June 1999, Dr Cala wrote to Detective Senior Constable Ryan in relation to all four children and said that further investigation was warranted.[883] In that letter, he noted that Laura did not die of SIDS, as she was too old for this diagnosis, but "had an intercurrent illness which *might* have explained her death."[884]

485. Following queries raised by police about whether myocarditis may have been an incidental finding, on 19 June 2001 Dr Cala responded, describing the inflammatory infiltrate in the sections of the heart that he examined as "light in amount and patchy in distribution."[885] He stated that if he had examined Laura's body in isolation, he might give the cause of death as myocarditis, but while the other deaths did not "need bias or prejudice my opinion", he could not ignore any known relevant family history.[886] He cited evidence in medical literature that this amount of inflammation "could be considered of no relevance in the deaths of some children" who had died by other means (choking, or accident), and then stated:

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874 15 April 2003 T723.1-7.

875 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 14.

876 15 April 2003 T713.45-58, T714.22-29; Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) p 10.

877 16 April 2003 T761.7-32.

878 15 April 2003 T714.11-37, T719.28-37; 16 April 2003 T761.17-20, T761.55-57.

879 15 April 2003 T714.22-29; 16 April 2003 T761.22-32, T763.19-764.12.

880 16 April 2003 T760.16-55.

881 15 April 2003 T714.39-715.4; 16 April 2003 T756.40-757.23.

882 15 April 2003 T715.6-12.

883 Exhibit H, Forensic pathology tender bundle, Letter from Dr Allan Cala to Detective Bernard Ryan (29 June 1999) p 3.

884 Exhibit H, Forensic pathology tender bundle, Letter from Dr Allan Cala to Detective Bernard Ryan (29 June 1999) p 2 (emphasis in original).

885 Exhibit H, Forensic pathology tender bundle, Letter from Dr Allan Cala to Detective Bernard Ryan (19 June 2001) p 2.

886 Exhibit H, Forensic pathology tender bundle, Letter from Dr Allan Cala to Detective Bernard Ryan (19 June 2001) p 2.

> *My opinion that the inflammatory infiltrate in the heart represents an incidental finding is not based on the family history, but rather after consideration of the history provided of Laura's very sudden and most unexpected death, the post mortem findings… and the histological assessment of the heart together with my own knowledge and experience of myocarditis.*[887]

486. In trial evidence, he maintained his finding of "undetermined".[888] So ascribed, it included natural causes unable to be found, as well as inflicted and accidental causes – quite different from SIDS, which is considered to be death due to natural causes.[889] Dr Cala opined that Laura "probably" died from an acute catastrophic asphyxiating event of unknown causes.[890] He agreed that it was consistent with smothering, including deliberate smothering.[891]

487. Dr Cala confirmed in his evidence at trial that the internal examination revealed no significant abnormalities apart from focal haemorrhaging (on the lungs), and collapsed lungs.[892] Collapse of the lungs may have been due to asphyxiation, or may have been resuscitation related, so it was "quite non-specific".[893] Similarly, while petechial haemorrhaging on eyelids and around the eyes is suggestive of smothering, its absence was non-specific and could not be used to differentiate SIDS over smothering.[894] Both the haemorrhaging and collapsed lungs were consistent with asphyxiation, "but there are other causes".[895]

488. Asked if there were any signs a pathologist might find on a post-mortem that would enable a pathologist to distinguish between SIDS and deliberate smothering, Dr Cala said it was very difficult to differentiate between the two; with SIDS, tiny haemorrhages on the surface of the heart and lungs and thymus (a gland in the top of the chest), while not diagnostic of SIDS (being a non-specific finding), were found with many children who died of SIDS.[896] Children who have been smothered may either have petechial haemorrhages on the heart, lungs and thymus, or they may not; smothering may well not leave some sign.[897] Similarly, the haemorrhages were not diagnostic of SIDS.[898] There had been reports of children who had petechial haemorrhages on eyelids and around the eyes as suggestive of smothering, but their absence did not exclude the possibility.[899]

489. Asked at trial whether he did not subscribe to a finding of non-accidental suffocation due to other reasonably possible causes of death (and if so, what they were), Dr Cala stated that non-accidental asphyxia "must be considered as a possible cause of death for Laura Folbigg, and… for the other Folbigg children."[900] The medical evidence did not allow him to take this higher than a suspicion.[901]

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887 Exhibit H, Forensic pathology tender bundle, Letter from Dr Allan Cala to Detective Bernard Ryan (19 June 2001) p 2.

888 15 April 2003 T719.43-721.13; 16 April 2003 T749.9, T762.3-5; Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) p 2. See also Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Laura (1 March 1999).

889 15 April 2003 T721.15-20, T721.40-48.

890 16 April 2003 T749.6-19.

891 16 April 2003 T749.11-19.

892 15 April 2003 T708.57-709.6; Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999) pp 4, 11.

893 15 April 2003 T709.34-38.

894 15 April 2003 T709.55-710.4, T710.27.

895 15 April 2003 T709.53.

896 15 April 2003 T709.55-710.18.

897 15 April 2003 T710.20-36.

898 15 April 2003 T710.6-22.

899 15 April 2003 T710.32-36.

900 Exhibit H, Forensic pathology tender bundle, Letter from Dr Allan Cala to Detective Bernard Ryan (19 June 2001) p 3.

901 Exhibit H, Forensic pathology tender bundle, Letter from Dr Allan Cala to Detective Bernard Ryan (19 June 2001) p 3.

490. Later, in a statement given on 28 March 2003, after watching a video of Laura taken on 28 February 1999, Dr Cala opined that she appeared in good health and that he believed more firmly that the myocarditis which was found at autopsy "played no role whatsoever in her death, and was an incidental finding."[902]

491. Professor Hilton said that at the time of her death, Laura was suffering from myocarditis, or the physical manifestations of it.[903] Myocarditis can lead to death, and it was of an intensity and severity that it could have caused Laura's death.[904] Professor Hilton described myocarditis as a serious condition which can be significant in extreme situations, such as:

> *If I saw it in someone who had run off the road and killed themselves in the car I would not regard it as an incidental finding, I would have to regard it as an important finding and in someone who unexpectedly was found dead I would regard it as a highly significant finding, although it may be incidental to something else.*[905]

492. Myocarditis was the only pathological lesion that was present that could account for Laura's death.[906] It usually has observable symptoms prior to death, such as shortness of breath and heart failure, but it could be entirely silent.[907] Professor Hilton said that there was no other indication – on autopsy, or the subsequent investigation – of what could have caused her death.[908] The video of Laura showing her to be apparently symptom-free, 24 hours before her death, did not preclude myocarditis as a cause.[909] He would not necessarily expect a person to feel very unwell with myocarditis and had seen well people drop dead from it.[910] Myocarditis could be caused by the common cold, as any viral illness can produce it.[911]

493. However, Professor Hilton opined that while the finding of myocarditis was highly significant, it did not exclude deliberate suffocation as a possible cause of Laura's death.[912]

494. Dr Bailey was a consultant cardiologist who gave evidence both in relation to cardiac rhythm tracing records for Laura from when ambulance officers attended the call out on 1 March 1999 and afterwards, and also in relation to myocarditis.[913]

495. Dr Bailey gave evidence before the jury that Laura's myocarditis was probably related to a viral infection.[914] He said that clinical myocarditis is rare whereas pathological myocarditis is common.[915] He considered that the myocarditis was unlikely to have accounted for her death because myocarditis can arise from illnesses (such as the flu) or situations (such as certain cardiac conditions) which do not lead to sudden death.[916] Sudden death from myocarditis is rare; however, there may be no clinical manifestation.[917] In mild or moderate stages, it is

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902 Exhibit H, Forensic pathology tender bundle, Statement of Dr Allan Cala (28 March 2003) pp 1-2.
903 24 April 2003 T907.34-36.
904 24 April 2003 T907.43-45.
905 24 April 2003 T907.53-908.2.
906 24 April 2003 T908.9-13.
907 24 April 2003 T908.15-27.
908 24 April 2003 T908.4-7.
909 24 April 2003 T908.41.
910 24 April 2003 T913.5-9.
911 24 April 2003 T912.39-41.
912 24 April 2003 T913.11-15.
913 5 May 2003 T1098.39-1099.5.
914 5 May 2003 T1100.44-46.
915 5 May 2003 T1100.20-22, T1105.36-1106.23.
916 5 May 2003 T1100.50-1101.34, relying on Exhibit H, Forensic pathology tender bundle, Final autopsy report of Laura (13 December 1999). See also T1101.53-1102.47.
917 5 May 2003 T1103.35-38, T1104.56-1105.1.

asymptomatic and is not unusual in perhaps four per cent or five per cent of postmortem examinations.[918] The possibility of myocarditis in Laura's conductive system (which could be fatal) could not be ruled out – if so, it would have had to have been in a very tiny area of the heart where the conductive system is small or affected a very large part of the conductive system.[919]

496. Dr Bailey gave evidence that the heart rhythm tracing showed an "agonal cardiac rhythm", that is, that the heart was no longer pumping any significant amount of blood but was retaining a tiny amount of very slow electrical activity.[920] This was consistent with, but not specific for, injury due to lack of oxygen.[921] Dr Bailey considered it was most likely that breathing stopped before the heart stopped, but he was not certain because the tracing was recorded a considerable time after the event.[922]

497. Taking the whole situation into account, Dr Bailey gave evidence that it was a lot more probable that the cause was not arrhythmia caused by myocarditis – while he could not be definitive, it was more consistent with cessation of breathing first because there was still cardiac electrical activity going on long after breathing stopped.[923]

498. Dr Beal would have thought the cause of Laura's death was undetermined, based on the history.[924] This "absolutely" included deliberate smothering. While she said she would bow to pathologists on the significance of the myocarditis findings, the findings were consistent with Laura having suffered a sudden acute catastrophic asphyxiating event, and with deliberate smothering.[925] While she acknowledged that "in medicine nothing is a hundred percent" she was of the view that if there was something wrong with the heart, then the heart would stop first.[926]

499. In oral evidence, Professor Herdson could not say what was the significance of the myocarditis in determining the cause of Laura's death; it appeared diffuse and relatively mild.[927] He preferred a view that the myocarditis was an incidental finding, because it was definitely viral myocarditis but not a "roaring one" and was fairly diffuse, although he would have been more confident in this view if necrosis was not present.[928]

500. In his report, Professor Herdson concurred with Dr Cala's finding of "undetermined" as the cause of death, and with the analyses by Professor Berry and Dr Ophoven.[929] In his recorded answers to the model questions, Professor Herdson said that by itself, he would probably have said SIDS until he saw the histology – but he would have been concerned that Laura was 19 months old. Therefore, he would have said undetermined. Deliberate smothering could not be excluded. "Of course", he stated, Laura died of an acute catastrophic asphyxiating event. Professor Herdson agreed with Professor Berry's histopathology and toxicologic analysis (referred to below).[930]

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[918] 5 May 2003 T1101.27-34.
[919] 5 May 2003 T1103.24-52.
[920] 5 May 2003 T1099.32-57.
[921] 5 May 2003 T1100.1-4.
[922] 5 May 2003 T1100.6-11, T1104.3-18, T1106.55-1107.12.
[923] 5 May 2003 T1106.53-1107.12.
[924] Responses to Crown model questions – Dr Beal, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.
[925] 5 May 2003 T1142.39-44, T1143.31-38, T1149.5-9.
[926] 5 May 2003 T1150.9-19.
[927] 1 May 2003 T1039.21-25, T1039.53-56, T1046.7-10.
[928] 1 May 2003 T1039.46-1040.8.
[929] Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Herdson (17 January 2002) p 3.
[930] Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Herdson (17 January 2002) p 3.

501. In evidence, Professor Berry said that Laura's myocarditis was consistent with the relatively mild viral infection she had before her death.[931] The myocarditis was moderate but quite significant, however, significance is not related to quantity – a small amount in the conductive system could be fatal, whereas quite diffused myocarditis may not lead to death.[932] In his first report, Professor Berry stated that the infiltrate in Laura's heart was quite extensive and most pathologists would have accepted it as the cause of death. In isolation he would have ascribed her death to myocarditis, although he was "unable to convince [himself] of actual damage to heart muscle cells."[933]

502. Professor Berry observed that it is recognised that an inflammatory infiltrate in the heart muscle is quite commonly found in those who die of other causes, must therefore be quite common in the general population and probably accompanies some common childhood illnesses – it does not necessarily mean it was responsible for death.[934] In oral evidence before the jury, Professor Berry said that in isolation the myocarditis presented a possible explanation for Laura's death.[935]

503. Professor Berry also gave evidence at trial that myocarditis might facilitate death by suffocation.[936] Qualifying his evidence by noting that there was no literature in this area, he thought it was highly possible – indeed probable – that subjecting a child with myocarditis to severe stress (such as an asphyxial episode) might precipitate an abnormal beat of the heart leading to sudden death.[937] He described "classical scenarios" of people with myocarditis from a mild viral infection who are stressed and die suddenly, including young athletes, people swimming and young servicemen doing strenuous activity. Professor Berry believed that myocarditis may have been "the straw that breaks the camel's back", pushing the heart into abnormal rhythm and causing death.[938] He concluded she "probably" died from an acute catastrophic asphyxiating event of unknown causes (but in isolation, myocarditis).[939]

504. Professor Byard agreed with Dr Cala and Professor Berry that the slides of Laura's heart demonstrated myocarditis.[940] He described myocarditis as "a well-known cause of sudden and unexpected death in children of all ages and may be found in infants who present in a similar manner to SIDS."[941] It is most commonly caused by viruses; Professor Byard could not find evidence of confirmatory viral studies at the time of Laura's autopsy.[942]

505. In his second report dated 14 April 2003, Professor Byard commented upon seven histological slides showing eight pieces of heart muscle, provided to him by Ms Folbigg's legal representatives.[943] In each of the pieces there was an inflammatory cell infiltrate with and without degeneration of heart muscle cells.[944] He stated that this "indicates established myocarditis".[945]

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931 1 May 2003 T1065.1-3.
932 1 May 2003 T1074.40-1075.3, T1075.54-1076.22, T1083.34-1084.17.
933 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 25.
934 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 25.
935 1 May 2003 T1065.1-9.
936 1 May 2003 T1065.15-40.
937 1 May 2003 T1065.29-33.
938 1 May 2003 T1065.15-27.
939 Responses to Crown model questions – Professor Berry, trial Exhibit C on voir dire: Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure C.
940 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 5.
941 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 5.
942 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 5.
943 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (14 April 2003) p 1.
944 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (14 April 2003) p 2.
945 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (14 April 2003) p 2.

He supplemented his explanation of myocarditis given in his first report, stating that in myocarditis the heart is infiltrated by inflammatory cells resulting in the death of these cells, "as we see with Laura".[946] He concluded that "completely normal behaviour on the day before Laura died with myocarditis does not in any way exclude myocarditis as a possible cause of death."[947] The clinical signs and symptoms are very variable; an affected child may have had no indication of any illness, or only very mild symptoms resembling a cold.[948] He had personally had several cases of infants and young children who had died from myocarditis with minimal or no symptoms.[949]

506. Professor Byard had also conducted a review of 16 children who died of myocarditis at the Adelaide Children's Hospital over approximately 35 years from 1951-1990. Sudden death occurred in five of the 16, three with no prodromal symptoms (early symptoms which precede clinical manifestations).[950] A number of similar reports could be found in the literature.[951] Myocarditis may also be completely incidental to the cause of death and Professor Byard had had several cases where this had happened.[952]

507. Professor Byard stated in his first report and in evidence, that because of the circumstances of the previous deaths, he would list Laura's death as "undetermined" (cannot exclude myocarditis).[953] The finding of an agonal rhythm is non-specific and didn't help Professor Byard say anything about how a patient died.[954] He noted sometimes babies get an agonal rhythm up to half an hour after their respirators have stopped. The lack of malfunction of the heart did not exclude death as a result of myocarditis.[955] Professor Byard agreed, under crossexamination by the Crown prosecutor, that myocarditis could have been incidental to Laura's death, however, his agreement was reluctant in the absence of microbiological and DNA hybridisation studies.[956] He said there was no finding or symptom that could amount to proof of suffocation.[957] Professor Byard also noted that there was a history of a recent upper respiratory tract infection.[958]

508. Professor Busuttil opined that myocarditis could have caused serious heart problems and even death "acutely and unexpectedly", although it may also have been completely incidental and could not have been induced by imposed airways obstruction.[959] He said that it was most likely caused by a virus which may have set off an abnormal fast rhythm of the heart and led to sudden death. Because no viral studies were conducted at the time of autopsy, he said the origins of the myocarditis were uncertain.[960] Professor Busuttil also noted the history of a recent upper respiratory tract infection.[961]

946 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (14 April 2003) p 2.
947 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (14 April 2003) p 3.
948 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (14 April 2003) p 2.
949 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (14 April 2003) p 2.
950 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (14 April 2003) p 2. See also 7 May 2003 T1245.47-1248.21.
951 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (14 April 2003) pp 2-3.
952 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (14 April 2003) p 3.
953 7 May 2003 T1220.48-54; Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 5.
954 7 May 2003 T1221.4-24.
955 7 May 2003 T1257.31-45.
956 7 May 2003 T1243.1-1245.28; see also 7 May 2003 T1257.47-1258.12.
957 7 May 2003 T1221.56-1222.1.
958 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 6.
959 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 14.
960 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) pp 13-14.
961 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 13.

509. Dr Owen Jones, consultant paediatric cardiologist, stated in his report that there was good evidence that Laura had myocarditis at the time of her death – the histopathological findings of all the experts who examined the heart sections were conclusive.[962] It was "a well-recognised cause of unexpected sudden death in children of all ages."[963] Alternatively, there may be presentation features ranging in severity from mild to severe, and individuals in whom the histopathological features are incidental and who neither die from it nor exhibit features of cardiac failure.[964]

510. Dr Jones stated that the "crux of the matters is whether myocarditis was causal, contributory, or incidental to the death of Laura."[965] He gave evidence on this issue at trial. In his report, he stated that to his knowledge there was no evidence that there is a threshold of severity of histopathological features below which appropriate attributed sudden unexpected death cannot occur, so the absence of clinical features of heart failure could not be used to argue that myocarditis had not caused sudden death.[966] In isolation, he would have "no difficulty" in attributing Laura's death to myocarditis.[967]

511. In his evidence, Dr Jones said that it was possible that myocarditis represented an incidental finding but also, without commenting on the probability, believed it could have accounted for Laura's death.[968] He referred to a 2001 study in which 13 patients (two of whom were children) died of myocarditis without abnormal findings on the heart to the naked eye.[969] Under cross-examination he agreed that mild myocarditis almost never leads to death and even with moderate myocarditis there are very few instances of sudden death.[970]

512. Dr Jones described an agonal rhythm as residual electrical activity that can be seen in a patient who is essentially deceased and can continue for many minutes after clinical death. He did not agree with Dr Bailey that the sequence of breathing and cardiac arrest leading to death could be determined from the ECG.[971]

513. In her first report, Dr Ophoven opined, again "to a reasonable degree of medical certainty", that Laura did not die of SIDS, being outside the age range. She considered that Laura's death was most consistent with death by suffocation, and that Laura was the victim of "probable homicide".[972] Her reasons were again similar to those she gave in relation to Patrick.[973]

514. Dr Hawker examined sleep study records relating to Laura.[974] The electrocardiographs showed no abnormalities, but they were absent fine time lines so the QT interval could not accurately be measured.[975]

962 Exhibit H, Forensic pathology tender bundle, Report of Dr Owen Jones (15 April 2003) p 7.
963 Exhibit H, Forensic pathology tender bundle, Report of Dr Owen Jones (15 April 2003) p 7.
964 Exhibit H, Forensic pathology tender bundle, Report of Dr Owen Jones (15 April 2003) pp 7-8.
965 Exhibit H, Forensic pathology tender bundle, Report of Dr Owen Jones (15 April 2003) p 8.
966 Exhibit H, Forensic pathology tender bundle, Report of Dr Owen Jones (15 April 2003) p 8.
967 Exhibit H, Forensic pathology tender bundle, Report of Dr Owen Jones (15 April 2003) p 8.
968 8 May 2003 T1263.33-47, T1271.4-56, T1275.8-10, T1278.55-1279.2.
969 8 May 2003 T1263.50-1264.57, T1269.31-52.
970 8 May 2003 T1269.7-17.
971 8 May 2003 T1261.30-1262.2, T1277.24-50.
972 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 12.
973 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (1 December 2001) p 12.
974 Exhibit H, Forensic pathology tender bundle, Statement of Dr Richard Hawker (6 March 2003) p 2.
975 Exhibit H, Forensic pathology tender bundle, Statement of Dr Richard Hawker (6 March 2003) pp 2-3.

Evidence in the Inquiry

SIDS risk factors

515. In her report, Professor Horne referred to the following "potential protective factors for SIDS" in respect of Laura: Laura was born at term at a normal weight, was found supine with her face uncovered and in her own bed, was vaccinated and used a dummy. In respect of Laura's older age, she noted that sudden unexplained death in childhood is much less common than SIDS. She also referred to an increased risk due to paternal smoking.[976]

516. While Laura was well outside the usual age range for SIDS, Professor Elder gave evidence in the Inquiry that again the common risk factors for sudden infant death were not present and that a history of recent infection has been seen in children who have died suddenly and unexpectedly.[977] Professor Horne observed sudden unexpected death in childhood is "more rare" than SIDS and is "certainly very uncommon."[978]

Myocarditis and opinions on cause of death

Professor Cordner

517. In his report Professor Cordner discussed an investigation into deaths from myocarditis in children under two years of age in New South Wales and Victoria since 2000, identified on the National Coronial Information System ("NCIS"), which showed a total of 39 cases.[979]

518. Twelve cases could not be used due to insufficient information or infection found elsewhere (so were not isolated myocarditis).[980] Of the remaining 27 cases, in two, there were no known circumstances, one had an incomplete history. Thirteen had evidence of a preceding illness (e.g. URTI, lethargy, poor oral intake). Two had a second registered cause of death (atrial septal defect and encephalitis). Two cases involved macroscopic descriptions of the heart (e.g. dilated, enlarged, heavy). Three were co-sleeping with their parent/s.[981]

519. Professor Cordner also referred to Weber et al (2008), in which the authors identified proven myocarditis diagnosed in 28 cases of 1,516 paediatric autopsies over a 10 year period (1.8 per cent), within an age range of 10 days to 16 years, median 10 months.

520. Of those 28 children with proven myocarditis in 11 (39 per cent) there was no macroscopic evidence of abnormality in the heart.[982] Sixteen presented as sudden death (57 per cent), five of whom had no apparent prodromal symptoms.[983] The symptoms in 12 were varying degrees of dyspnoea (laboured breathing) and/or tachypnoea and three with diarrhoea and vomiting, one with pyrexia (fever) and another with non-specific viral symptoms.[984]

521. More than half of the 28 children (54 per cent) were infants less than one year of age, five (18 per cent) were aged one to four years and the remainder aged five years and over.

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976 Exhibit J, Expert report of Professor Rosemary Horne (10 February 2019) pp 2-3.
977 Transcript of the Inquiry, 18 March 2019 T40.28-35.
978 Transcript of the Inquiry, 18 March 2019 T40.40-43.
979 Exhibit Q, Report of Professor Stephen Cordner (undated) p 68.
980 Exhibit Q, Report of Professor Stephen Cordner (undated) pp 68-69.
981 Exhibit Q, Report of Professor Stephen Cordner (undated) p 69.
982 Exhibit Q, Report of Professor Stephen Cordner (undated) p 78.
983 Exhibit Q, Report of Professor Stephen Cordner (undated) p 79; M A Weber et al, 'Clinicopathological Features of Paediatric Deaths Due to Myocarditis: An Autopsy Series' (2008) 93 *Archives of Disease in Childhood* 594, 594-595.
984 M A Weber et al, 'Clinicopathological Features of Paediatric Deaths Due to Myocarditis: An Autopsy Series' (2008) 93 *Archives of Disease in Childhood* 594,595.

522. Fatal myocarditis was "relatively more common" in older children, accounting for around five per cent of all childhood deaths over the age of five years.[985] The authors concluded that "[m]yocarditis is a rare cause of death in infancy and childhood, and the majority of cases present as sudden unexpected deaths", and routine histological sampling of the heart is required for detection.[986]

523. Professor Cordner said he did not refer in his report to Laura having fallen within the 18 per cent of the 28 cases where the children were aged one to four years because it was not relevant to "what use I was trying to make of the data".[987] However, he agreed with the Judicial Officer in his oral evidence in relation to the paper's findings that myocarditis is less common children aged one to four years than it is for older children.[988]

524. Professor Cordner then referred specifically to Dr Cala's evidence about myocarditis at trial and drew from it a number of elements that he addressed individually.

525. The first was that in Laura's case the myocarditis was patchy and mild compared to other cases where the inflammation was more marked.[989] Professor Cordner stated as to this that he did not think the myocarditis was patchy and mild, he thought it better described as widespread and at least moderate in degree, and went on to test the difference, including canvassing opinions of colleagues at the Victorian Institute of Forensic Medicine ("VIFM") after sending them photomicrographs of images of the slides taken of Laura's heart at autopsy.

526. Secondly, in relation to whether one would expect that there be macroscopic signs on autopsy if death was due to myocarditis, Professor Cordner said that lack of macroscopic evidence of the kind described by Dr Cala (for example, flabbiness, a striped appearance) as an indicator against the death being caused by myocarditis, is countered by literature that there may be no macroscopic abnormality of the heart yet the myocarditis might be fatal.[990]

527. Professor Cordner noted that in 13 of the 27 cases identified on the NCIS, the heart was regarded as having a normal naked eye appearance.[991]

528. Thirdly, in relation to whether Laura had preceding symptoms, Professor Cordner noted that Laura did have a runny nose in the couple of days prior to her death.[992] He also noted that of the 27 cases in the NCIS review, 15 had symptoms referable to a viral illness.

529. Fourthly, regarding myocarditis causing sudden and unexpected death only in a small percentage of cases, Professor Cordner noted that in the NCIS investigation, 13 of the 27 died in hospital, so were not sudden or unexpected.[993] Twelve arrived at hospital deceased.[994] There was no information for two. He stated that on this basis, it would appear that sudden and unexpected death is not all that unusual in this population of infants and toddlers dying from myocarditis.[995]

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985 M A Weber et al, 'Clinicopathological Features of Paediatric Deaths Due to Myocarditis: An Autopsy Series' (2008) 93 *Archives of Disease in Childhood* 594, 596.

986 M A Weber et al, 'Clinicopathological Features of Paediatric Deaths Due to Myocarditis: An Autopsy Series' (2008) 93 *Archives of Disease in Childhood* 594, 594.

987 Transcript of the Inquiry, 21 March 2019 T291.20.

988 Transcript of the Inquiry, 21 March 2019 T291.37-47.

989 Exhibit Q, Report of Professor Stephen Cordner (undated) p 76.

990 Exhibit Q, Report of Professor Stephen Cordner (undated) p 78, citing M A Weber et al, 'Clinicopathological Features of Paediatric Deaths Due to Myocarditis: An Autopsy Series' (2008) 93 *Archives of Disease in Childhood* 594,594-598.

991 Exhibit Q, Report of Professor Stephen Cordner (undated) p 78.

992 Exhibit Q, Report of Professor Stephen Cordner (undated) p 78.

993 Exhibit Q, Report of Professor Stephen Cordner (undated) p 79.

994 Exhibit Q, Report of Professor Stephen Cordner (undated) p 79.

995 Exhibit Q, Report of Professor Stephen Cordner (undated) p 79.

530. Professor Cordner concluded in his report that if Ms Folbigg's conviction in respect of Laura was "stand" it must do so "against the forensic pathology".[996]

531. In his peer review of Professor Cordner's report, Professor Pollanen stated that myocarditis was definitely present, and it is a well-recognised cause of unexpected natural death.[997] He ascribed Laura's death as a "class 2" in the system referred to above at [131].

532. Professor Cordner gave evidence in the Inquiry that sudden and unexpected death was not all that unusual in the population of infants and toddlers dying from myocarditis, happening in about half the cases.[998]

533. He was taken to other aspects of Weber et al (2008). He was asked whether he accepted the authors' conclusion that myocarditis is a rare cause of death in infancy and childhood.[999] He gave evidence on this as follows:

> *FURNESS SC: … The first one is the conclusion that myocarditis is a rare cause of death in infancy and childhood, do you accept that?*
>
> *WITNESS CORDNER: Well you know, I mean if you're making a distinction between rare and uncommon, I mean I'm not sure, I mean on page 596 under "Discussion", second line, "Myocarditis is an uncommon but distinct and recognisable cause of childhood death", so they're just using the word interchangeably.*
>
> *FURNESS SC: Do you accept their conclusion; I'm referring to what their conclusion is?*
>
> *WITNESS CORDNER: Well I'm referring to what they say elsewhere in the article, which is using the word "uncommon", I accept both of them.*
>
> *FURNESS SC: So, you accept myocarditis is a rare cause of death in infancy and childhood?*
>
> *WITNESS CORDNER: Where rare means also uncommon.*
>
> *FURNESS SC: Do you have some difficulty with the word rare Professor?*
>
> *WITNESS CORDNER: No, I'm just--*
>
> *FURNESS SC: It's their word?*
>
> *WITNESS CORDNER: --wondering why you're making such an emphasis on it, I'm happy--*
>
> *FURNESS SC: This is an article that you're relying on?*
>
> *WITNESS CORDNER: Yes.*
>
> *FURNESS SC: And that's their conclusion, that it's a rare cause of death?*
>
> *WITNESS CORDNER: And that's their way of referring to the word "uncommon."*

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996 Exhibit Q, Report of Professor Stephen Cordner (undated) p 91.
997 Exhibit C, Report of Professor Michael Pollanen (1 June 2015) p 5.
998 Exhibit Q, Report of Professor Stephen Cordner (undated) p 79.
999 Transcript of the Inquiry, 21 March 2019 T290.15-16.

FURNESS SC: And what they add in page 598, they say, "What this study adds is that myocarditis is a rare cause of death representing around 2% of paediatric deaths referred for autopsy", and you accept that?

WITNESS CORDNER: Yes.

FURNESS SC: Under the heading "Discussion", which is on the same page, the second column, the first sentence is that "The findings of this study have demonstrated that histologically proven acute myocarditis is an uncommon but distinct and recognisable cause of death", is it your view that Laura had acute myocarditis?

WITNESS CORDNER: Yes.[1000]

534. Professor Cordner's attention was drawn to the study done by Professor Byard (see [506] above), in which he identified a small percentage of children who had died suddenly and unexpectedly from myocarditis.[1001] Professor Cordner did not take issue with the statement.[1002]

535. Professor Cordner believed that the "middle of the road" conclusion in relation to Laura's death was that considered alone, most forensic pathologists would be comfortable ascribing myocarditis, and this was Professor Cordner's own view.[1003] It would, however, have been acceptable and he would support a pathologist who gave the cause of death as undetermined provided that they fully canvassed the possibility that the death could be due to myocarditis "but because it was the fourth death in the particular family there could be other factors, including but not limited to homicide, at work".[1004]

536. In his oral evidence, he maintained these views expressed in his written report, saying that where he had said he would support Dr Cala (who gave the cause of death as undetermined) and fully canvass the possibilities given it was the fourth death, that would include natural causes and also homicide at work.[1005] By the time of the fourth death, homicide would be in his mind.[1006]

Dr Cala

537. In his report dated 26 November 2018, Dr Cala confirmed he remained of the view that myocarditis does not adequately explain Laura's death.[1007]

538. In oral evidence in the Inquiry, Dr Cala was asked whether the description of the inflammatory infiltrate as "light in amount and patchy in distribution" – as stated in his letter of 19 June 2001 to police – was consistent with what he said in the autopsy report.[1008]

539. Dr Cala said that he had described the infiltrate as "moderate, up to moderate" but accepted that there appeared to be a discrepancy. He explained that in areas of examination of the heart, in particular in the left ventricle, the inflammatory infiltrate was light and patchy – in other words, small in amount, with a small number of lymphocytes aggregated around the cardiac cells. However, it was accentuated in areas, in portions in the middle of the left ventricle to put it up maximally to moderate intensity.[1009]

1000 Transcript of the Inquiry, 21 March 2019 T290.15-291.32.
1001 Transcript of the Inquiry, 20 March 2019 T206.35-45.
1002 Transcript of the Inquiry, 20 March 2019 T207.1.
1003 Exhibit Q, Report of Professor Stephen Cordner (undated) p 80; Transcript of the Inquiry, 20 March 2019 T207.15-17.
1004 Exhibit Q, Report of Professor Stephen Cordner (undated) p 80; Transcript of the Inquiry, 20 March 2019 T207.34-45.
1005 Transcript of the Inquiry, 20 March 2019 T207.13-40.
1006 Transcript of the Inquiry, 20 March 2019 T207.42-45.
1007 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 16.
1008 Transcript of the Inquiry, 20 March 2019 T198.1-12.
1009 Transcript of the Inquiry, T198.14-22.

540. Dr Cala also said that pathologists often describe things (such as infection, tumours) as being mild, moderate, severe in amount or intensity. The inflammatory infiltrate in Laura's heart was moderate in intensity at its most severe.[1010]

541. Dr Cala noted that the letter to police was written two years after the final report, and he did not recall going back to the autopsy report to see what his terminology had been. He said that overall it remained his view that the inflammation was light and patchy, but there were areas where it was more severe.[1011] However, he acknowledged that his view was better expressed in the autopsy report than in the letter.[1012]

542. In relation to the statement in his letter that he might, in isolation, give the cause of death as myocarditis, Dr Cala said in his evidence before the Inquiry, that this was because he would be cautious about giving an unequivocal cause of death based purely on a pathological finding. He knew that myocarditis is a potentially serious condition, but would be cautious about looking at slides and without knowing anything else about the case, say that that unequivocally was the cause of death.[1013] He emphasised in his letter that even though he knew of the previous deaths, he was not prejudiced to express any particular view, but his findings were determined just by looking at the material provided.[1014]

543. He agreed in the Inquiry that nonetheless, Laura could have been part of a small number of children who die of myocarditis without showing any symptoms beforehand.[1015] His view in the Inquiry was that Laura did not die of myocarditis but he could not positively exclude myocarditis as being the cause of death.[1016] However, his view remained that it was not a reasonably possible cause of her death and was instead incidental to her death.[1017]

Professor Hilton

544. In his report dated 22 January 2019 Professor Hilton concluded:

> *Laura died with, and highly probably because of, florid myocarditis. There was no medical evidence demonstrable or demonstrated in the report of the post mortem examination to support another cause for her death.*[1018]

545. In oral evidence, Professor Hilton said that he thought "very conservatively that in my opinion Laura might have died with or because of myocarditis", then said that "she may well have died of myocarditis".[1019] He said that he tended to feel myocarditis over any other objective feature in Laura's death.[1020] Referred back to his comments in his report ("highly probably because of"), and to his evidence at trial ("it was the only pathological lesion… that could account for her death" and that it "could possibly" have led to her death), he said:

> *there is no physical evidence, no pathological evidence of any other cause of death, dead she certainly is, myocarditis she certainly has, can myocarditis kill, yes it can, may it well have killed her, is it the favoured diagnosis in this particular case, yes it is.*[1021]

1010 Transcript of the Inquiry, 20 March 2019 T198.14-37.
1011 Transcript of the Inquiry, 20 March 2019 T198.24-48.
1012 Transcript of the Inquiry, 20 March 2019 T199.9-12.
1013 Transcript of the Inquiry, 20 March 2019 T200.42-46.
1014 Transcript of the Inquiry, 20 March 2019 T201.4-8.
1015 Transcript of the Inquiry, 20 March 2019 T203.40-47.
1016 Transcript of the Inquiry, 20 March 2019 T200.38, T204.6-19.
1017 Transcript of the Inquiry, 20 March 2019 T200.38, T204.6-19.
1018 Exhibit O, Report of Professor John Hilton (22 January 2019) p 2.
1019 Transcript of the Inquiry, 20 March 2019 T208.39-40.
1020 Transcript of the Inquiry, 20 March 2019 T208.48-50.
1021 Transcript of the Inquiry, 20 March 2019 T209.28-30.

546. Professor Hilton said that he would not have given "undetermined" but "I don't think it's an entirely unreasoned conclusion from what Dr Cala has told us."[1022]

Professor Duflou

547. In his report dated 13 February 2019, Professor Duflou stated that in his opinion there was "without doubt myocarditis of a severity which can readily cause sudden and unexpected death".[1023] He stated both that severe myocarditis can be incidental, while relatively mild myocarditis can readily cause death.[1024] He noted that there was no competing cause of death identified at autopsy, therefore based purely on autopsy findings cause of death would be given as myocarditis.[1025] If so, in all likelihood the myocarditis would have resulted in a lethal cardiac arrhythmia and caused sudden death, given an absence of features of congestive cardiac failure at autopsy and descriptions of Laura not being obviously short of breath in the days leading up to death.[1026]

548. Professor Duflou stated that myocarditis can either cause death through the gradual development of congestive heart failure or can result in a sudden onset lethal cardiac arrhythmia without evidence of prior illness in the patient. He had seen multiple examples of both.[1027] He endorsed Professor Cordner's conclusion.[1028]

549. However, Professor Duflou went on to note:

> *Acknowledging that there is no other obvious cause of death in Laura, I nevertheless consider it not unreasonable to give the cause of death as UNDETERMINED in the alternative, as proffered by Dr Cala. The reason for this is the knowledge that myocarditis can be incidental to death, and the fact that three siblings died leads one to consider causes of death where death is not simply due to myocarditis but that the myocarditis may have been a contributor or incidental to death in this case.*[1029]

550. In oral evidence Professor Duflou confirmed his view that a cause of death of undetermined was not unreasonable but said he was also "more than happy to give it as myocarditis".[1030] He considered it to be possible that there was involvement by a person causing the deaths of the children and accepted Professor Cordner's opinion that because Laura's death was the fourth death, there could be other factors including but not limited to homicide.[1031]

VIFM

551. The Inquiry received into evidence opinions provided by forensic pathologists at the VIFM and referenced in Professor Cordner's report.[1032]

552. In his report, Professor Cordner recounted consulting with 10 colleagues at the VIFM including giving them photomicrograph images from the slides of Laura's heart (not the whole seven slides). He provided a case outline to his colleagues which read:

1022 Transcript of the Inquiry, 20 March 2019 T209.30-210.6.
1023 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 34.
1024 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 35.
1025 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 35.
1026 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 35.
1027 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 34.
1028 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 41.
1029 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 35.
1030 Transcript of the Inquiry, 20 March 2019 T208.1-2.
1031 Transcript of the Inquiry, 20 March 2019 T208.21.
1032 Exhibit AM, Reports of seven forensic pathologists of VIFM.

> *This girl was 19 months old when she died. She had a runny nose for a couple of days. She was fed at 7 am, playing normally at about 11 am. She then had a sleep and when her mother went to check on her around midday, she was not breathing. Pathologist gave the cause of death as unascertained. Apart from myocarditis, which the pathologist reported as being present, the autopsy was negative. I would be happy with myocarditis as the cause of death. Any comments on this, or on the myocarditis itself? Would appreciate feedback.*[1033]

553. The responses that Professor Cordner received included "very impressive myocarditis", "apparently wide-spread", "florid". Seven or eight of the forensic pathologists said or indicated they would put myocarditis as cause of death.[1034]

554. Dr Cala was critical of this process and asserted that no conclusions should be drawn from the research.[1035] He noted that there were seven blocks of heart tissue that were made into glass slides and which became a part of the overall slides from tissue sampled at autopsy. Professor Cordner did not show the VIFM forensic pathologists the actual seven slides but rather photomicrographs, which Dr Cala said were not representative.

555. Dr Cala observed that one photomicrograph appears to be of a section of heart at low power with the rest "high power" and stated that this sampling would give a highly distorted picture of the amount of myocarditis present: other areas of heart sampled were not shown; hundreds of images at high powers of magnification would be needed to depict the entire heart tissue samples.[1036]

556. Further, Dr Cala considered that it appeared Professor Cordner may have sampled the "worst" or most severe areas of myocarditis which were not a true or accurate representation of the amount of inflammation that Dr Cala saw on the seven glass slides.[1037] Professor Cordner also provided minimal information on the autopsy findings, and even negative findings can be important in certain circumstances.[1038] Dr Cala noted that Professor Cordner's case outline related to only one death in isolation and did not refer, in particular, to the fact that this death was the fourth.[1039]

557. Finally, because in his email to VIFM pathologists, Professor Cordner stated specifically that "I would be happy with myocarditis as the cause of death", the group may have been biased in favour of a diagnosis of myocarditis before examining the images.

Professor Skinner

558. In his expert report paediatric cardiologist Professor Jonathan Skinner confirmed he had reviewed the ECG taken by the ambulance officers on arrival and concluded that the tracing showed an agonal rhythm, which is a sign of a very sick, dying heart. He said that this occurs in children most commonly during or after a failed resuscitation after respiratory arrest, asphyxia or from a neurological cause, but it can also occur following a primary cardiac arrest, most typically in those with an already sick heart.[1040]

559. Professor Skinner found that "the presence of this rhythm makes a non-cardiac death more likely than one from a primary cardiac arrhythmia, but I don't think that's conclusive."[1041]

1033 Exhibit R, Letter from Professor Stephen Cordner to the Inquiry (8 March 2019).
1034 Exhibit AM, Report of Dr Yeliena Baber (30 January 2019) pp 3- 4, Report of Professor Noel Woodford (18 January 2019) p 3; Exhibit Q, Report of Professor Stephen Cordner (undated) p 77.
1035 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 2.
1036 Exhibit N, Further report of Dr Allan Cala (13 February 2019) p 3.
1037 Exhibit N, Further report of Dr Allan Cala (13 February 2019) p 3.
1038 Exhibit N, Further report of Dr Allan Cala (13 February 2019) p 4.
1039 Exhibit N, Further report of Dr Allan Cala (13 February 2019) p 4.
1040 Exhibit Y, Report of Professor Jon Skinner (31 March 2019) p 6.
1041 Exhibit Y, Report of Professor Jon Skinner (31 March 2019) p 6.

560. In the Inquiry Professor Skinner accepted under cross-examination that chest compressions and/or medications have the potential to extend the agonal rhythm:

> *If one is giving CPR and providing some support to the heart then the agonal rhythm may go on longer than if you didn't.*[1042]

Counsel assisting's submissions on cause of Laura's death

561. Counsel assisting submitted that Dr Cala consistently acknowledged that considered in isolation, myocarditis might have caused Laura's death, in both of his letters to police, at trial and in the Inquiry. He did not consider it to be a reasonable possibility and described it as very unlikely.[1043]

562. Counsel assisting submitted that deaths of children from myocarditis are rare, and evidence on this was been given by Professor Byard at trial. Fewer than one child per year in South Australia was identified by Professor Byard in his study spanning over 35 years through to the late 1980s. The study by Weber et al (2008) also concluded that it is rare, representing around two per cent of paediatric deaths referred for autopsy. The analysis of 27 cases identified from the NCIS, discussed in Professor Cordner's report, amounts to about one each in New South Wales and Victoria per year covered by the NCIS search.[1044]

563. Counsel assisting submitted that deaths of children from myocarditis which are sudden and unexpected are even fewer. Professor Byard, for instance, identified five over 35 years; most who have myocarditis do not die and most who do die do not die suddenly and unexpectedly.[1045]

564. Further, the research reported by Weber et al (2008) showed that death from myocarditis in the age range of one to four years is much less frequent than in babies under one year of age.[1046]

565. Counsel assisting submitted that the weight of the evidence is that Laura's myocarditis was moderate, although this description alone does not adequately capture the diffusion or clustering of the infiltrate identified on the histology. It has been recognised and accepted by medical experts at trial and in the Inquiry that moderate (and even, on Professor Duflou's evidence at least, mild) myocarditis can cause sudden unexpected death in a child.[1047]

566. That said, Laura was of an age in which the research referred to above (and ultimately accepted by Professor Cordner) demonstrated that it is particularly rare to suffer a sudden and unexpected death from myocarditis. Laura's myocarditis was not observed upon forensic naked eye examination at autopsy. There was no evidence of other organ dysfunction indicating heart failure. It was fairly diffuse; it appears that there was cell necrosis, but little of it. That is not to suggest that it could not have caused her death; its equivocality, however, has caused reasonable expert minds to differ. No expert at trial or in the Inquiry has comprehensively excluded myocarditis as possibly causing Laura's death.[1048]

1042 Transcript of the Inquiry, 16 April 2019 T509.28-36.

1043 Submissions of counsel assisting (17 May 2019) Chapter 7, [209].

1044 Submissions of counsel assisting (17 May 2019) Chapter 7, [212].

1045 Submissions of counsel assisting (17 May 2019) Chapter 7, [213].

1046 Submissions of counsel assisting (17 May 2019) Chapter 7, [214]; M A Weber et al, 'Clinicopathological Features of Paediatric Deaths Due to Myocarditis' (2008) 93 *Archives of Disease in Childhood* 594, 595.

1047 Submissions of counsel assisting (17 May 2019) Chapter 7, [216].

1048 Submissions of counsel assisting (17 May 2019) Chapter 7, [217].

567. Dr Cala, however, was and remains of the view that myocarditis was incidental to Laura's death and that it does not adequately explain Laura's death. In that respect, counsel assisting submitted there is no change in his opinion between the trial and the Inquiry.[1049] At trial, Professor Herdson also favoured myocarditis as incidental; Professor Berry thought it could be incidental; Professor Byard did not exclude myocarditis but preferred undetermined in context. Professor Busuttil said Laura's death "could have been" caused by myocarditis but it may also have been incidental. Dr Bailey considered it an unlikely cause.[1050]

568. Counsel assisting submitted that Professor Hilton's view has been quite variable. His report provided to the Inquiry indicated he had significantly changed his opinion between the trial and the Inquiry, from that myocarditis "could have" caused Laura's death to a view that she died "highly probably because of" florid myocarditis. He also noted there was no medical evidence which demonstrated support for another cause of death. In the Inquiry, his opinion was ultimately to the effect that undetermined is not entirely unreasoned, but myocarditis is his favoured diagnosis. This appears to be a shift from his position at trial, although not as significant as first appeared from his report.[1051] Professor Duflou would be "more than happy" with myocarditis, but would support undetermined, in the knowledge that myocarditis can be incidental to death and that "it is possible someone was involved".[1052]

569. Professor Cordner considered undetermined would not be unreasonable, with myocarditis an unexceptional diagnosis. He stated in his report, and confirmed in his evidence, his view that, considered alone, it was a "middle of the road" conclusion that Laura's death was due to myocarditis and "undetermined" was acceptable in context provided the reasons (it being the fourth death and other factors could be at work) for this finding were explained.[1053]

570. Counsel assisting submitted that the views of the VIFM pathologists as to the significance of the myocarditis on microphotographs they received, should be afforded no weight in the Inquiry. First, they were only given a selection of microphotographs of varying resolution, and not representative of the slides of Laura's heart. Secondly, they were provided with no information on the circumstances of Laura's death or her clinical and family history. Thirdly, their opinions were obtained by Professor Cordner after he stated his own opinion, namely, that he would be happy with myocarditis as the cause of death by way of contrast to the opinion given by Dr Cala (unnamed in the email) as unascertained.[1054] Professor Cordner sought their comments without revealing that he would accept a finding of "unascertained".[1055]

571. Counsel assisting submitted that as with other evidence in the case, the evidence given at trial about myocarditis in relation to Laura's death needs to be considered in light of the further evidence received in the Inquiry. When Professor Cordner's and Professor Duflou's opinions in particular are weighed with expert evidence at the trial, there is a degree of difference. That difference is seen by inclusion of two expert opinions that when the autopsy findings in relation to Laura are considered alone, myocarditis would be an unexceptional diagnosis or would be the cause of death in the absence of a competing cause.[1056]

572. But, counsel assisting submitted, the degree of difference is tempered by the ultimate qualification which attended both opinions. Both were qualified as being on autopsy results alone. Neither excluded the possibility of an unnatural cause. Both considered "undetermined" would be supported and that the possibility of other factors should be considered or canvassed, including homicide in all the circumstances.[1057]

1049 Submissions of counsel assisting (17 May 2019) Chapter 7, [218].
1050 Submissions of counsel assisting (17 May 2019) Chapter 7, [219].
1051 Submissions of counsel assisting (17 May 2019) Chapter 7, [220].
1052 Submissions of counsel assisting (17 May 2019) Chapter 7, [221]; Transcript of the Inquiry, 20 March 2019 T208.1-15.
1053 Submissions of counsel assisting (17 May 2019) Chapter 7, [222].
1054 Exhibit R, Letter from Professor Stephen Cordner to the Inquiry (8 March 2019).
1055 Submissions of counsel assisting (17 May 2019) Chapter 7, [208].
1056 Submissions of counsel assisting (17 May 2019) Chapter 7, [223].
1057 Submissions of counsel assisting (17 May 2019) Chapter 7, [224].

573. Overall, counsel assisting submitted that it may be said that there is a difference in the range of opinions on the role of myocarditis in Laura's death now, upon autopsy findings alone, as compared with the range of opinions given at trial. There is no difference, however, in expert opinion on the possibility of an unnatural cause having caused her death.[1058]

574. In view of the microscopic findings by Dr Cala, the analysis by forensic pathologists both at trial and in the Inquiry in relation to the autopsy findings considered alone, the express acknowledgement by every forensic pathologist who has given an opinion in relation to Laura's death of the possibility of an unnatural cause, the particular rarity of sudden and unexpected deaths of children from myocarditis and even more so in the age range of one to four years, it is submitted that myocarditis is a possible cause of Laura's death. However, in counsel assisting's submissions there was no evidence received in the Inquiry which would elevate myocarditis as more than a possible cause.[1059]

Ms Folbigg's submissions on cause of Laura's death

575. Ms Folbigg submitted that Laura was not a well child, had myocarditis which could trigger a cardiac arrhythmia, and that there was great difference between trial and Inquiry evidence on this issue.[1060] She contended that the Crown prosecutor significantly overstated the evidence of experts on myocarditis in his "pigs might fly" address. (This address is considered in Chapter 3.)

576. Ms Folbigg submitted that Dr Cala's evidence at trial took no account of the potential for infection to cause a sudden cardiac arrhythmia, there was no evidence about a combination of genetic variants together with infection that could cause death, and the Crown prosecutor created a link between heavy infiltrate of myocarditis and/or a structural anomaly and death, whereas death can occur even with light infiltrate.[1061] Dr Cala gave no reasons for his opinion that it was very unlikely that myocarditis caused death.[1062] Ms Folbigg also submitted that Dr Cala's evidence at trial – that SIDS excludes unnatural causes – was incorrect and confusing.[1063]

577. Ms Folbigg observed that Dr Cala was the most junior of the forensic pathologists who gave evidence at the trial and the Inquiry and that his conclusions about causes of death at trial are not accepted by more senior and experienced forensic pathologists.[1064] She also submitted that he did not provide adequate reasons for his opinions as to the causative impact of myocarditis and shifted significantly from his evidence at trial.[1065] She submitted that I should be reluctant to accept Dr Cala's opinion on this issue.

578. Ms Folbigg also made the submission that:

> *the predominant basis for Dr Cala not to conclude the death was caused by myocarditis was the existence of three other deaths in the one family. In the event that for any of those deaths there is a reasonably available alternative natural cause then, by implication, Dr Cala would shift his opinion that myocarditis is the probable cause of death.*[1066]

1058 Submissions of counsel assisting (17 May 2019) Chapter 7, [225].
1059 Submissions of counsel assisting (17 May 2019) Chapter 7, [225].
1060 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Laura, [1].
1061 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Laura, [5]-[7].
1062 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Laura, [9].
1063 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Laura, [11].
1064 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Laura, [68].
1065 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Laura, [70].
1066 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Laura, [63].

579. Ms Folbigg submitted that the Crown's ninth point of coincidence, that:

> *Each child was discovered dead or moribund at around or shortly after death when they were still warm to the touch, and two of them still had a heartbeat, so they were found literally minutes after the cessation of breathing*[1067]

was wrong because the a heartbeat does not equate to an agonal rhythm.[1068]

580. Further, the agonal rhythm can be perpetuated by the administration of drugs and resuscitation attempts, which removes any certainty between the heart stoppage and detection of the agonal rhythm.[1069] Ms Folbigg submitted that Professor Skinner conceded that here the agonal rhythm was likely extended by paramedical interference, and in any event, Laura already had a sick heart as she suffered from myocarditis.[1070]

581. Ms Folbigg also submitted that Professor Skinner's evidence means that the following section of the Crown's closing was wrong:

> *Because of that agonal rhythm it is more likely that the breathing stopped before the heart which is not what you would expect from myocarditis as a cause of death. It is more consistent with smothering than with myocarditis.*[1071]

582. Ms Folbigg summarised that the majority of forensic pathologists in the Inquiry would have classified Laura's death as being caused by myocarditis.[1072] She submitted I should find that myocarditis was the likely cause of Laura's death.[1073]

Dr Cala's submissions on cause of Laura's death

583. The Inquiry received submissions from Dr Cala in response to the submissions made by Ms Folbigg in relation to the death of Laura.[1074]

584. Dr Cala submitted that there appears to be very little, if any, difference between what he wrote in Laura's autopsy report and what Professor Cordner agrees a pathologist should have written.[1075]

585. Dr Cala's submissions noted that while he might have been the most junior of the forensic pathologists who gave evidence at the trial, Professor Hilton gave evidence that the autopsy Dr Cala conducted was very thorough, he had no criticisms of Dr Cala at the time in relation to his autopsy report, and he "supported" Dr Cala in relation to his views expressed therein.[1076]

1067 Submissions of the ODPP to the Inquiry (24 May 2019) p 7.
1068 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [109]-[111], [113].
1069 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [112].
1070 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [127]-[130].
1071 13 May 2003 T1357.46-49.
1072 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Laura, [71].
1073 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Laura, [75].
1074 Submissions of Dr Allan Cala to the Inquiry (14 June 2019).
1075 Submissions of Dr Allan Cala to the Inquiry (14 June 2019) [4]-[5].
1076 Submissions of Dr Allan Cala to the Inquiry (14 June 2019) [6].

586. Dr Cala submitted that Ms Folbigg's submissions are premised on the fact that he was the only expert at trial who expressed the view that myocarditis was unlikely to be the cause of death. However, many other forensic pathologists at trial held the same and even stronger views.[1077] Contrary to the submissions made by Ms Folbigg, Dr Cala did not give evidence at the trial that smothering was the cause of death of Laura but instead expressed the view that deliberate suffocation or unnatural causes could not be excluded.[1078]

587. Dr Cala emphasised that Professors Cordner's and Duflou's views expressed at the Inquiry were qualified as being on autopsy results alone, in isolation from the deaths of the other three siblings, but that all forensic pathologists agreed Laura's death could not be considered in isolation. Professor Cordner also accepted that it was reasonable for Dr Cala to list Laura's cause of death as undetermined.[1079]

Professor Hilton's submissions on cause of Laura's death

588. In his submissions in response, Professor Hilton said that Ms Folbigg's submissions fairly reflected his opinion as to the possible cause of death of Laura.[1080]

589. He submitted that he maintained in the absence of another cause of death being found, he would have given the cause of death of Laura as myocarditis, and emphasised that he supported Dr Cala in his finding of undetermined in the sense that that was Dr Cala's opinion.[1081]

Findings: Laura

590. The evidence given in the Inquiry has not changed the evidence at trial where Professor Berry said most forensic pathologists would say myocarditis was the cause of death of Laura.[1082] That is clearly because from the point of view of a forensic pathologist, myocarditis is the only possible observable cause of Laura's death. Professor Berry also said the amount of myocarditis is not critical because a small amount can kill but a large amount may not.

591. Having regard to all of the evidence received in the Inquiry, the myocarditis found in Laura's heart at autopsy could have been incidental to her death, or it could have been fatal. The answer to the question of which of these it was lies not in the medical evidence in relation to Laura, but in a consideration of a number of different aspects of the evidence in this case.

592. However, as was given in evidence by Professor Byard at trial, deaths of children from myocarditis are rare. He said that he had identified fewer than one child per year in South Australia in the study he conducted covering 35 years. This conclusion is consistent with the study by Weber et al (2008). On the basis of the analysis of 27 cases identified from the NCIS, discussed by Professor Cordner, similarly there were about one each in New South Wales and Victoria per year.

593. There is force in counsel assisting's submissions also, that deaths of children from myocarditis which are sudden and unexpected are even fewer and that, per Weber et al (2008), death from myocarditis in the age range of one to four years is much less frequent than in young babies. Laura was of an age at which, on the research, it is particularly rare to suffer a sudden and unexpected death from myocarditis. However, this is simply one circumstance to take into account. It is a separate consideration from whether Laura's myocarditis was sufficient to kill; I have accepted that most forensic pathologist would say that it was.

1077 Submissions of Dr Allan Cala to the Inquiry (14 June 2019) [9].
1078 Submissions of Dr Allan Cala to the Inquiry (14 June 2019) [15].
1079 Submissions of Dr Allan Cala to the Inquiry (14 June 2019) [16]-[23].
1080 Submissions of Professor Hilton to the Inquiry (18 June 2019).
1081 Submissions of Professor Hilton to the Inquiry (19 June 2019) [2].
1082 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 26.

594. While the views of the VIFM pathologists were received into evidence in the Inquiry, I think the value of their opinions was compromised to a degree particularly by the limited information with which they were provided. That said, again their views simply bear out Professor Berry's opinion given at trial.

595. In respect of Ms Folbigg's submissions about the agonal rhythm, I accept that this rhythm does not equate to a "heartbeat" in the usual sense of the word.

596. However, this difference was identified in the summing up and was therefore brought to the attention of the jury:

> *You will remember, when the ambulance officers attended, a test was done that showed that Laura's heart was still exhibiting some electrical signs. It was not beating normally. In fact I do not know that one can say that the heart was beating in the sense of an ordinary heart beating, but there was still some electrical activity in the heart. The evidence was that that electrical activity might continue for some time after a person had stopped breathing for some minutes.*[1083]

597. The evidence is clear that Patrick was found with a heartbeat following his ALTE.[1084] I therefore reject the submission that the Crown's ninth point of coincidence has been eliminated.

598. Contrary to Ms Folbigg's submissions, I do not see that any significance can be attributed to the fact that intervention by compressions or medication can extend the agonal rhythm. The evidence was not that the intervention can *create* an agonal rhythm.

599. In any event, at trial, some experts considered that the presence of an agonal rhythm in Laura meant it was more likely that her breathing stopped before her heart stopped. Other experts were of the view that the presence of the rhythm did not assist in determining the sequence of events. In the Inquiry, Professor Skinner considered that in 2019 it was still not possible to be conclusive.

600. In these circumstances, I find that the evidence regarding the presence of an agonal rhythm does not assist in determining the cause of Laura's death.

601. No forensic pathologist has excluded the possibility of an unnatural cause of Laura's death.

602. Again, while Ms Folbigg's submission that evidence regarding the potential role of infection is new and was not adduced (except in a limited way) at trial is correct, for reasons set out in Chapter 6, I do not infer that the presence of infection makes it more (or less) likely that Laura's death was from myocarditis. Expert evidence was to the effect that her myocarditis was most probably viral in origin. It is accepted that Laura had a virus in the days prior to her death.

603. For clarity's sake, I find that there is no evidence of any contribution to Laura's death by genetic factors.

604. I find on the available medical evidence that myocarditis was a possible cause of Laura's death. On the available medical evidence it is also reasonably possible that her death was caused by an event leading to obstruction of her airways, which includes deliberate smothering.

[1083] 20 May 2003 T102.

[1084] Exhibit S, Medical Records of Patrick, Ambulance Report (18 October 1990) pp 526-527.

Evidence as to smothering

Overview

605. Smothering can leave signs which may be found at autopsy, but smothering can occur without such signs.[1085] The pathological findings following suffocation are often completely non-specific, or there may be virtually nothing to find. Even where smothering may be suspected, it is often impossible to distinguish between SIDS and deliberate or accidental suffocation.[1086] In this respect there is no relevant difference between the evidence given in the Inquiry, and evidence at the time of trial.[1087] As a consequence, in Professor Cordner's words, diagnosed smothering is "very, very unusual, rare".[1088]

606. Facial signs of smothering include petechia on eyelids, cheeks, surface of eyes; damage to the fraenulum; and bruising on the inside of the lips.[1089] Professor Duflou observed in this context that petechial haemorrhages are relatively uncommon in infant cases.[1090] The presence of external signs may depend on what was used.[1091] Absence of any or all of these signs does not exclude the possibility of smothering.[1092]

607. There may be petechial haemorrhages on the heart, lungs and thymus, although these are also non-specific and can be found in many children where SIDS is diagnosed.[1093]

608. In his report tendered in the Inquiry, and consistently with the above, Dr Cala stated that pathologists are generally unable to indicate the true extent of smothering.[1094] Physical evidence after fatal smothering could be limited – anything from nil to "some" – and whether there is evidence depends upon factors such as the age of child, the amount of struggle, whether the child has teeth, whether the child was debilitated or robust, whether the child was sedated, and the nature of any injuries (such as bruises or abrasions around the outer airways, compression marks from teeth on lips, blood in nose and mouth from local trauma – all of which may be explained by other mechanisms such as CPR).[1095]

609. Dr Cala noted that pressure on the chest may also leave physical signs, such as petechial haemorrhages on the face and rupture of tiny blood vessels in, for example, eyelids, cheeks and forehead.[1096] However, any physical signs left after smothering may be much more difficult to interpret when extensive CPR has occurred.[1097]

1085 14 April 2003 T650.46-651.7 (Professor Hilton); Transcript of the Inquiry, 19 March 2019 T105.26-43 (all forensic pathologists agreeing).

1086 1 May 2003 T1034 (Professor Herdson); 14 April 2003 T649.4-12, T653.30-34, T655.54-656.6 (Professor Hilton); 7 May 2003 T1222 (Professor Byard); Transcript of the Inquiry, 19 March 2019 T111.31-112.8 (all forensic pathologists agreeing).

1087 14 April 2003 T649.4-12, T653.30-34, T655.54-656.6 (Professor Hilton); 7 April 2003 T267.56-268.11 (Dr Springthorpe); 5 May 2003 T1136.27-48; Exhibit H, Forensic pathology tender bundle, Statement of Dr Susan Beal (8 December 1999) p 2; 28 April 2003 T982.14-30, T1136.27-48 (Dr Beal); 15 April 2003 T710.9-36, T713.6-16, T729.31-39 (Dr Cala); Transcript of the Inquiry, 19 March 2019 T111.31-112.8 (all forensic pathologists agreeing).

1088 Transcript of the Inquiry, 19 March 2019 T107.13-15.

1089 14 April 2003 T650.53-65, T651.1-4 (Professor Hilton).

1090 Transcript of the Inquiry, 19 March 2019 T106.30-33.

1091 Transcript of the Inquiry, 19 March 2019 T105.22-108.9 (Professor Hilton, Dr Cala, Professor Cordner, Professor Duflou).

1092 15 April 2003 T710.32-36 (Dr Cala).

1093 15 April 2003 T710.9-36 (Dr Cala); 1 May 2003 T1037.25-52 (Professor Herdson); Exhibit D, Roger W Byard, 'The Autopsy and Pathology of Sudden Infant Death Syndrome' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 497, 503-504.

1094 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 18.

1095 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 18.

1096 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 18.

1097 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 18.

Evidence at the time of trial

610. At trial, Dr Cala and Dr Beal gave evidence that in their view either all the children died in circumstances consistent with deliberate smothering, or suffocation in relation to the death of each of them could not be ruled out.[1098] Professor Herdson opined that the four children "probably died from intentional suffocation."[1099]

611. Dr Cala said that the absence of petechial haemorrhaging on eyelids and around the eyes in Laura was non-specific – their absence did not exclude the possibility and could not be used to differentiate SIDS over smothering.[1100] Dr Cala observed it may be very easy to smother a very young child but in a child of 19 months, it could take 20-30 seconds or longer.[1101] Similarly, Professor Hilton said that the amount of force required to deliberately smother a 10 month old child with a pillow is fairly small and agreed that one would not necessarily expect to find signs.[1102]

612. Dr Beal stated that the macroscopic and microscopic examination is rarely helpful, and facial bruising or petechiae on occasion may point away from SIDS.[1103]

613. At trial, Professors Berry and Byard gave evidence that suffocation in young children often leaves no trace.[1104]

614. Professor Berry opined that confined to the pathology, and in isolation, there were no positive findings of suffocation for any of the children.[1105] He observed in his first report that suffocation in young children is often unaccompanied by any external signs, and there are no diagnostic internal findings – petechial haemorrhages beneath the capsule of the thymus, the pleura and the pericardium are commonly found but are also described in SIDS.[1106] He stated that the deaths of Caleb, Patrick and Sarah were "entirely compatible with suffocation as the cause".[1107] He concluded that the sudden and unexpected death of three children in the same family without evidence of a natural cause was "extraordinary", and that he was unable to rule out that Caleb, Patrick, Sarah and "possibly Laura" were suffocated, and that he "believe[d] that it is probably that this was the cause."[1108]

615. Professor Byard gave evidence that suffocation could not be excluded.[1109] He said it is often impossible to distinguish between SIDS and suffocation, as suffocation often leaves no trace, particularly in a baby or young child.[1110] Deliberate suffocation was possible in all the deaths and the ALTE in the case of Patrick, but there was no definite pathology.[1111]

1098 16 April 2003 T749.27-33; 5 May 2003 T1138.42-48 (Caleb), T1139.58-1140.2 (Patrick), T1142.25-28 (Sarah), T1143.31-34 (Laura), T1145.42-47.

1099 Exhibit H, Forensic pathology tender bundle, Statement of Professor Peter Herdson (17 January 2002) p 3.

1100 15 April 2003 T709.55-58, T710.1-36.

1101 15 April 2003 T713.9-24.

1102 14 April 2003 T656.20-36.

1103 28 April 2003 T982.1-7.

1104 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 26; 7 May 2003 T1205.43-T1206.15, T1222.12-15, T1223.28-31 (Professor Byard); 1 May 2003 T1055.42-49, T1074.16-31.

1105 1 May 2003 T1074.27-31, T1082.45-51; Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 26.

1106 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 26.

1107 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 26.

1108 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 26.

1109 7 May 2003 T1222.17-23.

1110 7 May 2003 T1222.12-15.

1111 7 May 2003 T1222.17-23, T1225.35-39, T1249.5-16.

616. Professor Byard also gave evidence that when choosing between pathological findings at autopsy, one often needs information about the person's immediate history.[1112] However, he agreed that he could not say what the cause of death was for each of the children other than each was "undetermined", which includes death from natural and unnatural causes (including deliberate suffocation).[1113] It was possible that all the deaths and ALTEs were caused by deliberate suffocation, with the difficulty being that the pathology did not really help.[1114]

617. Professor Ouvrier (paediatric neurologist, who did not give oral evidence, and his report was not tendered in the trial), referring to Patrick's ALTE in the context of all four children, stated that:

> *[a] series of such events in four siblings with exclusion of other underlying pathological states (such as metabolic disorders, cardiac conditions or epilepsy) would be more likely to be due to deliberate suffocation than any other cause.*[1115]

618. Professor Busuttil, forensic pathologist, stated that it could not be said, "indeed beyond reasonable doubt", that the deaths were "irrefutably" due to imposed or induced airways obstruction (as by suffocation).[1116]

Evidence in the Inquiry

619. Professor Cordner said that smothering could not be excluded in any of the Folbigg children (but there are good grounds for thinking that Laura, at least, was not smothered).[1117] Professor Duflou could not exclude smothering in relation to any of the children, but could not include it either, there being no evidence for it.[1118]

620. In his peer review, Professor Pollanen stated that the medical determination of homicidal mechanical asphyxia in the case could not be sustained, for two reasons. First, the facts do not support the conclusion, since two of the deaths are reasonably interpreted as due to natural causes. Secondly, the diagnosis of asphyxia is not sufficiently evidence-based, in the case, to be considered medically or scientifically reliable.[1119]

621. Dr Cala's opinion remained that there exists the possibility that each of the Folbigg children died not from natural disease but from inflicted injury, most likely in the form of smothering.[1120] Dr Cala's basis for suspecting homicide in relation to the children was his concern about the existence of four deaths in one family where he was not satisfied with the causes of death that had been given.[1121] He agreed with Professor Cordner where he stated in his report that smothering could have occurred in this case but left no trace.[1122]

622. There were no damaged fraenula, facial bruises or abrasions (aside from on Sarah's chin), or petechial haemorrhages to the eyes found in the Folbigg children.[1123] In relation to Laura, Dr Cala conducted a facial dissection and did not find any bruises or other injuries.[1124]

1112 7 May 2003 T1225.7-12.
1113 7 May 2003 T1225.14-39.
1114 7 May 2003 T1225.14-39.
1115 Exhibit H, Forensic pathology tender bundle, Report of Dr Robert Ouvrier (28 October 2002) p 5.
1116 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002) p 15.
1117 Transcript of the Inquiry, 20 March 2019 T162.12-15.
1118 Transcript of the Inquiry, 20 March 2019 T181.38, T181.42.
1119 Exhibit C, Report of Professor Michael Pollanen (1 June 2015) p 3.
1120 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 25.
1121 Transcript of the Inquiry, 21 March 2019 T277.13-14.
1122 Exhibit M, Report of Dr Allan Cala (26 November 2018) p 23, referring to Exhibit Q, Report of Professor Stephen Cordner (undated) p 91.
1123 Transcript of the Inquiry, 21 March 2019 T247.41-T249.24; 16 April 2003 T752.16-23 (Dr Cala).
1124 Exhibit H, Forensic pathology tender bundle, Autopsy report of Laura (13 December 1999) pp 8-9.

623. In his report, Professor Cordner stated that the lack of facial injuries in the Folbigg children is evidence *against* a conclusion of smothering, particularly in relation to Laura, and should be regarded as having some weight.[1125] While it is true that fatal smothering may leave no signs, smothering can result in general and specific signs, the fact that none of which were seen in any of the four children, including in Patrick's ALTE, might be thought to be worthy of remark and should be acknowledged.[1126]

624. Professor Cordner also referred to results of a search of the NCIS showing first, that since 2000 smothering had rarely been concluded as the cause of death in children of two years or younger and second, that smothering does leave signs in some infant and childhood cases, with two fifths of cases having both general and some specific signs.[1127]

625. In oral evidence, Professor Cordner said that major signs of smothering include external injuries around the nose and mouth, and internal injuries generally around the mouth (such as bruising inside lips or fraenulum, and facial petechiae).[1128] He agreed that, broadly speaking, whether there are signs will depend upon the force used, instrument or implement, part of the body and the time taken.[1129]

626. Professor Hilton said smothering may be suspected but is almost impossible to prove, Professor Duflou agreeing in respect of a significant percentage of cases.[1130] Professor Hilton agreed that the fraenula is quite commonly bruised, Dr Cala saying it can be bruised and torn but not agreeing it was common.[1131]

627. Professor Duflou emphasised in his report that no smothering injuries of any type were seen in any of the children, and as such was of the opinion that a diagnosis of smothering could not be reasonably suggested by the expert in court proceedings.[1132] He stated that historically the classical autopsy signs of asphyxia, including petechial haemorrhages, congestion and oedema, cyanosis, and engorgement of the right heart and fluidity of blood have been "roundly debunked" as non-specific since at least 1974, with warnings since 1949.[1133] The mere finding of any of the non-specific features, without firm circumstantial or preferably physical evidence of mechanical obstruction of respiration, is quite insufficient to warrant a speculative diagnosis of asphyxia. If such collateral evidence is not forthcoming, the cause of death must be left undetermined.[1134]

628. Further, in his oral evidence, Professor Duflou thought that in the general population, there would be a greater likelihood of there being signs of smothering in subsequent deaths because of there being, in every case, a possibility of signs.[1135] Dr Cala did not agree.[1136]

1125 Exhibit Q, Report of Professor Stephen Cordner (undated) pp 52-53.
1126 Exhibit Q, Report of Professor Stephen Cordner (undated) pp 51-52.
1127 Exhibit Q, Report of Professor Stephen Cordner (undated) p 52.
1128 Transcript of the Inquiry, 21 March 2019 T248.49-50, T249.1-9.
1129 Transcript of the Inquiry, 19 March 2019 T107.48-50, T108.1.
1130 Transcript of the Inquiry, 19 March 2019 T111.24-38.
1131 Transcript of the Inquiry, 21 March 2019 T247.39-248.8.
1132 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 38.
1133 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 40.
1134 Exhibit L, Report of Professor Johan Duflou (13 February 2019) pp 40-41, citing Pekka Saukko and Bernard Knight, *Knight's Forensic Pathology* (CRC Press, 3rd ed 2004).
1135 Transcript of the Inquiry, 20 March 2019 T185.5-10.
1136 Transcript of the Inquiry, 20 March 2019 T185.46.

629. Consistently with Professor Duflou's recount of certain signs now being seen as non-specific, Professor Pollanen referred to the Goudge Inquiry report for a detailed discussion but summarised that:

> *it is now recognized in mainstream forensic pathology that there are no "signs of asphyxia" at autopsy. The only post-mortem findings that are highly relevant to the diagnosis of asphyxia are injuries produced by the trauma that caused the mechanical asphyxia. In the absence of the latter, there is no anatomical basis for a diagnosis of asphyxia… In most such cases, the conclusion is typically based on the history coupled with the inability to refute asphyxia at autopsy. However, this is rarely understood by non-pathologists*.[1137]

630. In his report, Dr Cala agreed with Collins and Byard (2014) that many forms of paediatric asphyxia will, more often than not, have negative autopsies or autopsies with non-specific findings and in most cases, findings will not differ from findings in SIDS. Therefore, the death investigation (scene, medical history, witnesses) is of paramount importance. The likelihood of petechiae is highly dependent on the type of asphyxia.[1138]

631. Professor Hilton said that in his limited experience of people dying from putting their heads in plastic bags, there are absolutely no signs of anything at all.[1139] Dr Cala agreed, having seen quite a lot of these.[1140]

632. Professor Cordner has never had a case in which he has diagnosed smothering in an infant. Indeed, he said that facial petechiae are rarely present in cases of infant smothering.[1141] Internal signs such as biting a cheek are more likely in an adult.[1142]

Submissions of counsel assisting on forensic pathology evidence as to smothering

633. Counsel assisting noted that no forensic pathologist at trial, or in the Inquiry, has excluded the possibility that each instance of death or ALTE could have been caused by smothering.

634. In relation to Professor Cordner's opinion that the lack of facial injuries is negative evidence against smothering, counsel assisting submitted that neither at trial nor in the Inquiry did other medical experts place this negative weight upon the evidence of post-mortem findings. It is, on the other hand, clearly accepted that as a general proposition, smothering is very hard for a forensic pathologist to distinguish from SIDS. It is rarely diagnosed. It may well leave no physical signs.

635. Counsel assisting submitted that in view of all of the forensic pathology evidence on the likelihood of finding injuries or petechiae indicative of smothering upon autopsy, there is little support for Professor Cordner's opinion that the absence of facial signs weighs against a conclusion of smothering.

636. In relation to Professor Duflou's opinion that in the general population, there would be a greater likelihood of there being signs of smothering in subsequent deaths because of there being, in every case, a possibility of signs, counsel assisting submitted that the opinion amounted to little more than conjecture and was not cogently argued or persuasive.

1137 Exhibit C, Report of Professor Michael Pollanen (1 June 2015) (citations omitted).

1138 Exhibit M, Report of Dr Allan Cala (26 November 2018) pp 20-21, citing Andrew M Baker, 'Pediatric Asphyxial Deaths' in Kim A Collins and Roger W Byard (eds), *Forensic Pathology of Infancy and Childhood* (Springer New York, 2014) 207.

1139 Transcript of the Inquiry, 19 March 2019 T115.16-18.

1140 Transcript of the Inquiry, 19 March 2019 T115.25-33.

1141 Transcript of the Inquiry, 19 March 2019 T106.28-35, T106.44-50, T107.44-49.

1142 Transcript of the Inquiry, 19 March 2019 T106.33.

Ms Folbigg's submissions on forensic pathology evidence as to smothering

637. In her submissions Ms Folbigg did not challenge the proposition that no expert had excluded smothering. Rather, she said this did not matter because the issue was whether any expert could exclude a reasonable alternative natural cause of death.[1143] Further, it is not a matter for asphyxia or smothering to be excluded but rather, "whether a state of facts existed or otherwise".[1144] Ms Folbigg submitted that, therefore, the Inquiry needs to be reasonably satisfied that asphyxia or smothering occurred on the evidence before it, and it could not be so satisfied in relation to any of the children.[1145]

638. Ms Folbigg submitted that given the charges were of murder, which were said to have been committed by smothering, evidence of smothering needed to be firmly established by the Crown to the exclusion of a reasonably available natural cause.[1146] The absence of evidence of smothering was not a sound basis to introduce the coincidence evidence.[1147]

639. Further, in the context of the coincidence relied upon by the Crown that there were no signs of injury found on any child, Ms Folbigg submitted that this fact points away from her involvement in their deaths (particularly given she was said to be in a blind rage at the time of the killings). If Laura had been smothered, it was likely there would have been injury or prior evidence of abuse.[1148]

640. Ms Folbigg noted that Professor Cordner had reported that in three out of five cases of confirmed smothering, there were identified injuries to the face or mouth, in support of a submission that filicide is rare, filicide by a woman is rarer than by a man, filicide by a mother rarer still, and by a mother leaving no signs rarer again.[1149] She submitted there was a lack of any evidence to support the smothering hypothesis, and the risk of unintentional bias and prejudgement is high.[1150]

Findings

641. In short, the medical evidence received in the Inquiry, when considered on its own and not in the light of any other evidence, neither proves nor disproves that any of the children were smothered.

642. In this regard, I agree with Professor Cordner that there is no forensic pathology or medical basis (in isolation from any other evidence in the case) for concluding homicide, findings on post-mortem are compatible with natural causes, and findings on post-mortem cannot rule out smothering in relation to any of the children.[1151]

643. No forensic pathologist whose evidence has been received in the Inquiry, including those who gave evidence or reports at the time of trial, has excluded the possibility that each instance of death or ALTE could have been caused by smothering. That is a circumstance which, when forming my conclusions, I take into account.

[1143] Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [39]; Part C – Laura, [74]; Part C – Patrick [9]; Part C – Sarah, [57].
[1144] Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [28].
[1145] Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [28]-[30].
[1146] Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [29]-[30]; Part C – Laura [74].
[1147] Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [11].
[1148] Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [19(i)].
[1149] Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [79(a)].
[1150] Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Patrick, [95].
[1151] Exhibit Q, Report of Professor Stephen Cordner (undated) p 8.

644. In a circumstantial case, a finding of guilt should be the only rational conclusion that can be drawn from the circumstances. The question of smothering is the ultimate issue, with an opinion on this to be formed upon all of the circumstantial evidence, to the criminal standard of proof. I have already addressed in further detail in Chapter 3 the principles that apply in a circumstantial case.

645. I do not agree with Ms Folbigg's submission that the (only) issue is whether any expert could exclude a reasonable alternative natural cause of death. A bare possibility of a natural cause may exist and not be excluded, but it does not prevent a finding of guilt if the inference of guilt is the only reasonable inference open upon consideration of all the facts. What needs to be determined is whether there is an inference consistent with innocence reasonably open, when all of the evidence is considered, including the opinions of medical experts of the likelihood that a particular death was due to a natural cause. Obviously, the stronger a reliable opinion in this regard, the more likely there will be an inference consistent with innocence reasonably open on the evidence.

646. In relation to Professor Cordner's opinion that the lack of facial injuries is negative evidence against smothering it was clearly and generally accepted amongst forensic pathologists that generally, it is very hard to distinguish smothering from SIDS at autopsy.[1152] It is rarely diagnosed. It may well leave no physical signs. I do not accept as relevant Professor Duflou's opinion that on a statistical basis the likelihood of signs of smothering being found increase with the number of homicides.[1153]

Terminology

647. In his 2015 report, Professor Cordner was critical of aspects of the manner in which the trial was conducted. First, he objected to the use of various terms including questions to medical experts as to whether a child had died from an acute catastrophic asphyxiating event and the phrase "consistent with".[1154]

648. Secondly, he was critical of evidence given by Dr Cala as to the circumstances of the deaths.[1155]

Asphyxiation

Professor Cordner's evidence

649. In his report, Professor Cordner described the term "asphyxia" as meaningless as it provides no information as to the cause of the asphyxiating event and forensic pathologists cannot determine whether a person stopped breathing or their heart stopped.[1156] Further, "asphyxia" is not a diagnosis, is not diagnosable and is not understood in a uniform way.[1157]

650. Professor Cordner stated a misconception throughout the trial was that expert forensic pathology can tell whether a person has died because they stopped breathing, as opposed to having died because their heart stopped, or from more complicated mechanisms.[1158] Forensic pathology cannot distinguish between, or identify or diagnose any of them, and cannot point to the pathophysiological mechanisms leading to death, except perhaps where obvious lesion is present.[1159]

1152 Transcript of the Inquiry, 19 March 2019 T111.4-112.5.
1153 See Transcript of the Inquiry, 20 March 2019 T182.5-185.10.
1154 Exhibit Q, Report of Professor Stephen Cordner (undated) pp 57-59.
1155 Exhibit Q, Report of Professor Stephen Cordner (undated) pp 55-57.
1156 Exhibit Q, Report of Professor Stephen Cordner (undated) pp 40, 46.
1157 Exhibit Q, Report of Professor Stephen Cordner (undated) p 6.
1158 Exhibit Q, Report of Professor Stephen Cordner (undated) p 40.
1159 Exhibit Q, Report of Professor Stephen Cordner (undated) pp 40-42.

651. As such, Professor Cordner stated, Dr Cala should have answered questions at trial about whether the children died from an asphyxiating event with "we cannot diagnose that; the pathology findings are also consistent with natural causes".[1160]

652. Professor Cordner's proposition appeared to be that the jury was therefore dealing with a concept central to the trial but which had no clear meaning and thus much of the forensic evidence at trial was misconceived.

653. Professor Cordner reported that he did not find terms such as "acute asphyxiating event" in a search of pathology databases, concluding that these terms are not used by pathologists in formulating the cause of death.[1161] He stated "whether the phrase was intended as a rhetorical flourish or ran risks of creating unjustified alarming prospects in the jury's mind is none of my business".[1162]

654. Professor Hilton stated that he would not entirely dismiss the use of the term "asphyxia".[1163] Further, terms such as "catastrophic asphyxia event" may be appropriate in at least some instances of SIDS, but it is not tenable to equate that term with a non-natural mechanism.[1164]

Counsel assisting's submissions

655. Counsel assisting submitted that there is no identified particular answer given by an expert in evidence that appears to have been non-responsive because of the expert's misconception of the meaning of the term. Nor do any of the experts appear to have demonstrated in his or her evidence, confusion about the meaning of what was being asked. Some examples follow.[1165]

656. Dr Wilkinson replied "absolutely" to a question about whether damage to Patrick's brain after the ALTE was consistent with him having suffered a catastrophic asphyxiating event from unknown causes,[1166] although quite possibly an epileptic seizure could have caused asphyxiation in Patrick's ALTE.[1167] Patrick's death "certainly could have been" consistent with having suffered a recent catastrophic asphyxiating event from an unknown cause, which could have been smothering.[1168] He discussed changes in the brains of children suffering "some asphyxial damage" and loss of visual function following "various asphyxial events".[1169]

657. Dr Singh-Khaira demonstrated no confusion in agreeing that a catastrophic asphyxiating event from some unknown cause could be one of the causes of Patrick's death,[1170] explaining that he was looking for any signs of manual asphyxia such as petechiae and changes in the airways (but found none).[1171] He also thought it possible that a seizure led to a catastrophic asphyxiating event and ultimately to cardiac arrest.[1172]

1160 Exhibit Q, Report of Professor Stephen Cordner (undated) p 42.
1161 Exhibit Q, Report of Professor Stephen Cordner (undated) pp 47-48.
1162 Exhibit Q, Report of Professor Stephen Cordner (undated) p 48.
1163 Exhibit O, Report of Professor John Hilton (22 January 2019) p 2, citing J M N Hilton, 'The Pathology of the Sudden Infant Death Syndrome' in J K Mason (ed), *Paediatric Forensic Medicine and Pathology* (Springer Science, 1989) 156, 161.
1164 Exhibit O, Report of Professor John Hilton (22 January 2019) p 2.
1165 Submissions of counsel assisting (17 May 2019) Chapter 7, [250].
1166 10 April 2003 T509.52-55.
1167 10 April 2003 T511.22-512.15.
1168 10 April 2003 T514.31-49, T516.41-517.5.
1169 10 April 2003 T510.1-18.
1170 11 April 2003 T560.43-48
1171 11 April 2003 T561.37-49.
1172 11 April 2003 T562.40-563.7.

658. Professor Herdson was "quite sure" Caleb died from a sudden catastrophic asphyxiating event of unknown causes and agreed to that proposition as to Sarah's death.[1173] Patrick's ALTE and death were each consistent with such an event (epilepsy could be a cause, but one would expect a history).[1174]

659. Professor Byard's evidence regarding Patrick on this point was to the effect that although it would be very unusual, the death was consistent with a seizure disorder causing a catastrophic asphyxiating event, such disorder resulting from the ALTE, the initial asphyxiating event which itself resulted from Patrick stopping breathing but the cause of which was unknown.[1175]

660. Counsel assisting submitted that there was clearly no misunderstanding at trial as to the use of this term by the expert witnesses. No complaint was made at trial as to the use of this term. Indeed, as is clear from the summary above, that term was used by the expert witness Professor Byard, called by the defence, without any demur.[1176]

661. In the Inquiry, the forensic pathologists were asked about the term asphyxia:

> *WITNESS DUFLOU: Yes I think in the end you probably end up using asphyxia in as meaningless a way as the term cardiac arrest, in that it doesn't provide any information really in terms of what happened.*[1177]
>
> *FURNESS SC: So the issue is why someone was asphyxiated rather than the state of asphyxiation which means you don't have enough oxygen?*
> *WITNESS DUFLOU: Yes, yes on its own it's to me, it's not a term that should be used, at least in the cause of death statement, you can certainly have qualifiers to that term, as an example, positional asphyxia, but on its own I don't think it serves much purpose.*[1178]
>
> *…*
>
> *WITNESS CORDNER: Just to make sure that everybody understands that, if the prosecutor was asking whether there was evidence that a particular medical diagnosis — catastrophic acute asphyxiating event, was present, it's an unanswerable question because asphyxia, as we've said, is meaningless and so it was a question that is empty.*[1179]

662. Counsel assisting submitted that Professor Duflou was particularly concerned that asphyxiation was not used in a cause of death certificate.

663. Professor Cordner stated in his report that "anyone in the street" does not understand the term asphyxia as a low level of oxygen; "most people think" of it as a mechanical interference with respiration or breathing. He also noted that the term "mechanical interference may sound a little strange to the layman".[1180]

664. Counsel assisting submitted that Professor Cordner then properly conceded that that is an assertion on his part and not based on evidence. He stated that forensic pathologists using the term "asphyxiation" and its derivatives in various ways, is a "further source of confusion among lay readers/consumers of forensic pathology".[1181]

1173 1 May 2003 T1035.26, T1038.53.
1174 1 May 2003 T1035.29-1036.11, T1042.47-1043.34.
1175 7 May 2003 T1214.48-1215.19, T1237.57-1238.1, T1238.14-1240.41.
1176 Submissions of counsel assisting (17 May 2019) Chapter 7, [255].
1177 Transcript of the Inquiry, 19 March 2019 T100.35-37.
1178 Transcript of the Inquiry, 19 March 2019 T100.43-49.
1179 Transcript of the Inquiry, 19 March 2019 T102.49-103.3.
1180 Exhibit Q, Report of Professor Stephen Cordner (undated) p 43, fn 43.
1181 Exhibit Q, Report of Professor Stephen Cordner (undated) p 44.

665. He referred to the 2008 Goudge Inquiry report from which he quoted: "asphyxia may be seriously misinterpreted or misunderstood".[1182]

666. Counsel assisting submitted that there was no basis to form the view that the use of those terms by the Crown prosecutor may have confused the jury so as to give rise to an error of process in the trial and further that there was no error in the conduct of the trial by admitting this evidence.

Ms Folbigg's submissions

667. Ms Folbigg submitted that concerns regarding the use of terms such as "asphyxiation" "asphyxiating event" and "catastrophic asphyxiating event" were not in existence at the time of her trial but have since become "accepted constraints" within Australia.[1183] Ms Folbigg relied on Professor Cordner's opinion that these terms have no place in forensic pathology and are likely to confuse, and submitted that I should accept Professor Cordner's views because there was no challenge to his reasoning.[1184]

668. Ms Folbigg annexed to her submissions a table which sets out the references to "asphyxia" or similar at trial and submitted that this table demonstrated:

    a. that the Crown prosecutor used the term as part of leading questions and the witnesses:

        unwittingly accede[d] *to the use of an everyday term which is ambiguous or confusing as a matter of science, assuming that the term [wa]s being correctly or appropriately used;*[1185] and

    b. that with some witnesses the Crown prosecutor sought to clarify the term "asphyxia" to make it clear he was using it as a concept similar to "hypoxia", but with others he did not. It was therefore unclear whether the latter witnesses were using the term with the intention of including or excluding accidental or deliberate suffocation.[1186]

669. Ms Folbigg also submitted that the trial judge erred in his summing-up by saying "it is virtually impossible to distinguish between a death resulting from asphyxiation and a death resulting from natural but unidentified causes" because this imported into the word "asphyxiation" a deliberate act which excluded a natural cause of death.[1187] This error was exacerbated when the Judge recorded the view of some of the experts as being that the events were "consistent with asphyxiation".[1188]

670. Ms Folbigg submitted that the submissions of counsel assisting sometimes made the same error, particularly when referring to Patrick as sustaining "a single hypoxic event or asphyxiating on 18 October 1990."[1189] She concluded that the confusion and ambiguity in the use of these terms contributes to the finding that there is a reasonable doubt as to her guilt.[1190]

1182 Stephen T Goudge, *Report of the Inquiry into Pediatric Forensic Pathology in Ontario* (Ontario Ministry of the Attorney General, 1 October 2008) 433; Exhibit Q, Report of Professor Stephen Cordner (undated) p 57.
1183 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [56].
1184 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [57]-[58].
1185 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [60(a)].
1186 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [60(b)-(c)].
1187 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [63]-[65].
1188 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [64(b)- (c)].
1189 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [67]; Submissions of counsel assisting (17 May 2019) Chapter 7, [75].
1190 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [66].

Findings

671. During the trial a number of the forensic pathologists and other expert witnesses were asked questions about the cause of death of the children and Patrick's ALTE using language of "asphyxiation". A number gave evidence of their understanding of what was meant by "asphyxia", its derivatives, and its combination with adjective phrases. Those explanations consistently either directly describe, or plainly contemplate, the term to mean an event leading to obstruction of airways, some experts going further in their explanation to describe obstruction of air into the lungs and/or impairment of oxygen levels in the blood and/or to the brain.[1191]

672. There is no reason to believe that any expert who used the term did not understand what it meant. There can also be no doubt the jury understood the prosecution case was that the children had all been smothered. The trial judge in his summing up made that plain when dealing with each of the four deaths and the ALTE. He used the word "smother" each time and it appears 37 times in the summing up.

"Consistent with"

673. Professor Cordner took issue with the use of the phrase "consistent with" by forensic pathologists. Professor Pollanen in his peer review also stated that "consistent with" simply means "not inconsistent with", and legal minds and jurors frequently misunderstand "consistent with" to imply corroboration, support or indication, and as a result, the phrase should be avoided.[1192]

Counsel assisting's submissions

674. Counsel assisting submitted that Professor Pollanen's opinion does not of itself indicate that there was in fact misunderstanding of what was meant by the phrase at trial. There is no evidence of any such indication at trial of such misunderstanding.[1193] Finally, what was meant by the phrase was explained by defence counsel in his closing to the jury, and by the trial judge in his summing-up.

675. In his closing address, defence counsel explained that the phrase "consistent with suffocation" is not proof of suffocation and may also mean consistent with a natural process.[1194] He emphasised the importance, when the phrase is used:

> *to say, hang on a moment, consistent with suffocation means that because a person or a child could be suffocated without there being any symptoms that consequently if there are no symptoms that that would be consistent with suffocation.*[1195]

He said, when experts say "consistent with" suffocation, they are not saying there is positive proof of suffocation.[1196]

1191 9 April 2003 T449.57-450.3; 10 April 2003 T511.32-44, T514.26-515.40; 14 April 2003 T619.14-22, T651.17-52; 23 April 2003 T876.17-25; 5 May 2003 T1139.30-34.

1192 Exhibit C, Report of Professor Michael Pollanen (1 June 2015) p 2.

1193 Submissions of counsel assisting (17 May 2019) Chapter 7, [263].

1194 14 May 2003 T1389.45-50.

1195 14 May 2003 T1389.36-41.

1196 14 May 2003 T1389.36-41.

676. Defence counsel noted Professor Herdson's evidence that Caleb's death was "consistent with" deliberate suffocation, but that meant there were no symptoms of suffocation because suffocation can occur with no symptoms.[1197] Also, regarding Professor Hilton's diagnosis of Sarah's death as being "consistent with" SIDS, counsel said "we can read into that that there were no other findings which permitted him to reach any other conclusion" (that is, of smothering).[1198] Evidence remained that there was no injury to Sarah and no medical proof of suffocation. It was necessary to distinguish medical proof of suffocation from the phrase "consistent with suffocation".

677. In summing-up, the trial judge explained that if a condition is not specific for a cause, this:

> *simply means that the proper medical conclusion to draw is that the postulated cause could have been the cause for the condition, but not that it must have been, or very likely or probably was, so that an opinion that a condition is consistent with a particular cause implies that it might also be consistent with another cause or causes.*[1199]

678. The question for the Inquiry is whether the use of those terms by the Crown prosecutor may have confused the jury so as to give rise to an error of process in the trial.

679. Counsel assisting submitted that Professor Cordner and Professor Pollanen are merely speculating as to the effect of those terms and that there is no basis for me to find that any error in the conduct of the trial arose from experts being questioned using language of asphyxiation and consistency. In addition, the summing-up and the defence submissions adequately addressed the use of the terminology.

Ms Folbigg's submissions

680. In her submissions, Ms Folbigg pointed to Professor Cordner's report,[1200] Professor David Ranson's report (a forensic pathologist at VIFM asked to view images of Laura's slides)[1201] and the Goudge Inquiry[1202] as support for the proposition that the term "consistent with" can be misleading.

681. She submitted that I should reject counsel assisting's submissions because it does not matter whether the experts at trial were confused: the relevant question is whether the use of the word would mislead the jury.[1203]

Findings

682. I fully agree with the submissions of counsel assisting on this point. The term "consistent with" is one that a jury should be able readily to understand.

1197 14 May 2003 T1412.23-29.

1198 15 May 2003 T1494.24-26.

1199 19 May 2003 T26.

1200 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [70]; Exhibit Q, Report of Professor Stephen Cordner (undated) p 58

1201 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [73]; Exhibit AM, Report of Dr David Ranson (31 December 2018) pp 7-12.

1202 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [72]; Stephen T Goudge, *Report of the Inquiry into Pediatric Forensic Pathology in Ontario* (Ontario Ministry of the AttorneyGeneral, 1 October 2008).

1203 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [68], [71].

683. If there was any doubt about this, the trial judge clearly explained the use of the phrases "consistent with" and "specific for" in forensic pathology or medical expert evidence. He said:

> *Just a word about this terminology. You have heard a number of experts observe that in such matters one cannot be certain in a diagnosis. When a particular condition is established on a post-mortem examination to exist, the pathologist will be able to say whether that condition could have come about from a particular cause. There the pathologist is speaking of a mere possibility. That seems to be the same as saying that the sign or condition found is consistent with having been caused in the manner postulated. The expression often used, that the condition is not specific for that cause, simply means that the proper medical conclusion to draw is that the postulated cause could have been the cause of the condition, but not that it must have been, or very likely or probably was, so that an opinion that a condition is consistent with a particular cause implies that it might also be consistent with another cause or causes.*[1204]

684. In my opinion, there was nothing misleading about the use of the phrase "consistent with" nor, indeed, "specific for".

"Rare"

Ms Folbigg's submissions

685. Also raised in Ms Folbigg's submissions was her concern about the use of the term "rare".

686. Ms Folbigg says that this term was used in many different contexts at trial, and that as both smothering and sudden infant death are "rare", little weight can be placed on the rarity of one as opposed to the other.[1205] She also criticised the submissions of counsel assisting as having "no balance to the use of the word" and making references to rarity to "unfairly… reduce the likelihood of the occurrence of a potential cause of early death which falls in favour of Ms Folbigg."[1206]

Findings

687. The word "rare" was used by Professor Cordner to describe both unexplained natural deaths and homicides.[1207] Similarly, Professor Pollanen used it to describe serial natural deaths and serial homicidal deaths in infancy and childhood.[1208] Professor Duflou used it to describe repeat SIDS deaths.[1209]

688. Not in response to any leading question, Professor Horne discussed in oral evidence that more than one SIDS in a family is very rare, and SIDS itself is rare.[1210] She was asked further about this, because language can be important in terms of degree, and again in response to a non-leading question said that the risk of recurrence was "rare and very rare maybe".[1211] Category SIDS 1A is very rare.[1212]

1204 19 May 2003 T26-27.
1205 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [75]-[81].
1206 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [82].
1207 Exhibit Q, Report of Professor Stephen Cordner (undated) p 90.
1208 Exhibit C, Report of Professor Michael Pollanen (1 June 2015) p 1.
1209 Exhibit L, Report of Professor Johan Duflou (13 February 2019) p 45.
1210 Transcript of the Inquiry, 18 March 2019 T32.27-32.
1211 Transcript of the Inquiry, 18 March 2019 T33.31-40.
1212 Transcript of the Inquiry, 18 March 2019 T49.17-26.

689. The word was used by Wood CJ at CL in his summary of the circumstantial evidence – "the rarity of repeat incidence of SIDS and of unexplained infant deaths or ALTEs".[1213] The trial judge directed the jury that SIDS deaths are rare in the community, and the lack of authenticated record of three or more in a single family illustrated the rarity.[1214]

690. Professor Byard gave evidence at trial using the word "rare" to describe the incidence of child deaths from myocarditis.[1215] Associate Professor Fahey wrote that SUDEP is rare in his report.[1216]

691. It is true that the word "rare" has been used in a number of different contexts by a number of different expert witnesses to describe the incidence of different conditions and phenomena, by experts who prepared reports both at the request of the Inquiry and at the request of Ms Folbigg's representatives. On a number of occasions they were pressed in oral evidence to give thought to the words they were using. I reject Ms Folbigg's submissions: the repeated use of the word or similar expressions to describe particular conditions and circumstances by well-qualified experts rather emphasised the extraordinary nature of the collocation of events in the Folbigg family.

1213 *R v Folbigg* [2002] NSWSC 1127, [107].

1214 19 May 2003 T24-25.

1215 7 May 2003 T1246.10-22.

1216 Exhibit AK, Report of Professor Michael Fahey (30 March 2019) p 14.

Report of the Inquiry into the convictions of Kathleen Megan Folbigg

July 2019

Volume 1: Chapter 6

Chapter 6: Immunology

Introduction

1. This chapter considers evidence relevant to immunology, microbiology and infection.

2. First, the role of infection in SIDS deaths generally is examined. I then consider the findings on autopsy in this case, and whether infection may have played a role in any of the deaths of the children.

The role of infection in SIDS deaths

Research

3. Professor Caroline Blackwell is a con-joint Professor in Immunology and Microbiology at the School of Health, University of Newcastle and has a PhD in Medical Microbiology. She is a researcher and has no clinical qualifications. The Inquiry met with Professor Blackwell in November 2018 to request her assistance. She did not have the capacity to assist and recommended that the Inquiry contact Professor William Rawlinson AM to inquire as to the possibility of conducting microbiological testing on samples from one or more of the children.[1] She gave oral evidence in the Inquiry and four statements she had prepared were in evidence.

4. Professor Blackwell commented on a number of studies that she said provided a "growing body of evidence that infection plays a role in these infant deaths".[2] For example, in Duncan and Byard (2018), Professor Siri Hauge Opdal stated:

> *Both experimental and observational studies provide evidence indicating that infection and inflammation might play a role in sudden infant death syndrome (SIDS)…*
>
> *There are also several studies indicating that virus infections may play a role in SIDS, and higher rates of viruses have been isolated in samples from SIDS compared to controls. The involvement of viruses may be direct, by induction of a cytokine storm upon viral infection, or indirect, through synergistic interactions with bacterial virulence factors and/or immunoregulatory polymorphisms. However, so far, no single respiratory virus has been exclusively found in a high proportion of SIDS cases: rather, a range of viruses are found at a higher frequency in SIDS compared to controls.*[3]

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1 Professor Rawlinson is a Senior Medical Virologist and Director of Virology at South Eastern Sydney and Illawarra Health Service. The Inquiry met with Professor Rawlinson in November 2018 and provided him with a list of the samples held in respect of each of the children by NSW Health. Professor Rawlinson prepared a short written statement for the Inquiry in which he confirmed his opinion that testing of the available samples for infectious pathogens, including viruses, could not be conducted in a way so as to be useful in determining cause of death in this case. His statement was tendered before the Inquiry and he was not called to give evidence.

2 Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) p 5.

3 Siri Hauge Opdal, 'Cytokines, Infection and Immunity' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 689, 689-690 (citations omitted).

5. In that chapter of Duncan and Byard (2018), Professor Opdal depicted diagrammatically the role that the immune system may play in the risk factors in an adaptation of the "fatal triangle", itself proposed by Rognum and Saugstad in 1993:[4]

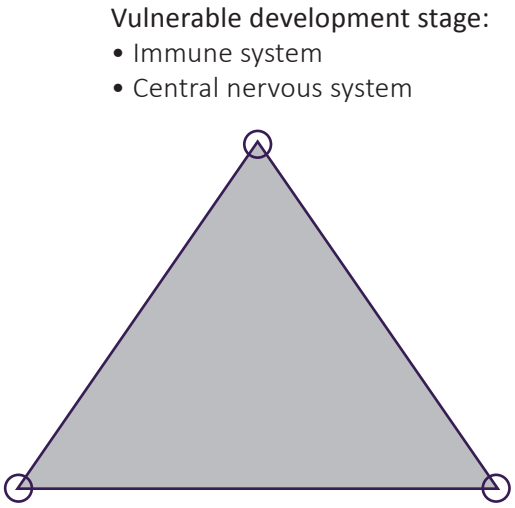

Figure 1: "Fatal triangle" adapted by Professor Opdal to represent risk factors in SIDS 2018 (Duncan and Byard), p 701

6. Professor Opdal concluded:

> *Finally, death in SIDS cases may be due to more than one mechanism. It is, however, likely that a dysregulation of inflammatory responses to apparently mild infections is involved in a proportion of SIDS. Genetic variations in cytokine genes are most likely involved, as they contribute to differences in the expression, translation, cellular transport, and secretion of the cytokine. However, it is important to interpret cytokine SNP data with caution and to consider the effects of other genetic, developmental, and environmental influences on the responses.*[5]

7. Consistently with this passage, Professor Blackwell gave evidence that in any of the Folbigg children's deaths, there is no single cause. It is a multifactorial series of events.[6] She further gave evidence about the way that minor infections could trigger death in children aged two to four months:

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4 Siri Hauge Opdal, 'Cytokines, Infection and Immunity' in Jhodie R Duncan and Roger W Byard (eds), SIDS – *Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 689, 701, adapted from T O Rognum and O D Saugstad, 'Biochemical and Immunological Studies in SIDS Victims. Clues to Understanding the Death Mechanism' (1993) 389 *Acta Paediatrica* 82.

5 Siri Hauge Opdal, 'Cytokines, Infection and Immunity' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 689, 703.

6 Transcript of the Inquiry, 22 March 2019 T335.46.

> *[They] have the lowest level of immunoglobulins that would be protective against infection, the material they received from their mother before birth has waned probably to the lowest, and they will have the lowest level of protective antibodies that they will ever have in their lives. If an infection or infective organism gets into the body they're going to be dependent on the non-specific immune system, the white cells, to go in and deal with this, to kill the organism, to mop up the pieces and these will then be turned into antibodies, the white cells then produce antibodies against the organisms that they've dealt with.*
>
> *A minor infection, say a large number of organisms get in, might trigger a very massive inflammatory response, it might not be a major pathogen like meningococcus, it could be a minor pathogen like Staphylococcus aureus or Escherichia coli, so the damage is done not by the organism itself but by the body's response to the organism; it's very powerful.*[7]

8. Researchers do not know what actually causes the death in SIDS, but some researchers propose "different mechanisms by which the physiology of the child could be disrupted and… inflammatory responses to infection can affect all of these."[8]

9. Professor Robert Clancy AM is a mucosal immunologist and Foundation Professor of Pathology at the University of Newcastle. Professor Clancy's field of specialised knowledge, mucosal immunology, concerns immune system responses that occur at mucosal membranes of the intestines, the urogenital tract and the respiratory system, i.e. surfaces that are in contact with the external environment.

10. Professor Clancy was engaged by those representing Ms Folbigg to prepare a report on mucosal immunology. He was provided with reports by Professors Duflou, Horne, Hutchinson and Dr Drucker (in relation to the IL-10 gene tested for in the children at the time of the trial), the autopsy reports, and Professor Blackwell's report dated 9 March 2019.[9]

11. Professor Clancy also gave his opinion to the Inquiry that there is a causal connection between mild infection and SIDS:

> *In my opinion, current evidence would have as a primary cause in half of the population of sudden death infants a mild intercurrent airways infection at a critical time of immaturity of the local mucosal immune response leading to an inappropriate excessive immune response - leaving the airways parsed and unable to clear bacteria that descend all the time from the upper airways.*[10]

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7 Transcript of the Inquiry, 22 March 2019 T321.34-48.

8 Transcript of the Inquiry, 22 March 2019 T322.50-333.4.

9 Exhibit W, Report of Professor Robert Clancy (13 March 2019) and Further report of Professor Robert Clancy (17 March 2019) Letter of instruction.

10 Exhibit AT, Further report of Professor Robert Clancy (27 March 2019) p 15 (emphasis in original).

12. Professor Paul Goldwater is a specialist in infectious diseases and a specialist clinical microbiologist. He was engaged by those representing Ms Folbigg to provide a peer review of the opinions offered by Professor Blackwell and Professor Clancy.[11] He was provided only with those reports and their annexures, together with transcripts of the oral evidence given by Professor Blackwell and Professor Clancy in the Inquiry on 22 March 2019.[12] He was not provided with any of the reports by, or evidence of, the forensic pathologists.[13] He did not give oral evidence. He expressed similar opinions to Professors Blackwater and Clancy in relation to the role of infection in SIDS deaths.

13. Each of the forensic pathologists and Professor Elder (paediatrician) and Professor Horne (SIDS researcher) gave evidence before the Inquiry about infection in association with the sudden death of infants, as well as the role it may have played in the deaths of the four Folbigg children. It is fair to say that none were persuaded that there was clear evidence of a connection between the deaths of any of the children and an infection.

14. Professor Elder said:

> *We have to always remember that risk factors are risk factors, they're not – they won't always cause death, so some – many babies have slept prone and not died, many babies have been bottle fed, such as myself, and not died. It's – the model is about things that might work together, and for all risk factors, as a clinician, faced with a baby who's died, I still need to be able to process some mechanism by which that risk factor might have resulted in the death of a child. Now, there is some of these factors when they work together – I certainly feel that there is a plausible evidence base, as I discussed earlier, the in utero exposure to smoking affects serotonin supply in the brainstem, so that when you are faced with an asphyxial insult you can't respond and gasp and self-resuscitate. That's reasonably well-documented.*
>
> *For all the other risks, such as exposure to infection, there are some theories about how that might cause death, as has been discussed, through a toxin effect on the heart rate, but all, all these things you have, you have to kind of go to the end point to truly understand how the infants died.*[14]

15. Professor Elder also gave evidence that there have been theories about the role of infection for a long time between a recent not apparently very severe infection and infant death.[15] The issue, however, remained, in her opinion, to explain how that can cause the death of four children "in a row".[16]

16. Professor Horne told the Inquiry, and Professor Elder agreed, that a mild respiratory infection is common and half of babies who die have had a mild respiratory infection not severe enough to be attributed to the cause of death.[17]

17. In relation to a statement by Professor Blackwell that infectious agents identified in SIDS/SUDI can elicit inflammatory responses, Professor Cordner described a gap between such research and practice.[18]

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11 Exhibit AU, Report of Professor Paul Goldwater (29 March 2019). I record that counsel assisting submitted there should be redactions to Professor Goldwater's report on the basis of opinions given outside Professor Goldwater's area of expertise. Ms Folbigg's representatives agreed to only some of the redactions in the report as tendered.

12 Exhibit AU, Report of Professor Paul Goldwater (29 March 2019) p 2.

13 Exhibit AU, Report of Professor Paul Goldwater (29 March 2019) p 2.

14 Transcript of the Inquiry, 18 March 2019 T52.16-32.

15 Transcript of the Inquiry, 18 March 2019 T48.23-32.

16 Transcript of the Inquiry, 18 March 2019 T48.30.

17 Transcript of the Inquiry, 18 March 2019 T38.13-25.

18 Transcript of the Inquiry, 19 March 2019 T135.13-16.

18. Professor Hilton said there has been a suspicion that there had been immunological-type problems associated with SIDS for decades – he described both Professors Clancy and Blackwell's research on immune bodies in lung exudates as an "interesting research technique" which may or may not have technical application.[19]

19. Professor Hilton said a slight infection may be associated with sudden infant death, which is very much a work in progress, and it's a concept which is gathering scientific validation.[20] He agreed this was only in its very nascent stages in 2003.[21]

20. All of the forensic pathologists agreed that since then, the science about the link between infection and the cytokine[22] response continues to be consolidated (Dr Cala saying "it appears to").[23] However, Professor Hilton did not agree that there is a strong link between *Staphylococcus aureus* and staphylococcal endotoxins triggering sudden infant death.[24] Forensic testing of tissue to identify immune reaction would tend to support the view that a particular organism detected at autopsy was an active bacterium rather than contamination.[25] This was not widely available in 2003 and did not form part of forensic pathology practice.[26]

21. Professor Hilton said that "germs are irritants that can elicit inflammatory responses".[27] On occasion, bugs detected in the lungs of post-mortem specimens taken from dead babies raise questions – very often, a pathologist cannot tell if they are real or a contaminant.[28] Professor Hilton described this as an interesting theory relating to factors which may be involved in the death of a child, at the research stage.[29]

22. Dr Cala and Professor Duflou agreed.[30] Professor Duflou added that forensic pathologists generally view organisms in lungs as clinically relevant if there is discernible inflammation under the microscope, and it would probably not change his view about whether or not the death was SIDS.[31] There has also been no broad acceptance by the forensic medical community about Professor Clancy's statement that:

> *[t]here is in SIDS and near miss SIDS an exaggerated secretion of immunoglobulins (proteins) into mucosal secretions. Thus, the finding of 'eosinophillic exudate' heightens diagnostic confidence of SIDS.*[32]

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19 Transcript of the Inquiry, 20 March 2019 T236.47-237.10.
20 Transcript of the Inquiry, 21 March 2019 T273.1-4.
21 Transcript of the Inquiry, 21 March 2019 T273.18-21.
22 The term "cytokine" defines a large group of small non-structural proteins that are involved in cell signals, Siri Hauge Opdal, 'Cytokines, Infection and Immunity' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 689, 690.
23 Transcript of the Inquiry, 21 March 2019 T273.27-39.
24 Transcript of the Inquiry, 21 March 2019 T273.50-274.6.
25 Transcript of the Inquiry, 21 March 2019 T274.22-23.
26 Transcript of the Inquiry, 21 March 2019 T274.28-275.10.
27 Transcript of the Inquiry, 19 March 2019 T136.13.
28 Transcript of the Inquiry, 19 March 2019 T136.14-17.
29 Transcript of the Inquiry, 19 March 2019 T136.37-46.
30 Transcript of the Inquiry, 19 March 2019 T137.9-15.
31 Transcript of the Inquiry, 19 March 2019 T137.16-38.
32 Transcript of the Inquiry, 19 March 2019 T138.9-139.9, referring to Exhibit W, Report of Professor Robert Clancy (13 March 2019) p 2.

Genetics and immunological responses

IL-10 gene

23. In her second report of 27 March 2003, Dr Ophoven (paediatric forensic pathologist) was asked to give an opinion in light of it having been discovered by microbiologist Dr Drucker that Sarah was homozygous for IL-10, the so-called "cot death gene" (meaning she had identical mutations of both her maternal and paternal alleles[33]).[34]

24. Dr Ophoven stated that there is no "SIDS gene" – the IL-10 gene is associated with immunity, specifically a type of inflammatory molecule called a cytokine (in this case, interleukin).[35] Publications in literature about the IL-10-572 allele (except for an article referenced by Dr Drucker) all related to the reaction of the human body to immune stressors or inflammatory disease; there was no consensus or reported discussion that this molecular finding was linked genetically to SIDS.[36]

25. The theory, that there was a genetic basis for SIDS linked to variation in the interleukin cytokine production in the immune response, was not verified nor accepted by the medical community and Dr Drucker's paper was the only paper suggesting a connection between IL-10 and SIDS.[37] His conclusions were "pure speculation"; the presence and profiling of the genome required a much wider population analysis.[38]

26. Professor Berry's second report dated 29 April 2003 also concerned Dr Drucker's finding that Sarah was homozygous for the gene IL-10. Professor Berry observed that SIDS research is "littered" with abandoned theories; most researchers did not accept new findings until they were independently confirmed.[39] Placing babies to sleep in the prone position, for example, had been confirmed by more than a dozen separate studies.[40] SIDS studies involving statistics had common problems of small numbers of cases, case selection, and inappropriateness of controls.[41] The IL-10 study was essentially a statistical study.[42]

27. Professor Berry considered that the size of the study (23 SIDS cases) could not be regarded as anything more than an "interesting preliminary study"; it was not possible from the paper alone to be confident that the control group was free from selection bias. Even assuming no selection bias, the gene conferred about a threefold increased risk, a relatively weak association. There was no data to support the theory of association.[43] IL-10-592*A was a possible association only and could not be invoked as a cause of SIDS.[44]

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33 There are usually two copies of a gene. These two copies are called alleles. In some cases, one or both alleles will be mutated or altered in some way, NSW Health, 'Glossary', *Centre for Genetics Education* (Web Page,26 February 2016) <https://www.genetics.edu.au/publications-and-resources/glossary#A>.

34 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (27 March 2003) p 1.

35 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (27 March 2003) p 2.

36 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (27 March 2003) pp 2-3.

37 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (27 March 2003) p 3.

38 Exhibit H, Forensic pathology tender bundle, Report of Dr Janice Ophoven (27 March 2003) p 3; David Drucker et al 'Association of IL-10 Genotype with Sudden Infant Death Syndrome' (2000) 61 *Human Immunology* 1270.

39 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (29 April 2003) pp 1-2.

40 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (29 April 2003) pp 1-2.

41 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (29 April 2003) p 2.

42 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (29 April 2003) p 2.

43 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (29 April 2003) pp 2-4.

44 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (29 April 2003) pp 3-4.

28. Dr Drucker had found Sarah had two copies of the gene, and so was "at higher risk than even a baby with one copy would have been."[45] However Professor Berry concluded that because the threefold increase was an "average", it was not possible to say that the risk for babies with two copies of the gene variant was greater than that for babies with one copy of it.[46]

Genetic testing conducted by the Inquiry

29. As discussed later in this report, the genetic testing undertaken by the Inquiry identified no known pathogenic or likely pathogenic genetic variant which could have caused the children's deaths, including in genes associated with immunological responses.

30. In considering the link specifically between genetics, infection and cardiac events possibly causing death, Professor Jonathan Skinner, paediatric cardiologist and electrophysiologist, gave evidence that:

> *Infants, as we heard earlier on, repeatedly have upper respiratory tract infections. It's a normal and repeated phenomenon and it wouldn't surprise you to find that a child that had died with one had an infection, if it's routine, to get about eight infections a year, then we're bound to find some of that, yes. And I guess one of the questions that logically would arise from that is did the virus somehow trigger some sort of cardiac event? In our field we've been looking for that, that evidence, and the only evidence really to date that we've found is related to the cardiac sodium channel gene I referred to earlier and it's linked to Brugada syndrome and the fever. However, that tends to really be older children, but I am quite sure that that could happen in the infant as well, high fever and triggering a cardiac event in somebody with Brugada syndrome.*[47]

31. Professor Skinner clarified that he was referring to SCN5A variants as providing a trigger for an event in somebody who is genetically predisposed.[48] As discussed further in Chapter 7 of this report, none of those variants were found in the Folbigg family.

32. Dr Michael Buckley, genetic pathologist, agreed with Professor Skinner:

> *[P]eople with SCN5A pathogenic variants are susceptible to cardiac dysfunction when they have a high, a high temperature. I'm not sure that that has any relevance to the family that we are looking at here because none of the children, as far as either group have been able to define, do have those variants.*[49]

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45 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (29 April 2003) p 1.
46 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (29 April 2003) p 3.
47 Transcript of the Inquiry, 16 April 2019 T526.49-527.10.
48 Transcript of the Inquiry, 16 April 2019 T533.31-32.
49 Transcript of the Inquiry, 16 April 2019 T533.46-534.1.

Findings on autopsy

33. Professor Blackwell drew particular attention to the microbiological findings on autopsy of each of the children except for Caleb, set out above in Chapter 5. She sought to draw two conclusions from these findings.[50] First, that they represent an increased relative risk for SIDS of 29,[51] and second that they are indicative of the children having an infection.[52]

Contamination

34. Professor Blackwell also suggested the microbiological findings at autopsy were indicative of the children having an infection and were not post-mortem contaminants. The evidence before the Inquiry on this issue is as follows.

Patrick

35. Patrick's "post mortem blood cultures grew mixed cocci and bacilli identified as E.coli, Enterococcus faecolis and Enterococcus avium."[53] It was recorded by the forensic pathologist who conducted the autopsy that "these findings are not significant and probably reflect contamination."[54]

36. Dr Drucker in his report queried whether the organisms found in relation to Patrick arose after death by contamination or before death.[55] He noted that one of the major species he considered to be associated with SIDS was present (*E coli*), but that the other species found were not characteristic of SIDS but of the gut flora.[56] He concluded there was "little evidence of SIDS associated bacteria" and noted also other experts' views regarding encephalitis as the likely explanation for death rather than SIDS.[57]

37. In her March 2019 report, Professor Blackwell opined that as the post-mortem examination was carried out two hours after Patrick's death, "it is difficult to dismiss the findings as contamination as there would have been little time for breakdown of mucosal barriers".[58]

38. Professors Cordner, Hilton and Duflou gave evidence in the Inquiry that given the autopsy was started very soon after death, it was notable that the post-mortem blood cultures showed such a rich yield of bacteria.[59] However, all of the forensic pathologists accepted that the cultures probably reflected contamination.[60]

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50 Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure A.

51 Relative risk is the ratio of probability of an event in an exposed group to the probability of an event in a non-exposed group: Miquel Porta (ed), *Dictionary of Epidemiology* (6th ed, 2014) 'Relative risk'.

52 Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) pp 8-9.

53 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Patrick (2 September 1991) p 2.

54 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Patrick (2 September 1991) p 2.

55 Exhibit BM, Report of David Drucker (18 February 2003) p 6.

56 Exhibit BM, Report of David Drucker (18 February 2003) p 6.

57 Exhibit BM, Report of David Drucker (18 February 2003) pp 6, 8.

58 Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure A, pp 9-10.

59 Transcript of the Inquiry, 20 March 2019 T153.33-35 (Professor Cordner), T153.48-49 (Professor Duflou),T154.9-15 (Professor Hilton).

60 Transcript of the Inquiry, 20 March 2019 T153.44 (Professor Cordner) T153.50-154.1 (Professor Duflou), T154.5 (Dr Cala), T154.20-21 (Professor Hilton).

Sarah

39. In relation to Sarah, the following histology was reported:

    a. lung: profuse coliform; profuse streptococcus, alpha haemolytic; scanty *Staphylococcus aureus*; and

    b. spleen: moderate coliforms of 3 colonial types.[61]

40. In addition, it was noted that "one section of larynx shows a light mixed lymphocytic inflammatory infiltrate deep to the respiratory epithelium" and "in one section there is a light interstitial acute inflammatory infiltrate which could be seen around the occasional bronchiole".[62]

41. In his report, Dr Drucker noted that in Sarah's autopsy the presence of coliforms together with *S aureus* was:

> *interesting because both have been associated with SIDS and together their toxins act synergistically having a far greater effect than separate toxins would".*[63] *He concluded "species associated with SIDS present and after an URTI. It is entirely possible that Sarah died as a SIDS case.*[64]

42. He recommended more detailed microbiology interpretation.[65]

43. The only expert at trial who gave evidence in relation to contamination was Professor Hilton. He said that the findings of staphylococcus in Sarah's samples were of no significance whatsoever because it is a post-mortem contaminant, regularly found postmortem.[66] In relation to streptococcus he said it:

> *might indicate that at or about or prior to the death there was a genuine streptococcul* [sic] *infection present in the throat or in the respiratory tract, most probably within the throat, and this again would help to explain the reddening of the uvula and perhaps the inflammation, the light inflammation in the larynx.*[67]

44. Professor Hilton did not make a diagnosis of a lower respiratory tract infection at autopsy in respect of Sarah and concluded at trial that he "would normally not have expected this degree of inflammation to have contributed significantly to this child's death."[68]

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61 Exhibit H, Forensic pathology tender bundle, Microbiology reports of Sarah (13 September 2019, 21 September 2019), tabs 33A and 33B.
62 Exhibit H, Forensic pathology tender bundle, Final Autopsy report of Sarah (25 November 1993) p 100.
63 Exhibit BM, Report of Dr David Drucker (18 February 2003) p 7.
64 Exhibit BM, Report of Dr David Drucker (18 February 2003) p 8.
65 Exhibit BM, Report of Dr David Drucker (18 February 2003) p 1.
66 14 April 2003 T628.2-10.
67 14 April 2003 T628.18-24.
68 14 April 2003 T628.33-35.

45. In the Inquiry Professor Hilton confirmed that his views regarding Sarah's histology reports were the same as for Patrick.[69] He also said:

> *[A]t the time of her death she had some signs of, minor signs of respiratory tract infection in the lung, now these would not occur, these signs would not occur as a post mortem artefact because it's a cellular, infiltrate cellular exudate, that ain't going to happen post mortem to any extent. Basing it then on the presence of post mortem cultures I think would be a leap of faith which I would not be prepared to take.*[70]

46. Dr Cala opined that they were contaminants and that he "wouldn't put any weight on those results."[71] Professor Duflou said his comments from the discussion about Patrick also applied to Sarah, and noted that there was a prolonged interval between the taking of the lung specimens and the specimens arriving in the laboratory, which raised contamination as a "greater possibility".[72]

47. Professor Clancy gave evidence that there was "strong data" that they were not contaminants in Sarah's lungs, however there was a stronger argument that it could be contamination in the cultures in the spleen.[73]

48. Professor Goldwater said in his report that the findings in Sarah's lung, "on the balance of probability, would have played a role in her death".[74]

49. In her March 2019 report Professor Blackwell said that:

> *[c]oliform bacteria are not part of the normal flora of the respiratory tract of healthy adults or infants and are unlikely to have been lung contaminants acquired through resuscitation efforts.*[75]

Laura

50. In relation to Laura, the following histology was reported:

    a. lung: profuse post-mortem contaminants; profuse coliform; [76]

    b. spleen: moderate coliforms of 2 colonial types; profuse alpha haemolytic; and streptococcus of 2 colonial types; moderate *Staphylococcus aureus*.[77]

51. Dr Drucker considered the dismissal of the coliforms in Laura's lungs as post-mortem contaminants and the presence of coliforms in her spleen as "interesting".[78] He also considered that the *S aureus* present in Laura's spleen did not cause a major infection.[79] He concluded "some evidence of SIDS associated bacteria" and recommended more detailed microbiology interpretation.[80]

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69 Transcript of the Inquiry, 20 March 2019 T186.13-15.
70 Transcript of the Inquiry, 20 March 2019 T190.16-21.
71 Transcript of the Inquiry, 20 March 2019 T186.42-43.
72 Transcript of the Inquiry, 20 March 2019 T186.47-187.10.
73 Transcript of the Inquiry, 22 March 2019 T351.19-22, T351.29-35.
74 Exhibit AU, Report of Professor Paul Goldwater (29 March 2019) p 5.
75 Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) p 5.
76 Exhibit H, Forensic pathology tender bundle, Microbiology report of Laura (9 March 1999) tab 47C.
77 Exhibit H, Forensic pathology tender bundle, Microbiology report of Laura (9 March 1999) tab 47D.
78 Exhibit BM, Report of Dr David Drucker (18 February 2003) p 6.
79 Exhibit BM, Report of Dr David Drucker (18 February 2003) p 8.
80 Exhibit BM, Report of Dr David Drucker (18 February 2003) p 1.

52. In her 2004 report, Professor Blackwell opined that the bacteriological findings in relation to Laura were not postmortem contamination.[81]

53. In the Inquiry, Professor Duflou, with whom the other forensic pathologists agreed, considered the orthodox view in relation to the microbiology concerning Laura to be likely contamination in the main.[82]

54. Professor Clancy opined that the coliforms found in Laura were very different from Sarah and that post-mortem contamination was likely to account for them.[83]

55. Professor Goldwater opined:

> *Laura probably died as a result of myocarditis, but Staphylococcus aureus was isolated from her spleen; this could have played a role in her death as could the coliforms isolated from her lungs.*[84]

Reasons for findings

56. While she tempered her view in her oral evidence in the Inquiry by acknowledging that contaminants is a contentious area,[85] Professor Blackwell referred to a number of studies to support her opinion that the organisms found in Patrick, Sarah and Laura were not postmortem contaminants.[86]

57. Professor Blackwell relied, in part, on a 2008 article by Weber et al which reported on a review of autopsies done at one specialist centre between 1996 and 2005. The authors interpreted the results of the case review thus:

> *Although many post-mortem bacteriological cultures in SUDI yield organisms, most seem to be unrelated to the cause of death. The high rate of detection of group 2 pathogens, particularly S aureus and E coli, in otherwise unexplained cases of SUDI suggests that these bacteria could be associated with this condition.*[87]

58. In a related 2010 publication Weber et al noted in summary that the contribution of contaminants remains controversial.[88]

59. Professor Blackwell was taken to a 2006 article by Morris, Harrison and Partridge in which it was noted that:

> *A pure growth of a pathogen in a blood or cerebrospinal fluid should be regarded as a possible contributing factor to death at all ages, but corroborative evidence should be sought using a range of techniques.*[89]

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81 Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure A, p 6.

82 Transcript of the Inquiry 20 March 2019 T218.43-45 (Professor Duflou), T219.3 (Dr Cala), T219.12-16 (Professor Hilton), T219.28-29 (Professor Cordner).

83 Exhibit W, Further report of Professor Robert Clancy (17 March 2019) p 3.

84 Exhibit AU, Report of Professor Paul Goldwater (29 March 2019) p 5.

85 Transcript of the Inquiry, 22 March 2019 T318.28.

86 Transcript of the Inquiry, 22 March 2019 T318.28-319.28.

87 M A Weber et al, Infection and Sudden Unexpected Death in Infancy: A Systematic Retrospective Case Review (2008) 371 Lancet 1848, 1848.

88 M A Weber et al, 'Postmortem Interval and Bacteriological Culture Yield in Sudden Unexpected Death in Infancy (SUDI)' (2010) 198 *Forensic Science International* 121, 125.

89 Transcript of the Inquiry, 22 March 2019 T348.11-14; J A Morris, L M Harrison and S M Partridge, 'Postmortem Bacteriology: A Re-Evaluation' (2006) 59 *Journal of Clinical Pathology* 1, 8.

60. Professor Blackwell accepted that that was a "valid point".[90]

61. When asked whether the Inquiry should prefer the studies to which she referred to the evidence of four forensic pathologists, two of whom had conducted the autopsies, she said "I would not say 'prefer' I would say 'consider'."[91] Asked what the finding should be, following that consideration, Professor Blackwell gave evidence that the microorganisms "probably contribute to a proportion of those deaths."[92]

62. While Professor Blackwell gave evidence in the Inquiry after the forensic pathologists, her reports were drawn to their attention prior to them giving their evidence. When Professor Blackwell's views on contamination were put to the forensic pathologists, Professor Duflou observed that Professor Blackwell is an expert in microbiology, he is an expert in autopsies.[93] The forensic pathologists did not change their opinions that the histology results probably reflected contamination in respect of Patrick, Sarah and Laura, although Professor Cordner noted in respect of Sarah that "you've got to keep open the possibility that there is something there, I personally am not dismissing that."[94]

Increased ratio

63. In her March 2019 report, Professor Blackwell referred to a 1992 publication by Gilbert et al to support the proposition that a finding of coliforms in an infant confirmed an increased relative risk for SIDS of 29.[95]

64. Professor Blackwell was taken to that article in her oral evidence.[96]

65. It was put to Professor Blackwell that the odds ratio of 29 in the article upon which she relied was based on coliforms found in places other than where they were found in Sarah and Laura.[97] According to the article, most of the 37 subjects were found with coliforms in their tracheal aspirate, 14 had them in their lungs (as was the case in Sarah and Laura) and only three in their spleen (as was found in Sarah and Laura).[98] Professor Blackwell agreed that that was the correct reading of the article, although did not retreat from the proposition that an increased risk applied.[99]

66. Professor Blackwell also agreed in oral evidence that the abstract of the article included the finding that:

> *[v]iral infection was not a major risk as long as babies were lightly wrapped. In heavily wrapped babies the presence of a viral infection greatly increased the risk of [sudden unexpected death].*[100]

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90 Transcript of the Inquiry, 22 March 2019 T348.48.
91 Transcript of the Inquiry, 22 March 2019 T320.42.
92 Transcript of the Inquiry, 22 March 2019 T320.48-49.
93 Transcript of the Inquiry, 20 March 2019 T155.18.
94 Transcript of the Inquiry, 20 March 2019 T155.10-12, T189.32-33 (Professor Cordner), T156.8-9 (Professor Duflou), T156.14 (Dr Cala), T157.21-31, T158.8-13 (Professor Hilton).
95 Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) p 8, citing Ruth Gilbert et al, 'Combined Effect of Infection and Heavy Wrapping on the Risk of Sudden Unexpected Infant Death' (1992) 67 *Archives of Disease in Childhood* 171.
96 Transcript of the Inquiry, 22 March 2019 T346.24-348.3.
97 Transcript of the Inquiry, 22 March 2019 T347.1-43.
98 Ruth Gilbert et al, 'Combined Effect of Infection and Heavy Wrapping on the Risk of Sudden Unexpected Infant Death' (1992) 67 *Archives of Disease in Childhood* 171, 174.
99 Transcript of the Inquiry, 22 March 2019 T347.6-43.
100 Transcript of the Inquiry, 22 March 2019 T346.31-40.

67. There is no evidence in the Inquiry that the children were heavily wrapped.[101]

68. I accept the submission from counsel assisting that the particular findings of the publication Professor Blackwell relied upon do not support her conclusion as to the extent of the increased risk of SIDS. The publication provides support for the contrary proposition that the risk was low, because the children were not heavily wrapped and the organisms were not found in the tracheal aspirate. Further, if they were contaminants, the article has no application as to risk.

Causes of death

69. Each of Professor Blackwell, Professor Clancy and Professor Goldwater have qualifications relating to immunology and microbiology. Professor Blackwell is not a clinician while Professor Clancy and Professor Goldwater were medically trained. None of them have any training, study or expertise in cardiology, forensic pathology or neurology.

70. Notwithstanding the fields that are – and are not – covered by those areas of study and training, each opined as to the causes of death of the children.

71. Professor Clancy concluded that two of the children died from SIDS, one likely had an ALTE leading to brain damage and Laura died from arrhythmia secondary to significant myocarditis.[102] He then emphasised by use of capitals that there is "NO evidence of any alternate cause of death".[103]

72. He also opined that "there are many reports of multiple cases of SIDS within a family".[104] He cited no publications to support this statement.

73. In his first report Professor Clancy referred to the histology findings as "real" with respect to all three children and then provided a different opinion when presented with the actual microbiology reports.[105]

74. Professor Goldwater was not given any of the primary evidence as to the causes of death, including autopsy reports, reports and evidence of treating practitioners and the forensic pathologists, medical records of the children or reports of others with different and pertinent expertise.[106]

75. He also was not provided with any evidence as to the environmental and historical circumstances of the children. For example, he did not know that none of the children were found prone.

76. On the basis of the reports of Professors Blackwell and Clancy, Professor Goldwater concluded that:

> *there is cogent and persuasive evidence that the Folbigg children died of natural causes. This conclusion is upheld by historical, pathological and microbiological evidence.*[107]

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101 2 April 2003 T104.17-18, T110.10-14, T128.15-17, T131.51-52; Exhibit E, ERISP of Kathleen Folbigg Q269.
102 Exhibit W, Report of Professor Robert Clancy (13 March 2019) p 3.
103 Exhibit W, Report of Professor Robert Clancy (13 March 2019) p 3.
104 Exhibit W, Report of Professor Robert Clancy (13 March 2019) p 3.
105 Exhibit W, Report of Professor Robert Clancy (13 March 2019) p 2.
106 Exhibit AU, Report of Professor Paul Goldwater (29 March 2019) p 2.
107 Exhibit AU, Report of Professor Paul Goldwater (29 March 2019) p 5.

77. However, I accept the submissions of counsel assisting that there were significant errors in respect to aspects of her reports. In her 2004 report, with respect to Patrick, Professor Blackwell stated that there was no evidence that an infective process had taken place.[108] In her March 2019 report, she noted that Patrick had had a fever the night before he died.[109] In her oral evidence she described Patrick as being "very ill" the night before he died.[110]

78. A record made by Dr Colley when consulting with Mr and Ms Folbigg after Patrick's death noted that the night before Patrick's death on 13 February 1991 he had a raised temperature, was sweating, vomiting and clinging.[111] However, contemporaneous hospital notes record that the night before he may have had a seizure and had a mild temperature but otherwise had "no problems".[112]

79. In her 2004 report, repeated in her March 2019 report, Professor Blackwell opined that there were indications in the children's medical histories that they had more frequent or more severe bouts of infection.[113] In her oral evidence, she said:

> *From the medical histories they seem to have attended the doctor for various coughs, colds and flu. I've never had any young children so I don't know if that was normal or if that was more frequent but certainly infection and referral to the GP for treatment seemed to come up in some of the material that I read.*[114]

80. She gave evidence that the children did not have any classical immunodeficiencies, by which she meant that the children did not have any pre-existing immunodeficiency which would have explained their death.[115]

81. I accept counsel assisting's submissions that Professor Blackwell's observations, upon which Professor Goldwater relied as accurate, as to the frequency of infections in the children and the nature of Patrick's fever, were contrary to the primary evidence.

Submissions of counsel assisting

Contamination

82. Counsel assisting submitted that I should prefer the evidence of the four forensic pathologists who gave evidence at the Inquiry, because they are trained clinicians who have performed autopsies and reported upon them for decades. They were essentially unanimous in their view.[116] Professor Hilton's evidence on contamination was consistent with the evidence he gave at trial. Counsel assisting submitted that the evidence of the immunologists suffered from the deficiencies set out above and should be rejected.

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108 Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure A, p 3.
109 Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) p 3.
110 Transcript of the Inquiry, 22 March 2019 T337.48.
111 Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure G.
112 Exhibit S, Section of Patrick's medical records, p 507.
113 Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure A, p 3; Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) p 4.
114 Transcript of the Inquiry, 22 March 2019 T317.23-27.
115 Transcript of the Inquiry, 22 March 2019 T317.22-27, T340.13-42.
116 Transcript of the Inquiry, 19 March 2019 T135.13- 45 (Professor Cordner), T136.13-137.5 (Professor Hilton), T137.9-11 (Dr Cala), T137.15-38 (Professor Duflou), T138.9-9 (all forensic pathologists agreeing); 20 March 2019 T153.29-22, T186.13-15 (Professor Hilton), T186.41-15 (Dr Cala and Professor Duflou), T187.27-30 (Dr Cala), T189.33 (with the exception of Professor Cordner in relation to Sarah, who preferred to leave "the possibility open that there is something there").

Role of infection in the deaths

83. Counsel assisting submitted that there is no reasonable possibility that those organisms, even if not contaminants, caused the deaths of any of the children.[117]

84. Counsel assisting further submitted that I should be comfortably satisfied on the basis of evidence received in the Inquiry that a mild infection may be a risk factor when considering a diagnosis of SIDS.[118] However, the evidence does not, on the whole, permit a conclusion that a mild infection can and does of itself cause sudden unexplained death in infants.[119]

85. As set out earlier in this report, Sarah and Laura each had a mild infection in the 24 hours before they died and Patrick had a mild temperature.[120]

86. Counsel assisting submitted that two questions arise. First, the extent to which, if at all, on the basis of evidence received in the Inquiry, the presence of infection in any of the children caused their death by sufficiently increasing their risk of SIDS. The second is the extent to which, if at all, infection otherwise contributed to their death. For the reasons which follow, counsel assisting submitted the presence of infection created no more than a theoretical possibility of an increase in SIDS risk, and also of contributing more directly to any of the deaths.

87. The Folbigg children were at low risk for SIDS, having none of the major identified risk factors. Importantly, they all slept alone on their backs, appropriately covered and their mother did not smoke.[121] In her oral evidence Professor Blackwell referred to susceptibility to infection being associated with two to four months of age, presence of older siblings, exposure to cigarette smoke, sleeping in a prone position, night time body temperature cycle and not being immunised.[122] However, the article on which Professor Blackwell sought to rely for the proposition that they were at a higher risk of SIDS establishes to the contrary – a viral infection was not a major risk as long as babies are not heavily wrapped.[123]

88. None of the children were in the two to four months age range when they died (nor was Patrick when he had his ALTE), none of them had older siblings who were alive, their mother did not smoke and their father smoked outside, they all slept supine and were immunised.[124] Laura and Patrick were asleep during the day time when they died, while the other two were sleeping at night and Patrick's ALTE was also during the night.[125]

89. In addition, evidence in the Inquiry established that none of them had genetic susceptibility to infection, impaired inflammatory response, or cardiac dysfunction. Further, it is clear that developmentally each was normal and prior to their death or in Patrick's case, his ALTE, each was healthy. In particular, none of them suffered from infections in excess of what may be expected in healthy children.[126]

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117 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [366].
118 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [367].
119 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [367].
120 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [368].
121 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [370].
122 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [371] referring to Transcript of the Inquiry, 22 March 2019 T341.28-344.12; Ruth Gilbert et al, 'Combined Effect of Infection and Heavy Wrapping on the Risk of Sudden Unexpected Infant Death' (1992) 67 *Archives of Disease in Childhood* 171.
123 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [370].
124 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [372].
125 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [372].
126 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [373].

90. Applying the risk factors set out by Professor Opdal: none of the children were at a vulnerable developmental stage at their death, none of them had a genetic predisposition and none had any significant risk factor. All they had was a mild infection.[127]

91. No forensic pathologist who gave evidence at the trial or in the Inquiry opined that any death or the ALTE was caused by infection. Indeed, even Professor Blackwell accepted that infection alone did not cause any of the deaths.

92. In counsel assisting's submission I should accept the evidence of Professor Elder that whereas the reason maternal smoking may cause an asphyxial insult is reasonably well-documented, the final mechanism where the risk is exposure to infection is not completely understood.[128] None of the experts identified a final mechanism connected with the role of infection that may have applied to any of the Folbigg children's deaths or ALTE.[129]

93. In the absence of any identified mechanism in this case and given that genetic susceptibility has been excluded, counsel assisting submitted that the evidence received in the Inquiry goes no further than raising for my consideration a theoretical possibility that slight infection in each of the older children may have contributed to their deaths. The evidence does not support a finding that this was reasonably possible or indeed, that any possibility was higher than theoretical.[130]

Submissions of Ms Folbigg

Infection as a cause of death

94. Ms Folbigg submitted that this is the area of the most considerable advances.[131] While sudden infant death is rare, just under 50 per cent of SIDS deaths are associated with a mild viral illness in the days beforehand.[132] Ms Folbigg submitted that the trial was conducted on the basis that all four children were otherwise healthy apart from a cold or sniffle.[133] The whole concept that the Folbigg children were healthy is apt to mislead and confuse with the scientific evidence now available.[134] On a statistical basis alone, the assertion in the Crown case that the children were healthy is scientifically dubious.[135] The statistic demonstrates a clear link between mild infection and sudden infant death.[136]

95. Professor Horne gave evidence that just under half of SIDS infants had a mild respiratory infection prior to death; she deferred to Professor Blackwell on issues of whether infection and the cytokine response can give rise to toxicity which can trigger an arrhythmia.[137] Professor Elder was not familiar with literature on infection.[138] Neither Professor Horne nor Elder mentioned infection as a risk factor.[139]

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127 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [374].
128 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [376] referring to Transcript of the Inquiry, 18 March 2019 T52.43-45.
129 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [376].
130 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [377].
131 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [126] nn 94-96.
132 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [126] nn 94-96.
133 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [125]-[126] nn 94-96.
134 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [127].
135 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [127].
136 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [127].
137 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [128] nn 97-99.
138 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [129] n 100.
139 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [130] nn 101-104.

96. Further, there was no reference to infection in the triple risk model proposed by Professors Horne and Elder, although it was part of the triple risk model contained in Chapter 30 of Duncan and Byard (2018) (Figure 1 above); this model, or triangle, is important.[140]

97. Professor Blackwell gave evidence beyond statistics as to how a mild infection can trigger sudden deaths, which explains the statistic and also the success of the Back to Sleep campaign (whereas at trial, the reasons for its success were not known).[141] She explained parallels between susceptibility to infection and risk factors for SIDS.[142] First, infants aged two to four months have the least amount of antibodies they will ever have.[143] Secondly, any older siblings bring home infection. Thirdly, exposure to cigarette smoke enhances susceptibility to infection.[144] The overall picture is like a jigsaw puzzle, with a hypothesis that the factors that make a child more susceptible to infection are those found amongst risk factors for SIDS.[145] The hypothesis includes infection, invasive infection, mucosal infection but also the production of toxins by bacteria that can cross the mucosal barrier and induce inflammatory responses.[146] So far, studies have found in relation to the prone sleeping position, that when a baby rolled onto its front the secretions in the nose would pool, increasing quantity and variety of bacteria.[147]

98. Professor Blackwell continued that the prone sleeping position is also important because it raises the temperature of the nose. Staphylococcus produces toxins between 37 and 40 degrees; normally nose temperature is well below 37 degrees but children in tests who were lying on their tummies had a significant rise in temperature in their nose.[148]

99. The toxins can cause a massive inflammatory response. Toxic shock syndrome from tampons infected with *Staphylococcus aureus* is an example.[149] The inflammatory response in white blood cells, caused by the bacteria and induced by the very powerful toxin, can affect all of the physiological systems in the body.[150]

100. In relation to the fatal triangle in Figure 1 above, Professor Blackwell said she would add to the developmental stage, the maturation of the night time body temperature cycle, which has been shown to be associated with other hormonal changes in babies.[151] While during the day, cortisol levels are quite steady and are fairly steady during the night, when a baby develops the lower night time body temperature associated with maturation (a "developmental switch"), the nighttime cortisol level "drops like a stone". Assessments have shown that once the switch occurs, there is a period when the very low levels of cortisol enhance (rather than dampening, which cortisol usually does), inflammatory responses.[152] The switch also takes place when many infants (of European extraction – for reasons unknown, the switch occurs later in Asian children) are susceptible to infection.[153]

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140 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [132] n 105, [130], citing Exhibit D, Siri Hauge Opdal, 'Cytokines, Infection and Immunity' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 689, 701.

141 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [131].

142 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [131].

143 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [131], citing Transcript of the Inquiry, 22 March 2019 T341.20-343.13.

144 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [131], citing Transcript of the Inquiry, 22 March 2019 T341.20-343.13.

145 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [131], citing Transcript of the Inquiry, 22 March 2019 T341.20-343.13.

146 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [131], citing Transcript of the Inquiry, 22 March 2019 T341.20-343.13.

147 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [131], citing Transcript of the Inquiry, 22 March 2019 T341.20-343.13.

148 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [131], citing Transcript of the Inquiry, 22 March 2019 T341.20-343.13.

149 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [131], citing Transcript of the Inquiry, 22 March 2019 T341.20-343.13.

150 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [131], citing Transcript of the Inquiry, 22 March 2019 T341.20-343.13.

151 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [133], citing Transcript of the Inquiry, 22 March 2019 T343.35-345.4 and Exhibit D, Siri Hauge Opdal, 'Cytokines, Infection and Immunity' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 689, 701.

152 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [133], citing Transcript of the Inquiry, 22 March 2019 T343.35-345.4 and Exhibit D, Siri Hauge Opdal, 'Cytokines, Infection and Immunity' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 689, 701.

153 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [133], citing Transcript of the Inquiry, 22 March 2019 T343.35-345.4.

101. In Ms Folbigg's submission this evidence was not relevantly challenged, and is supported by Professors Clancy and Goldwater.[154] Their opinions are consistent with the contents of Chapter 30 in Duncan and Byard (2018).[155] Professor Horne did not refer to recent studies on the relationship between sudden infant death and infection; Professor Elder said she was not familiar with relevant literature and it is not specifically within her expertise.[156] No relevantly qualified expert has gainsaid the opinions of Professors Blackwell, Clancy and Goldwater.[157]

102. Microbiological tests on autopsy of Patrick, Sarah and Laura returned positive results. Whether contaminants or not there were bacteria present in their bodies and also clinical signs of bacterial or illnesses at the time.[158] On autopsy, there were signs of inflammatory response in these three children, strengthening the inference that the virus or bacteria had triggered an immune response.[159] Tests available at the time would have confirmed this, but it is now too late.[160]

103. Ms Folbigg submitted that the evidence about infection "is of critical importance". Because of the association between mild viral infection and sudden infant death, one cannot submit they were healthy, and the medical conditions cannot simply be discussed as "the odd cold or sniffle" given that strong association.[161] This is more than a risk factor, and has become apparent since trial, and makes clear that evidence and submissions at trial that the children were healthy were misplaced – whether the expert evidence above is accepted or not, the jury were not entitled to assume the children were in perfect health with no underlying condition that could cause their sudden death.[162]

104. Whether or not the children presented with childhood illnesses more or less frequently does not affect this position, given infective or immunological findings at the time of their deaths.[163] Nor does the fact that the evidence above is not widely accepted by the forensic pathology community affect the position: first, the forensic pathologists accepted the proposition but described it as a "work in progress"; there are limits to forensic pathology expertise, noting Dr Cala described forensic pathology as a "blunt tool".[164]

105. Ms Folbigg sought findings that the research and scientific material on infection:

    a. was not available at trial;

    b. demonstrates a scientific theory that explains the continuing mortality rate from sudden infant death;

    c. explains the mortality rate arising from the Back to Sleep campaign, which was otherwise incapable of scientific explanation;

    d. explains the death of children predominantly at night or in the morning due to suppressed cortisol levels;

    e. explains racial differences; and

    f. explains the link between sudden infant death and cigarette smoking.[165]

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154 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [135] n 107.

155 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [141], citing Exhibit D, Siri Hauge Opdal, 'Cytokines, Infection and Immunity' in Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018) 689.

156 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [137]-[138] nn 109-111.

157 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [139].

158 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [142] nn 113-114.

159 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [143] nn 115-116.

160 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [144] nn 117-119.

161 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [145].

162 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [145].

163 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [146] nn 121-123.

164 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [147] nn 125-126.

165 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [148].

106. Ms Folbigg submitted that these matters raise a reasonable doubt about her guilt. Further, the submission by counsel assisting that evidence in relation to infection only posited a theory should be rejected, because this was Professor Hilton's observation from which he and the other forensic pathologists resiled.[166]

107. Rather, the Inquiry should find that mild infection may have triggered a cardiac arrhythmia as a potential natural cause of death, alternative to murder.[167] Given the presence of bacteria and mild inflammatory changes consistent with infection in Patrick, Sarah and Laura, infection was a reasonable available alternative cause of their deaths.[168]

Contamination and established pathological process

108. In respect of contamination, Ms Folbigg submitted that the determination of whether a bacterium within a body is a contaminant or not is a matter for forensic pathology and also for microbiology.[169] Antibodies to the pathogen in blood would indicate whether the person has been exposed to the bacteria for at least a week beforehand, and one also looks for clinical signs of infection.[170]

109. Professor Hilton's study found *Staphylococcus aureus* in half the samples examined, consistent with studies in France and Hungary, and lower than 65 per cent in a German study.[171] The evidence established a significant link between the presence of *Staphylococcus aureus* and SIDS, and corroborates Professor Horne's evidence.[172]

110. Caleb's left lung had mottled pleural surfaces (no microbiology tests were undertaken).[173] Patrick had a fever the night before he died, congestion in both lung and in his liver, and microbiology grew mixed organisms (with a debate as to contamination).[174] Sarah had a cold in the week prior to her death, was seen for a croupy cough on 26 August 1993, had a congested and red uvula, had congestion and infiltrate in the lungs, and inflammatory foci in salivary glands.[175] Bacterial organisms were propagated from the lungs, and there was moderate growth of coliforms of three types in the spleen (a sterile organ).[176] Laura had been ill for about a week before her death, and bacteria were isolated in the lungs and spleen.[177]

111. Each of Patrick, Sarah and Laura demonstrated clinical signs consistent with infection prior to their deaths, and each had bacteria found on testing. Each demonstrated signs of inflammation on autopsy.[178]

166 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [150] n 130.
167 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [150] n 130.
168 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [150] n 130.
169 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [175] n 146.
170 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [176] nn 147-147.
171 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [177] nn 149-151.
172 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [177] nn 149-151.
173 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [178].
174 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [179] n 152.
175 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [180].
176 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [181], Part C – Sarah [7] nn 7-8.
177 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A [182] n 153, Part C – Sarah [7] nn 7-8.
178 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [184].

112. In Ms Folbigg's submission, relevantly to whether detection of bacteria was contaminant or pathological, Professor Blackwell thought Sarah's swollen uvula may have resulted from inflammatory responses to a respiratory infection.[179] In any event, a contaminant is likely to be from the body of the deceased and testing as to source was limited.[180] Each of Patrick, Sarah and Laura had evidence of inflammatory response suggestive of infective disease process.[181] The forensic pathologists accepted that minor infection could be prelude to sudden infant death.[182]

113. Compared with evidence at trial (and with forensic pathology evidence in the Inquiry), which proceeded on the basis that the microbiology results reflected contamination, the combination of the previous ill health of Patrick, Sarah and Laura and the pathological signs of infection in each, there is a reasonable likelihood the microbiology results reflected an active infective process.[183] Importantly, the bacterium must have been somewhere on their bodies.[184]

114. Ms Folbigg submitted that is reasonably likely that Patrick, Sarah and Laura were each suffering from infection at the time of death.[185] There is a strong association with SIDS and *Staphylococcus aureus*.[186]

115. Further, the clinical signs in the hours or days before death, the microbiology results and examination of tissues combined so as to point to a physiological response to infection in Patrick, Sarah and Laura. This infection was a potential cause of death.[187] In that regard, Caleb (although with an absence of microbiological tests) had laryngomalacia, Patrick had a complicated encephalopathy and infection, Sarah had infection and a displaced uvula and Laura had myocarditis (heart infection).[188]

179 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [183] nn 154-155.
180 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [185] nn 156-157.
181 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [185] nn 156-157.
182 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [185] nn 156-157.
183 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [187].
184 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [187].
185 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [188].
186 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part A, [188].
187 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [7].
188 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [8].

Findings

116. Evidence concerning the histology findings and presence of infection found on autopsy was given at the trial by forensic pathologists as well as treating practitioners.

117. The evidence was that there were signs consistent with mild infection in Sarah and Laura and the organisms found were largely thought to be post-mortem contaminants. None of the findings on autopsy were considered significant or causative of death.

118. The forensic pathologists who gave evidence at the Inquiry were all of the view that those findings were likely post-mortem contaminants, although Professor Cordner preferred to "keep open the possibility that there is something there" in respect of Sarah.[189]

119. The immunologists who gave evidence were not, by contrast, at one in interpreting the organisms found on autopsy. In relation to Laura, Professor Blackwell opined they were not contaminants, however, did not specifically attribute Laura's death to them. Professor Goldwater opined that Laura probably died from myocarditis, however, the organisms could have played a role in her death. Professor Clancy in his first report said they were "real" findings (without having read the microbiology reports) and then in his second report, adopted the contrary view that post-mortem contamination was likely to account for Laura's histology results.[190]

120. Professors Blackwell, Goldwater and Clancy expressed the opinion that the findings in Sarah's lungs on autopsy were likely indicators of infection, with again, only Professor Goldwater opining directly that they would have played a role in her death. Professor Clancy differed from his colleagues in finding that the organisms found in Sarah's spleen suggested contamination. However, again in his first report he had said they were "real" findings.[191]

121. Dr Drucker was equivocal in his opinions and ultimately recommended more information be sought.

122. The evidence of the microbiologists goes no further than to speculate that infection may have played a part in some of the Folbigg children's deaths.

123. I accept that current research and scientific material on infection was not available at trial which refers to a scientific theory that might explain the continuing mortality rate from sudden infant death. However, it cannot be elevated beyond that.

124. I also note that it would be remarkably coincidental if, as submitted by Ms Folbigg, each of these deaths occurred from an infection.

189 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Caleb, [8].

190 Exhibit W, Report of Professor Robert Clancy (13 March 2019) p 2; Further report of Professor Robert Clancy (17 March 2019) p 3.

191 Exhibit W, Report of Professor Robert Clancy (13 March 2019) p 2.

Report of the Inquiry into the convictions of Kathleen Megan Folbigg

July 2019

Volume 1: Chapter 7

Chapter 7: Genetics evidence

Introduction

1. The medical evidence in the Crown case at trial included evidence relating to testing for a limited range of genetic and other disorders which had been conducted both during the course of Patrick's life, and after the children's deaths prior to the trial.

2. This chapter outlines the genetics-related evidence at trial, the advances in the field of genetics since the trial, the further genetic testing undertaken by the Inquiry and the evidence in the Inquiry about the interpretation of the results of that testing. This chapter also outlines the submissions of counsel assisting and Ms Folbigg in relation to the genetics evidence, and my findings on this topic.

Genetics evidence at the trial

Dr Bridget Wilcken – clinical geneticist

3. As set out earlier, Dr Bridget Wilcken was a clinical geneticist and director of the NSW Newborn Screening Program. She was also employed as a senior staff physician at the Children's Hospital at Westmead.[1]

4. Dr Wilcken gave evidence in the Crown case at trial about the genetic testing which had been conducted in December 1999 in respect of newborn blood samples from all four children. She explained that taking a blood sample from all newborn babies was a practice which had been in place since the 1970s.[2]

5. Dr Wilcken explained that at the time of their respective births, routine tests were conducted on the children's fresh samples, though these tests were only for a certain limited number of disorders. She said subsequent to that, the process of tandem mass spectrometry became available. That process permitted testing for "pretty well all important disorders related to amino acid metabolism and fatty protein metabolism and fat metabolism."[3] She said the results from this testing in respect of each child were "entirely normal".[4]

6. Dr Wilcken described that "something like a total of 50" genetic metabolic disorders which might be associated with unexpected death, including MCAD, had been tested for from the blood samples.[5] She explained that MCAD meant "medium chain acyl CoA dehydrogenase deficiency" and she was "confident that has been excluded utterly."[6]

1 16 April 2003 T817.30-57.
2 16 April 2003 T819.21-23.
3 16 April 2003 T819.55-820.7.
4 16 April 2003 T820.12-14.
5 16 April 2003 T820.48-821.6.
6 16 April 2003 T821.20-26.

7. Dr Wilcken said that testing of urine taken from Patrick after his ALTE had also been conducted by her facility. The urine was examined for markers or metabolic disorders in amino acids, organic acids and fatty acids. However no genetic or metabolic disorders were detected and many "could be ruled out utterly."[7]

Dr Christopher Seton – sleep specialist

8. Dr Seton was a sleep physician with SIDS expertise who assessed Laura after her birth when she attended the Sleep Disorders Unit at the Children's Hospital at Westmead.[8]

9. He said testing had been done for potentially inherited breathing and non-breathing disorders, the latter including MCAD and some potentially inheritable metabolic disorders.[9]

10. Dr Seton described the metabolic disorders tested for as:

> *potentially inheritable… very rare disorders and would be highly unlikely to occur, but in a family where there's more than one death suddenly in a baby we would routinely do that testing.*[10]

11. He opined that in terms of inheritable disorders that were:

> *known to cause sudden death in babies, I would think our testing was exhaustive and I don't think that any other institution in the world would test for any other disorders that we don't know about.*[11]

Advances in the field of genetics since the trial

12. Significant advances have been made in the field of genetics since Ms Folbigg's trial. Those advances permit a much broader scope of investigation than was possible in 2003.[12]

13. As part of the Inquiry's initial investigations, a report was obtained from (the now) Professor Wilcken, a staff specialist at the Centre for Clinical Genetics at the Sydney Children's Hospital at Randwick.[13]

14. Professor Wilcken explained that the technology used prior to the trial, which tested for all significant disorders of organic acid metabolism and fatty acid oxidation, remains state of the art. Accordingly, she concluded that the exclusion of all such significant disorders remained valid. She opined that the other negative studies including the clinical data in each case, and the urine metabolic screening tests carried out on Patrick, Sarah and Laura Folbigg, supported this conclusion.[14]

15. Professor Wilcken opined that if a cause of death other than human interference were to be found in relation to each of the children, it was very likely that there must be a strong genetic component. She observed there had been a great deal of development in genetic testing since the time of the trial and recommended consulting with a clinical geneticist and a genomics expert.[15]

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7 16 April 2003 T818.38-819.12.
8 15 April 2003 T690.3-5, T691.24-54.
9 15 April 2003 T697.15-698.3.
10 15 April 2003 T697.42-49.
11 15 April 2003 T697.56-698.3.
12 Exhibit AA, Report of Dr Alison Colley (26 November 2018) p 2; Exhibit AB, Report of Dr Michael Buckley (25 February 2019) p 2.
13 Exhibit AC, Genetics tender bundle, Report of Professor Bridget Wilcken (24 November 2018) p 3.
14 Exhibit AC, Genetics tender bundle, Report of Professor Bridget Wilcken (24 November 2018) p 2.
15 Exhibit AC, Genetics tender bundle, Report of Professor Bridget Wilcken (24 November 2018) p 3.

16. The Inquiry also obtained an initial report from Dr Alison Colley, a clinical geneticist at the Newcastle and Northern NSW Genetics Service to whom Mr and Ms Folbigg were referred in 1991. Dr Colley is now the director of South West Sydney Local Health District Clinical Genetic Services. Dr Colley also identified there had been significant changes in genetic testing since the time of the trial. She recommended considering the samples available from each child for the purpose of DNA extraction and genomic study and analysis of genetic variants by a multi-disciplinary team.[16]

17. Genomic sequencing technologies emerged in 2009. Since 2013, two major genomics sequencing technologies have become mainstream.[17]

18. Whole Exome Sequencing ("WES") sequences the exome, which is that small part (approximately one to two per cent of the whole) of the genome (the complete set of genes carried by an individual)[18] that is involved in coding for proteins. Proteins are the key components of cells and damage to them can cause serious, if not catastrophic, problems. This part of the genome is the location of the majority of the variants that cause developmental or cognitive disabilities and disorders.[19]

19. Whole Genome Sequencing ("WGS") sequences all of the genome that is accessible. In addition to the exome, this comprises non-coding elements in the genome and mitochondrial DNA.[20]

20. This technology enables hypothesis-free study of DNA where a known or presumed diagnosis as a starting point is not needed. Rather, DNA sequences are studied and variants are interrogated against the known healthy human genome and the phenotype or clinical features of a person.[21]

21. Since the introduction of genomic sequencing, the pace at which the genes underlying genetic disorders are discovered per year has increased.[22] The proportion of discoveries made by genomic approaches as compared with conventional approaches has steadily increased. Together WES and WGS have discovered nearly three times as many genes as conventional sequencing approaches that were available in the 1990s.[23]

22. In addition, international information sharing and efforts to systematise the evidentiary basis for gene pathogenicity (determining whether a genetic variant is likely to cause disease) have been instrumental in translating voluminous sequencing data into information which can be transferred to the clinical setting.

23. These efforts have produced:

    a. reference population datasets (which continue to grow);

    b. standards and guidelines for the interpretation of sequencing information and the categorisation of gene pathogenicity; and

    c. a regulatory framework to ensure that laboratory practice offers acceptable levels of patient safety, clinical utility and cost effectiveness.

24. In 2015, the American College of Medical Genetics and Genomics published Standards and Guidelines ("the ACMG Guidelines") for the interpretation of genetic sequence variants, including assessing the pathogenicity of variants.[24]

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16 Exhibit AA, Report of Dr Alison Colley (26 November 2018) p 2.
17 Exhibit AA, Report of Dr Alison Colley (26 November 2018) p 2; Exhibit AB, Report of Dr Michael Buckley (25 February 2019) p 1.
18 NSW Health, 'Glossary', *Centre for Genetics Education* (Web Page, 26 February 2016).
19 Exhibit AA, Report of Dr Alison Colley (26 November 2018) p 2; Exhibit AB, Report of Dr Michael Buckley (25 February 2019) p 1.
20 Exhibit AA, Report of Dr Alison Colley (26 November 2018) p 2; Exhibit AB, Report of Dr Michael Buckley (25 February 2019) p 1.
21 Exhibit AA, Report of Dr Alison Colley (26 November 2018) p 2; Exhibit AB, Report of Dr Michael Buckley (25 February 2019) p 2.
22 Exhibit AB, Report of Dr Michael Buckley (25 February 2019) p 1.
23 Exhibit AB, Report of Dr Michael Buckley (25 February 2019) p 1.
24 Exhibit AC, Genetics tender bundle, ACMG Guidelines.

25. The purpose and limitations of the ACMG Guidelines are as follows:

> *The following approach to evaluating evidence for a variant is intended for interpretation of variants observed in patients with suspected inherited (primarily Mendelian) disorders in a clinical diagnostic laboratory setting. It is not intended for the interpretation of somatic variation, pharmacogenomic (PGx) variants, or variants in genes associated with multigenic non-Mendelian complex disorders. Care must be taken when applying these rules to candidate genes ("genes of uncertain significance" (GUS)) in the context of exome or genome studies… because this guidance is not intended to fulfil the needs of the research community in its effort to identify new genes in disease.*[25]

26. The ACMG Guidelines continue:

> *it is important to consider the differences between implicating a variant as pathogenic (i.e., causative) for a disease and a variant that may be predicted to be disruptive/ damaging to the protein for which it codes but is not necessarily implicated in a disease.*[26]

27. The ACMG Guidelines provide for the classification of genetic variants as "pathogenic", that is, causative of disease, "likely pathogenic", "of uncertain significance", "likely benign" and "benign".[27] It is referred to as a five-tier system. The terms "likely pathogenic" and "likely benign" are used to mean greater than 90 per cent certainty of a variant either being disease causing or benign.[28]

28. The ACMG Guidelines provide two sets of criteria: one for classification of pathogenic or likely pathogenic variants, and one for classification of benign or likely benign variants. For a given variant, the user selects the variously weighted criteria ("very strong", or "strong", or "moderate", or "supporting" for pathogenic and "stand-alone", or "strong" or "supporting" for benign), which are then combined according to scoring rules to choose a classification from the five-tier system. If the variant does not fulfil criteria using either of the pathogenic or benign criteria sets, or the evidence for benign and pathogenic is conflicting, the variant defaults to uncertain significance.[29]

29. The variant interpretation process provided for by the ACMG Guidelines requires the interrogation of evidence from:

    a. scientific and medical literature;

    b. databases such as population databases, disease databases and sequence databases; and

    c. the tested person's clinical information.

30. The ACMG Guidelines explain the significance of a person's clinical information in interpreting variant data as follows:

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25 Exhibit AC, Genetics tender bundle, ACMG Guidelines, p 410.
26 Exhibit AC, Genetics tender bundle, ACMG Guidelines, p 411
27 Exhibit AC, Genetics tender bundle, ACMG Guidelines, p 405.
28 Exhibit AC, Genetics tender bundle, ACMG Guidelines, p 407.
29 Exhibit AC, Genetics tender bundle, ACMG Guidelines, p 411.

> *When a health-care provider orders genetic testing, the patient's clinical information is integral to the laboratory's analysis. As health-care providers increasingly utilised genomic (exome or genome) sequencing, the need for detailed clinical information to aid in interpretation assumes increasing importance. For example, when a laboratory finds a rare or novel variant in a genomic sequencing sample, the director cannot assume it is relevant to a patient just because it is rare, novel, or de novo. The laboratory must evaluate the variant and the gene in the context of the patient's and family's history, physical examinations, and previous laboratory tests to distinguish between variants that cause the patient's disorder and those that are incidental (secondary) findings or benign… Many genetic variants can result in a range of phenotypic expression (variable expressivity), and the chance of developing may not be 100% (reduced penetrance), further underscoring the importance of providing comprehensive clinical data to the clinical laboratory to aid in variant interpretation.*[30]

31. As to the significance of clinical information from family members to variant interpretation, the ACMG Guidelines explain:

> *testing other family members to establish when a variant is de novo, when a variant cosegregates with disease in the family, and when a variant is in trans with a pathogenic variant in the same recessive disease-causing gene is valuable. Filtering out or discounting the vast majority of variants for dominant diseases when they can be observed in healthy relatives is possible, making the interpretation much more efficient and conclusive. To this end, it is strongly recommended that every effort be made to include parental samples along with that of the proband, so-called "trio" testing (mother, father, affected child), in the setting of exome and genome sequencing, particularly for suspected recessive or de novo causes. Obviously this will be easier to achieve for paediatric patients than for affected adults. In the absence of one or both parents, the inclusion of affected and unaffected siblings can be of value.*[31]

Samples available for genetic testing

32. Material produced to the Inquiry by NSW Health in November 2018 in compliance with summonses included biological samples containing DNA from each of the four children.

33. The blood spots taken from each of the children at the time of their birth as part of the Newborn Screening Program and held at the Children's Hospital at Westmead were available.[32]

34. In respect of each of Patrick, Sarah and Laura, tissue samples taken at the time of their autopsies in 1991, 1993 and 1999 respectively and fixed in glass and wax block slides held at the Coroner's Court were also available.[33]

35. In respect of Patrick, additionally available were kidney, liver, skin, skeletal muscle and heart tissue samples taken at the time of his autopsy in 1991 and frozen at minus 80 degrees Celsius.[34]

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30 Exhibit AC, Genetics tender bundle, ACMG Guidelines, p 422.
31 Exhibit AC, Genetics tender bundle, ACMG Guidelines, p 422.
32 Exhibit AC, Genetics tender bundle, List of samples held by NSW Health (15 November 2018).
33 Exhibit AC, Genetics tender bundle, Histology request forms of Patrick, Sarah and Laura (undated), pp 1-5.
34 Exhibit AC, Genetics tender bundle, Email from Amber Richards to genetics data interpretation teams (10 December 2018).

36. In respect of Sarah, additionally available was one tube of extracted genomic DNA from fibroblasts, and two ampules of archived fibroblast cells stored in liquid nitrogen, held at the Children's Hospital at Westmead.[35]

37. In respect of Laura, held at the Children's Hospital at Westmead was formalin-immersed brain tissue taken at the time of her autopsy in 1999.[36]

38. In December 2018 the Inquiry was informed that Ms Folbigg had provided to her legal representatives a sample for the purpose of genetic testing.[37] Ms Folbigg consented to the sample being made available to the Inquiry for further genetic testing.[38]

Engagement of multi-disciplinary panel of experts and consultation with Ms Folbigg's legal representatives and experts

39. In light of the significant advances relevant to the scope of the Inquiry, I determined that further investigations into genetic testing of the four deceased children should be pursued.

40. The Inquiry engaged Dr Michael Buckley to advise and assist in the task of arranging for genetic testing to be undertaken. Dr Buckley is a genetic pathologist and clinical director of the NSW Health South Eastern Area Laboratory Services at the Prince of Wales Hospital in Sydney. He holds a PhD in the field of molecular genetics, obtained in 1991.[39]

41. The interpretation of genetic data involves consideration of both the genetic pathology and the clinical presentation of a person. It is a single, but multi-faceted, interpretation process.[40]

42. Accordingly, the Inquiry gathered a multi-disciplinary panel of experts to interpret and provide opinions about the data produced by the genetic testing undertaken for the Inquiry, and the available clinical information in respect of each of the children and Ms Folbigg.

43. In pursuing genetic investigations, the Inquiry adopted a decision-making process which involved experts retained by Ms Folbigg as well as the Inquiry. The Inquiry convened a series of meetings of those experts, at which lawyers assisting the Inquiry and representing Ms Folbigg's interests were present.

The experts

44. The experts were associated with two separate laboratories with genetic sequencing interpretation capabilities: in Sydney and in Canberra.

45. In addition to his initial advisory role, Dr Buckley was engaged to interpret the genetic sequencing data.

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35 Exhibit AC, Genetics tender bundle, List of samples held by NSW Health (15 November 2018).
36 Exhibit AC, Genetics tender bundle, List of samples held by NSW Health (15 November 2018).
37 Exhibit AG, Report of Professor Carola Vinuesa (2 December 2018).
38 Transcript of the Inquiry, 15 April 2019 T404.3-11.
39 Exhibit Z, Joint report of Sydney genetics team (29 March 2019) CV of Dr Michael Buckley; Transcript of the Inquiry, 15 April 2019 T372.33-38.
40 Transcript of the Inquiry, 15 April 2019 T400.40-46, T410.28-411.8.

46. Professor Edwin Kirk is a genetic pathologist and clinical geneticist at the NSW Health South Eastern Area Laboratory Services as well as co-head of the Centre for Clinical Genetics at the Sydney Children's Hospital. He has a PhD in cardiac genetics and was head of the Metabolic Diseases Service at Sydney Children's Hospital for 12 years. He has additionally trained in paediatrics and provides a cardiac genetics clinical service which focuses on adults and children with cardiomyopathies and disorders of cardiac rhythm.[41]

47. Dr Alison Colley is a clinical geneticist and the director of the Newcastle and Northern NSW Genetics Service. She has trained in paediatrics as well as clinical genetics. She is a conjoint Senior Lecturer at the University of New South Wales. Dr Colley is a renowned dysmorphologist.[42]

48. Dr Buckley, Professor Kirk and Dr Colley undertook the interpretation exercise together ("the Sydney Team") and produced a joint report.

49. Professor Matthew Cook is a Professor of Medicine at the Australian National University, and a practising clinical immunologist at Canberra Hospital. He is also co-director of the Centre for Personalised Immunology at the Australian National University, and medical director of the Canberra Clinical Genomics laboratory.[43] That laboratory is accredited to conduct bioinformatics analysis of DNA and RNA sequences, such as those produced by WES or WGS.[44]

50. Professor Carola Vinuesa is an Australian National Health and Medical Research Council Principal Research Fellow and Professor of Immunology at the Australian National University. She is also the chief scientist at the Canberra Clinical Genomics laboratory of which Professor Cook is the medical director. Together with Professor Cook, she is also the co-director of the Centre for Personalised Immunology.[45]

51. Professors Cook and Vinuesa undertook the interpretation exercise together ("the Canberra Team") and produced a joint report. Professors Cook and Vinuesa were assisted by Dr Todor Arsov, a visiting fellow at the Centre for Personalised Immunology. He holds a PhD in biomedical sciences and a Master of Genetic Counselling.[46]

52. Professor Jonathan Skinner is a paediatric cardiologist and cardiac electrophysiologist working as a consultant at Starship Children's Hospital in Auckland, New Zealand. He is an Honorary Professor in Paediatrics, Child and Youth Health at the University of Auckland.[47] Professor Skinner prepared a separate report focussing on the cardiac aspects of the results.[48]

The Inquiry's consultation process

53. The Inquiry held three consultation meetings from December 2018 to February 2019 at which the experts identified above discussed the options for genetic testing on the produced samples, and the process to be adopted in the interpretation phase.

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41 Exhibit Z, Joint report of Sydney genetics team (29 March 2019) CV of Professor Edwin Kirk; Transcript of the Inquiry, 15 April 2019 T370.25-47.

42 Exhibit Z, Joint report of Sydney genetics team (29 March 2019) CV of Dr Alison Colley; Transcript of the Inquiry, 15 April 2019 T373.39-50.

43 Exhibit AF, Joint report of Canberra genetics team (29 March 2019) CV of Professor Matthew Cook; Transcript of the Inquiry, 15 April 2019, T366.19-25.

44 Exhibit AF, Joint report of Canberra genetics team (29 March 2019) p 4.

45 Exhibit AF, Joint report of Canberra genetics team (29 March 2019) CV of Professor Carola Vinuesa; Transcript of the Inquiry, 16 April 2019 T460.15-18.

46 Exhibit AF, Joint report of Canberra genetics team (29 March 2019) p 6; Transcript of the Inquiry, 16 April 2019 T462.19-21, T462.25-26.

47 Exhibit Y, Report of Professor Jonathan Skinner (31 March 2019) CV of Professor Jonathan Skinner; Transcript of the Inquiry, 15 April 2019 T369.8-9, T369.13-21.

48 Exhibit Y, Report of Professor Jonathan Skinner (31 March 2019).

54. Given the age of the samples, and in some instances uncertainty as to the conditions in which the samples had been stored, the experts assessed that the quality of the DNA able to be extracted from the samples was unknown and potentially likely to be poor. Nonetheless, an important consideration was the interest in retaining, if possible, some of the samples available for future testing in the event of further developments relevant to cause of death.

55. There was consensus among the experts that there would likely be a significant advantage to having both parents' DNA, particularly in circumstances where it was expected that good quality DNA from the children would only be likely in respect of Sarah.[49] This was described as "trio sequencing".[50] It was anticipated at that stage that the consequences of some variants may not be able to be fully understood without the father's genetic information.[51] Nonetheless, there was consensus that undertaking sequencing of available DNA material from the children and Ms Folbigg would still be a worthwhile exercise if Mr Folbigg's DNA was not available.[52]

56. Mr Folbigg was invited to provide a DNA sample for the purpose of genetic testing. Mr Folbigg declined to provide a sample. There was no power available to the Inquiry to compel Mr Folbigg to provide such a sample.

57. At the 10 December 2018 meeting Professor Vinuesa discussed a report prepared by her and dated 2 December 2018 concerning the results of whole exome sequencing undertaken at the Centre for Personalised Immunology on Ms Folbigg's DNA extracted from a buccal swab and saliva. That report identified two candidate genes in Ms Folbigg: CALM2 and MYH6. Professor Vinuesa opined "given the associations between heterozygous mutations in both genes and sudden cardiac death, cardiac investigation in Kathleen Folbigg is advisable".[53]

58. At a further consultation meeting on 4 February 2019, Professor Vinuesa agreed without qualification to the application of the ACMG Guidelines for assessing the pathogenicity of variants.[54] It was also agreed that the Sydney and Canberra teams would pursue parallel analyses of the primary data through dual pipelines and then amalgamate the results into a single list for filtering and assessment of pathogenicity.[55]

59. Following the meeting, on 11 February 2019 Professor Skinner provided a list of recommended cardiac investigations in respect of Ms Folbigg. He recommended:

    a. a thorough clinical history relevant to cardiac issues;

    b. a good clinical examination;

    c. resting 12 lead ECG;

    d. standing and exercise ECG with post-exercise recording to at least six minutes;

    e. echocardiogram; and

    f. Holter (24 hours ECG).

60. Professor Vinuesa agreed with Professor Skinner's list of investigations.[56] Over the course of the following weeks and months, results from these investigations were produced to the Inquiry. Those results were considered by the experts in their reports, and in their evidence before the Inquiry, as detailed further below.

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49 Exhibit CB, Transcript of first meeting with expert geneticists (10 December 2018) p 5.
50 Exhibit CB, Transcript of first meeting with expert geneticists (10 December 2018) p 17.
51 Exhibit CB, Transcript of first meeting with expert geneticists (10 December 2018) p 4.
52 Exhibit CB, Transcript of first meeting with expert geneticists (10 December 2018) pp 4, 8-9.
53 Exhibit AG, Report of Professor Carola Vinuesa (2 December 2018) p 1.
54 Exhibit CC, Transcript of second meeting with expert geneticists (4 February 2019) T10.40-50.
55 Exhibit CC, Transcript of second meeting with expert geneticists (4 February 2019) T10.10-35.
56 Exhibit CD, Emails exchanged between Professor Skinner and Professor Vinuesa (11 and 12 February 2019).

61. At a final consultation meeting on 27 February 2019, after the data had been initially reviewed by the experts, it was agreed the data coverage was sufficient and further genetic testing by way of segregation in respect of the CALM2 and MHY6 variants (the subject of Professor Vinuesa's 2 December 2018 report) was not required. The experts also discussed the list of terms to be included in the literature search to be undertaken as part of the interpretation process, and agreed that any terms Professor Vinuesa wished to add would be included. That occurred.

The genetic sequencing of the children and Ms Folbigg

62. On the basis of the discussions at the December 2018 meeting, with the assistance of Dr Buckley the Inquiry engaged two genetics services laboratories to conduct extraction and testing of some of the available samples.

63. The Australian Genomics Research Facility ("AGRF") was engaged to conduct DNA extraction and WGS in respect of available samples from Sarah (fibroblasts) and Patrick (frozen liver tissue), and from Ms Folbigg (buccal sample). The AGRF laboratory was selected because it is accredited for WGS on the types of samples provided to it.

64. Accreditation in this context refers to accreditation of the qualification, training and experience of staff; correctly calibrated and maintained equipment; adequate quality assurance processes; and appropriate sampling practices against an international standard by the National Association of Testing Authorities ("NATA").[57]

65. The Victorian Clinical Genetics Service ("VCGS") was engaged to conduct DNA extraction and WGS in respect of blood spot samples from Caleb and Laura. The VCGS laboratory was awaiting approval of accreditation for WGS at the time of sequencing, though this accreditation would not extend to the age of the samples provided to it. No laboratory in Australia is accredited for WGS on that type of sample.

66. On 2 January 2019, samples from Sarah, Patrick and Ms Folbigg for WGS were delivered to AGRF in Melbourne. On 10 January 2019, samples from Caleb, Patrick, Sarah and Laura were provided to VCGS, also in Melbourne.

67. Ultimately, WGS was conducted on:

    a. DNA extracted from a frozen liver tissue sample from Patrick;

    b. DNA extracted from fibroblasts from Sarah;

    c. DNA extracted from a blood spot sample from Caleb; and

    d. DNA extracted from the buccal sample from Ms Folbigg.[58]

68. WES was conducted on DNA extracted from a blood spot sample from Laura, which was unsuitable for WGS because of microbial contamination of the sample.[59]

69. Completion of the extraction and sequencing exercises by AGRF and VCGS took some four to five weeks. The quality of the results and data produced, given the age of the samples, exceeded the experts' initial expectations. Professor Kirk gave evidence in the Inquiry that:

> *I think it's remarkable that it was possible to perform testing of this type on these samples, and the resulting data quality was good... I don't know that something like this has been attempted before, at least not in this kind of context, and the outcome was that we got very high quality data that was able to be interpreted.*[60]

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57 See 'Accreditation', *AGRF* (Web Page) <http://www.agrf.org.au/about/accreditation>.

58 Transcript of the Inquiry, 15 April 2019 T403.24-404.1; Exhibit Z, Joint report of Sydney genetics team (29 March 2019) p 4.

59 Transcript of the Inquiry, 15 April 2019 T407.3-20; Exhibit Z, Joint report of Sydney genetics team (29 March 2019) p 4.

60 Transcript of the Inquiry, 15 April 2019 T407.36-44.

70. By 15 February 2019, all data from the sequencing was provided to the Sydney and Canberra teams. The experts were requested to, and did, report on their interpretation of the results by 29 March 2019.

Analysis and reports

71. At the NSW Health Pathology Genetics Laboratory at the Prince of Wales Hospital in Sydney, variant analysis of the sequencing data was conducted through a genomic analysis bioinformatics pipeline called the Genomic Annotation and Interpretation Application.[61]

72. At the Canberra Clinical Genomics laboratory, variant analysis of the sequencing data was conducted through a separate bioinformatics pipeline.[62]

73. Ultimately, each laboratory analysed the same data and the same genes. Almost 1,400 unique candidate genes were identified for analysis.[63]

74. In addition, the data was re-analysed considering:

    a. cardiac/non-cardiac genes which had been published in relation to sudden death in infancy/childhood;

    b. genes associated with childhood neurological disorders;

    c. genes associated with immunology;

    d. genes associated with metabolics; and

    e. likely pathogenicity in any phenotype not restricted to sudden death in infancy/childhood.[64]

75. As noted above, it was agreed by the experts that the ACMG Guidelines would be used for assessing the pathogenicity of variants.[65]

76. All experts involved in the interpretation of the sequencing data were provided with documents relevant to the phenotype or clinical presentation of the children and Ms Folbigg.[66] The phenotype or observable clinical features of the children was of healthy, well-grown, normally developing children who are normal in appearance, each of whom suffer a catastrophic event leading to death in three of them, and severe neurological sequelae in the fourth child which precedes his later death.[67]

77. The relevant medical history and results of historical and recent cardiac-related investigations on Ms Folbigg were considered by the experts in the interpretation process.

78. The Sydney Team prepared a joint report interpreting the significance of genetic variants, identified through the Sydney pipeline, present in the children and in Ms Folbigg and potentially relevant to the children's causes of death ("the Sydney report").[68]

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61 Exhibit Z, Joint report of Sydney genetics team (29 March 2019) p 6.
62 Exhibit AF, Joint report of Canberra genetics team (29 March 2019) p 3.
63 Exhibit AW, Gene lists from Sydney and Canberra genetics teams.
64 Exhibit Z, Joint report of Sydney genetics team (29 March 2019) p 9.
65 Transcript of the Inquiry, 15 April 2019 T402.25-31.
66 See generally Exhibit AC, Genetics tender bundle.
67 Exhibit Z, Joint report of Sydney genetics team (29 March 2019) p 5; Transcript of the Inquiry, 15 April 2019 T367.26-30, T381.9-382.21.
68 Exhibit Z, Joint report of Sydney genetics team (29 March 2019) p 6.

79. The Canberra Team prepared a joint report and a supplementary report interpreting the significance of genetic variants identified through the Canberra pipeline ("the Canberra report").[69]

80. Professor Skinner prepared a report specifically addressing cardiac-related variants in the children's and Ms Folbigg's genes as reported by the Sydney and Canberra pipelines, and the cardiac-related clinical presentation of each of them.[70] Upon receipt of additional cardiac testing information in relation to Ms Folbigg, he prepared a supplementary report and provided a further advice addressing the results of the additional testing and Ms Folbigg's clinical presentation.[71]

Limitations to results interpretation suggested by the Canberra team

81. It became clear during Professor Vinuesa's evidence that she was not satisfied that there was sufficient clinical information about the children and Ms Folbigg and that this accounted for the Canberra Team assigning different pathogenicity scores using the ACMG Guidelines.[72]

82. Professor Vinuesa gave evidence that she did not consider using the ACMG Guidelines was appropriate.[73] It appeared from the written response from both Professors Vinuesa and Cook dated 12 April 2019 that Professor Cook agreed with this position. The reasons offered for the position were:

    a. the absence of "full phenotypes" of the children, meaning many of the criteria in the Guidelines could not be scored;

    b. the task as related to the Inquiry was not a diagnostic exercise that aimed to identify clinically-actionable mutations;

    c. that the Guidelines are not designed to identify digenic genetic causes of death; and

    d. that the Guidelines focus on the one to 10 per cent margin of uncertainty, whereas a 15 per cent margin of uncertainty may be tolerable in the Inquiry's non-clinical setting.[74]

83. Professors Cook and Vinuesa suggested "the current exercise should lie somewhere between a research approach and a clinical approach".[75]

84. By contrast, the Sydney Team comprising three clinical geneticists and/or pathologists (including one with specific cardiac and metabolic experience), and one paediatric cardiologist, were confident in applying the ACMG Guidelines to the investigation of sudden death of a child, which Dr Buckley, Professor Kirk and Dr Colley described as "intrinsically a clinical matter".[76]

85. The Sydney Team were also confident of the phenotype.[77] It was suggested to Dr Colley that:

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69 Exhibit AF, Joint report of Canberra genetics team (29 March 2019) p 3.

70 Exhibit Y, Report of Professor Jonathan Skinner (31 March 2019).

71 Exhibit BJ, Further report of Professor Jonathan Skinner (24 April 2019); Exhibit BK, Letter from Professor Jonathan Skinner to the Inquiry (30 April 2019).

72 Transcript of the Inquiry, 16 April 2019 T475.8-25.

73 Transcript of the Inquiry, 16 April 2019 T504.31-33.

74 Transcript of the Inquiry, 16 April 2019 T477.1-10, T479.20-32, T504.28-32; Exhibit AY, Response from Canberra genetics team to response of Professor Kirk and Dr Buckley (12 April 2019) p 1.

75 Exhibit AY, Response from Canberra genetics team to response of Professor Kirk and Dr Buckley (12 April 2019) p 1.

76 Transcript of the Inquiry, 17 April 2019 T579.1-18.

77 Transcript of the Inquiry, 16 April 2019 T480.9-19, T520.5-14, T520.30-33.

MORRIS SC: … In relation to the phenotypes, we talked about phenotypes yesterday, and to that extent is it a fair comment - I know that the postulated phenotype has been sudden infant death, unexplained infant death, for the purpose of everybody's analysis. And is it fair to say that because of the breadth of the phenotype it's a little difficult from a genetic point of view to target genetic investigation or not really?

WITNESS COLLEY: … The phenotype is not only both sudden unexpected early death, but also normalcy. The children were well grown, appeared normal, did not have any dysmorphic features, did not have any birth defects or malformations, were meeting their milestones, and then had a catastrophic acute onset life threatening event. Now, because of that we don't have a particular disease phenotype that we were targeting, so that's why we used Whole Genome Sequencing and when we couldn't, Whole Exome Sequencing, to look with as much breadth as possible at all possible genetic causes of being entirely normal and then having a catastrophic event.

So no, I think that phenotype was quite clear because it was so consistent between the four children, including Patrick up to four and a half months…[78]

We haven't found something in a phenotype which is not in the genotype or vice versa, and that would have worried us if we'd had inconsistency. We would definitely have gone back and done further testing, or worried about what that might have been, but we didn't find any inconsistency.[79]

86. There was also a question as to whether the unavailability of Mr Folbigg's DNA rendered the results of the genetic testing less reliable. The following evidence was given:

WITNESS BUCKLEY: Rather surprisingly it didn't have much effect. We did not identify any variant in the children that we were concerned about that appeared to have been inherited from Craig, and the interpretation did not hinge on his clinical state.

WITNESS KIRK: Yeah, I'd agree with that. If, if upfront we had had the option, we would certainly have preferred to do that because there is a possibility of a mechanism for which interpretation would require both parents. But in the end it didn't make any difference.

WITNESS COLLEY: Yes, I'd agree with what's being said, and I was pleased that I had had an opportunity to meet him in person, so I did know that he was of normal statute, normal intelligence and normal appearance.[80]

87. Dr Colley was also asked whether the absence of other members of the Folbigg family gave rise to uncertainty as to the phenotypes so as to render the results less reliable:

WITNESS COLLEY: No, I don't think so. I think if we had found a possibly pathogenic or likely pathogenic variant, that we wanted to trace or we say segregate through the family, then it would have been a disadvantage not to have DNA from other family members. But as such, as you've heard we didn't actually identify such a variant, so therefore we didn't need the DNA from the other family members.[81]

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78 Transcript of the Inquiry, 17 April 2019 T553.19-38

79 Transcript of the Inquiry, 17 April 2019 T554.23-26.

80 Transcript of the Inquiry, 15 April 2019 T404.28-44.

81 Transcript of the Inquiry, 15 April 2019 T404.50-405.5.

88. While genetic sequencing data from Mr Folbigg was not available, there was relevant information available about the paternal side of the Folbigg family. During the course of genetics-related consultations with Mr and Ms Folbigg in 1991, 1992 and 1993, Dr Colley met also with Mr Folbigg's sister and drew a family tree of Mr Folbigg's side of the family, including identification of the death of his mother at age 43 from a cerebral haemorrhage and his father, at that stage living, having had a heart bypass.[82]

89. Additionally, the report by Ms Folbigg to Dr Arsov of an infant death of one of Mr Folbigg's brother's children (which was referred to by Professor Vinuesa), was corrected during the course of the Inquiry to have been a death following a five and a half weeks' premature birth, with the death certificate recording the cause of death as "respiratory distress syndrome" and "prematurity".[83]

Interpretation of results

90. The findings of each of the teams were almost identical. Neither found variants in genes which were assessed as pathogenic or likely pathogenic in all four children so as to cause their sudden death.[84]

91. The Sydney report identified nine variants worthy of close examination. The diagram below, prepared by Dr Buckley, demonstrates the "filtering process" applied in the interpretation phase to identify the nine variants worthy of close examination, by reference to Patrick Folbigg's data:

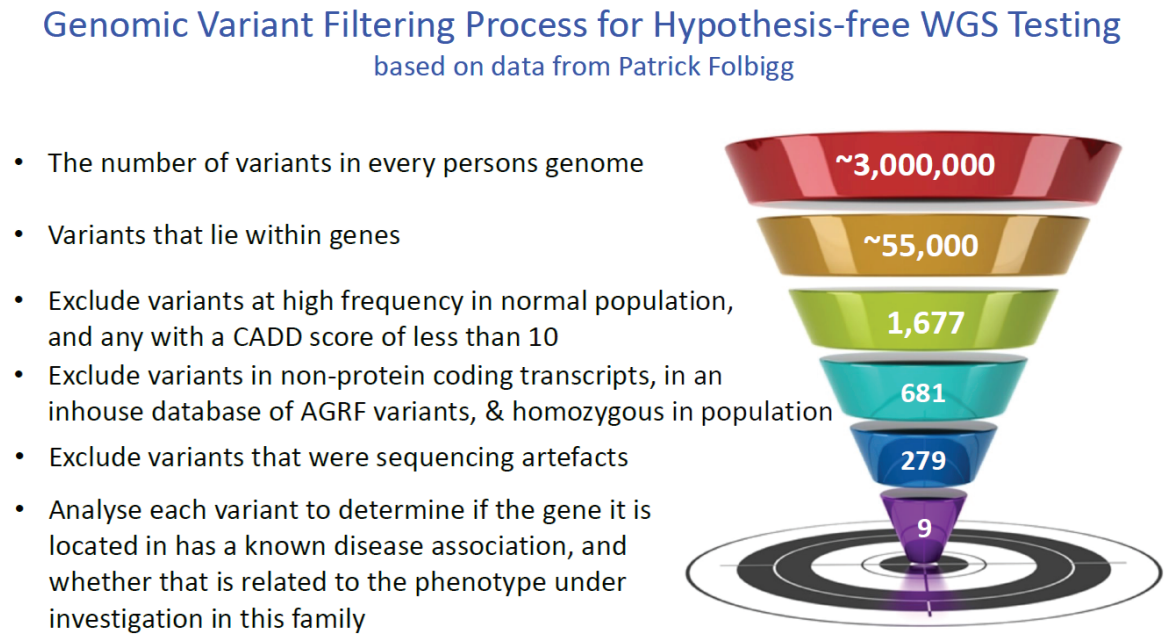

Figure 1: Diagram of variant filtering process of Sydney genetics team: Exhibit AC of the Inquiry

[82] Exhibit AC, Genetics tender bundle, Notes of the Regional Medical Genetics Unit for Newcastle and Northern NSW (12 November 1991 – 5 November 1992) pp 1-6.

[83] Exhibit BG, Statement of Craig Folbigg (19 April 2019); Transcript of the Inquiry, 17 April 2019 T611.48-612.13.

[84] Exhibit Z, Joint report of Sydney genetics team (29 March 2019) p 8; Exhibit AF, Joint report of Canberra genetics team (29 March 2019) p 13.

92. After analysis, five of the nine variants were deemed by the Sydney report to be variants of uncertain significance (including CALM2), two were considered benign and one was classified as likely benign. The final gene the Sydney report determined had no definite association with a disease (MYH6). The report concluded none of the variants identified were deemed causal for the phenotype in the children.[85]

93. The Canberra report identified two variants as being likely pathogenic (IDS, found in Patrick, and CALM2, found in all but Patrick and Caleb), and one borderline variant of uncertain significance or likely pathogenic (MYH6, found in all but Sarah), which were missense novel or ultrarare variants that could contribute to the observed phenotypes found in the children.[86]

94. Professor Kirk gave the following reason for these three variants being interpreted slightly differently by the Sydney and Canberra teams:

> *I think this highlights the difference of approach. Professor Vinuesa is a very experienced and eminent researcher and is, I guess, approaching this in the way that you might approach a research project, thinking about possibilities, expanding the different, the different areas of knowledge that we currently have. Whereas our approach is more focused on known disease associations.*[87]

Cardiac variants generally

95. Professor Skinner gave evidence about the "top" gene variants which were not identified as present in any of the children and which have been associated with sudden infant death:

> *Genes that have been associated with sudden infant death. For example, SCN5A, sodium channel disease, this is not found here. Triadin, autosomal recessive, this causes severe disease, could potentially cause cardiac death in infancy. CACNA1C, that's not here and caveolin is another one. So the four top genes that I came into, in terms of causing infant - sudden infant cardiac death and no, no significant variants, no variants have been produced in this list and I find that an important thing to document at this stage.*[88]

96. Professor Skinner concluded that the available clinical phenotype data and genetic analyses in respect of the children and Ms Folbigg provide no convincing evidence for the presence of any known form of cardiac inherited disease as a potential cause for the sudden death of the four children.[89]

Ms Folbigg's cardiac phenotype following completed investigations

97. The experts ultimately had available to inform their interpretation results from the following investigations undertaken in respect of Ms Folbigg's cardiac clinical presentation:[90]

    a. variously dated clinical notes relating to "syncopal episodes", GP progress notes and cardiologist reviews;

    b. ECG dated 31 January 2011;

    c. ECG dated 17 May 2011;[91]

    d. ECG dated 24 December 2018;

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85 Exhibit Z, Joint report of Sydney genetics team (29 March 2019) pp 8-9.
86 Exhibit AF, Joint report of Canberra genetics team (29 March 2019) p 13.
87 Transcript of the Inquiry, 16 April 2019 T514.38-43.
88 Transcript of the Inquiry, 15 April 2019 T450.5-12.
89 Exhibit Y, Report of Professor Jonathan Skinner (31 March 2019) p 10.
90 Exhibit AC, Genetics tender bundle, Material Relating to Kathleen Folbigg (various dates), tabs 34-59.
91 Exhibit AH, ECG of Kathleen Folbigg (17 May 2011).

e. chest x-ray report dated 27 December 2019;

f. clinical (including historical clinical information) consultation dated 20 February 2019;[92]

g. echocardiogram report dated 22 February 2019;

h. postural ECG and exercise test dated 18 April 2019;[93] and

i. 24 hour ambulatory Holter monitor test dated 18-19 April 2019.[94]

98. The 2019 investigations were undertaken by Associate Professor Hariharan Raju, a cardiologist and electrophysiologist at the Macquarie University Health Sciences Centre. Associate Professor Raju expressed the following opinions upon conclusion of his investigations:

> *With respect to further investigation of Kathleen, my opinion is that the likely yield of cardiac pathology will be negligible. Given her as yet recurrent unexplained syncope, prolonged heart rhythm monitoring with implantable loop recorder may be prudent if her 24-hour ambulatory Holter monitor fails to document any pathological arrhythmia. This offers potential for symptom-rhythm correlation (or absence thereof). Additional investigations may be performed if clinical suspicion of cardiogenetic disease changes in the future…*
>
> *In summary, following comprehensive non-invasive evaluation, Kathleen has no phenotypic evidence of either cardiomyopathy or primary arrhythmia syndrome. The only abnormality detected is the presence of possible exertional ventricular ectopy which is consistent with an idiopathic focus and likely of no clinical relevance. The borderline repolarisations changes seen on her resting ECG in 2003 are also not diagnostic of pathology. Her multiple syncopal episodes are likely to be of reflex aetiology, which is benign.*[95]

99. Professor Skinner reviewed the raw results of the investigations conducted by Associate Professor Raju and concluded as follows:

> *If we look at this as the evidence as we know it at this stage:*
>
> *Kathleen is over 50 and has not had a cardiac arrest*
>
> *Assessments suggests she has never had what would be a likely arrhythmic syncope*
>
> *Her four children all died during sleep*
>
> *Her four children all died at an age before CPVT presents*
>
> *She has had two exercise tests with no symptoms and no ventricular arrhythmias, other than a small number of isolated ventricular ectopic beats on one of them.*
> *She does not have a recognised genetic variant linked to CPVT, in particular none in RyR2 or CASQ2…*
>
> *The exercise test shows a good heart rate and blood pressure response and average exercise tolerance. The test is within normal for age.*

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92 Exhibit BL, Report of Associate Professor Hariharan Raju (18 April 2019) p 2.
93 Exhibit BH, Further cardiac testing of Kathleen Folbigg (18 April 2019).
94 Exhibit BH, Further cardiac testing of Kathleen Folbigg (18 April 2019).
95 Exhibit BL, Report of Associate Professor Hariharan Raju (18 April 2019) pp 2, 3.

> *The QT behaviour on the standing tests is within normal, and also in recovery of the exercise test though defining the end of the T wave is challenging due to the T wave distortion from the mild ST segment changes, which tends to prolong the T wave. The appearances of the T waves are not suggestive of long QT syndrome.*
>
> *The apparent large deflections are mostly if not all artefact, and I discount, in particular, the apparent VE triple and couplet as evident artefact, most likely from leads becoming partly detached. Most of the apparent single VEs also appear at a time when there are many other artefacts and with the exception of the first ventricular ectopic type, which appears twice on the traces, I consider that these broad deflections can be discounted as artefact.*
>
> *Therefore I find no evidence of long QT syndrome, and no evidence of significant ventricular arrhythmia. There are a small number of single ventricular ectopic beats, common on exercise tests in people of this age and which are not likely to be of clinical significance. The ECG modified to detect Brugada syndrome was negative.*[96]

100. Professor Vinuesa did not agree that the results of the cardiac investigations into Ms Folbigg that she had originally recommended would necessarily enable her to come to firmer conclusions about the cardiac variants she interpreted in her reports. In the course of her evidence, Professor Vinuesa suggested, for the first time, that it would be "ideal" if Ms Folbigg additionally underwent a drug induction test, though said she would defer to a cardiologist on this issue.[97] Ultimately the basis for her view that the test results would not be sufficient to enable firmer conclusions was that the tests would not exclude the possibility of Ms Folbigg being a "non-penetrant" (not yet manifested) carrier of a pathogenic variant.[98]

101. Professor Skinner recommended no further testing. In respect of an "epinephrine challenge", or drug provocation testing, involving the infusion of adrenaline by a standardised protocol to see if ventricular arrhythmias are induced as with an exercise test, he said he would not recommend it and it would not be recommended current clinical practice in the current scenario.[99]

102. Associate Professor Raju also opined that epinephrine testing: "remains controversial" in the diagnosis of CPVT and long QT. CPVT is another heart rhythm disorder associated with sudden death. He considered "in the absence of other clinical indicators of the diagnosis, I am not certain that it will offer any additional diagnostic yield".[100]

CALM2

103. CALM2 was present in Sarah, Laura and Ms Folbigg and not present in Caleb and Patrick. The clinical presentation most commonly associated with the gene is a severe form of long QT syndrome.[101] Long QT syndrome is a heart rhythm disorder indicated by a long QT interval measured on ECG tests. The condition can cause sudden syncope (loss of consciousness), sudden cardiac arrest and sudden death.[102] In very rare instances the clinical presentation of catecholaminergic polymorphic ventricular tachycardia ("CPVT") has been observed. CPVT may not be indicated on resting ECG tests, but rather on exercise ECG tests.[103]

96 Exhibit BJ, Further report of Professor Jonathan Skinner (24 April 2019) pp 3-4; Exhibit BK, Letter from Professor Jonathan Skinner to the Inquiry (30 April 2019) p1.

97 Transcript of the Inquiry, 16 April 2019 T503.37-42.

98 Transcript of the Inquiry, 16 April 2019 T503.25-504.5.

99 Exhibit BK, Letter from Professor Jonathan Skinner to the Inquiry (30 April 2019) p 1; Exhibit BJ, Report of Professor Jonathan Skinner (24 April 2019) pp 3-4.

100 Exhibit BL, Report of Associate Professor Hariharan Raju (18 April 2019) p 2.

101 Exhibit Z, Joint report of Sydney genetics team (29 March 2019) p 12.

102 Transcript of the Inquiry, 15 April 2019 T388.1-5, T397.38-48,T454.24-30.

103 Transcript of the Inquiry, 15 April 2019 T393.24-30; T395.49-396.1, T454.25-30.

104. Professor Kirk gave the following evidence:

> *WITNESS KIRK: … there are some situations [in families reporting CPVT] where there are parents who are either affected in a minor way or who are healthy but have abnormalities on stress testing or on other, other provocative testing. In those families, the age of onset of symptoms is much later and the earliest death, I think, is at about four years for any of those, and most of the deaths have been in the range of 10 to 16 years. So, for those - for those particular ones, it's, it's a different condition. It's still a severe condition, but not as severe as the, the more commonly seen situation.*
>
> *So, I think that's an important difference because we would expect that if someone had a condition that was severe enough to cause death in the first months or in the first year or two of life that that would be one of the more severe manifestations. That's certainly the universal experience in the literature so far. Whereas, if someone were surviving longer term, they could conceivably have the less severe form but we would not expect death in infancy…*[104]
>
> *The way that manifests in people is that the problem occurs in an awake state, typically during exercise but sometimes in response to a startle or occasionally, in strong emotional situations, but exercise. The classic story is of a ten year old who is swimming in a swimming carnival and he sinks like a stone to the bottom of the pool…*[105]
>
> *Kathleen Folbigg does not have clinical features that would be consistent with any of the known manifestations of the condition. It is true that we could not exclude the possibility of [CPVT] in her without additional testing, but then that would not be consistent with infant deaths in the family because it's a less severe form of the condition, and also, as I say, associated with death while awake, usually during exercise…*[106]
>
> *And yes, it is conceivable that there could be something that is completely outside the experience we've had so far, but I think we're addressing current knowledge and within current knowledge I think I feel quite strongly that we can apply BS2, which makes this a variant of uncertain significance, because we've got conflicting evidence, and one where the weight of evidence is against it being pathogenic, but I accept that there are limitations to our knowledge and it's not inconceivable that this could prove to be relevant to the death of two of the children. I think very unlikely but not inconceivable.*[107]

105. Professor Vinuesa's evidence at its highest was that she "would not feel comfortable with excluding its potential for pathogenicity" on the basis that it is "conceivable" that the mutation in Ms Folbigg was non-penetrant.[108]

106. Professor Skinner considered it to be "stretching credibility" that the CALM2 variant identified in the Folbigg family could be pathogenic. He gave the following evidence:

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104 Transcript of the Inquiry, 16 April 2019 T469.42-470.7.
105 Transcript of the Inquiry, 16 April 2019 T470.12-16.
106 Transcript of the Inquiry, 16 April 2019 T471.8-13.
107 Transcript of the Inquiry, 16 April 2019 T482.16-23.
108 Transcript of the Inquiry, 16 April 2019 T476.10-16; T479.1-10.

> *Yes, of course Professor Vinuesa is correct, that all of these inherited heart conditions have variable penetrance and sometimes expression. But the penetrants issue really is covered quite well with the literature on this gene. It's - degree of penetrants, its likelihood of causing disease has been in the literature to date related very much in the way that Dr Kirk said, if it's a de novo mutation you have severe disease which might cause a lethal outcome in the first year or two. If you have familial disease, there's not a single case of a sudden death under the age of two. All of the deaths produced in the literature have been over the age of two and the majority of those are in teenagers or above during exercise, while awake, not while asleep.*
>
> *So not only is the phenotype wrong, but the penetrance issue I think really is covered by the literature so far. Is it possible that there would be some – that this particular variant might behave differently? It's stretching credibility but I imagine it's not impossible, but it doesn't follow the pattern of disease established to date.*[109]

107. Professor Skinner gave a further reason for not assigning this variant as being likely pathogenic: that it has not appeared in any study of SIDS victims.[110]

108. On the basis of the results of exercise testing conducted on Ms Folbigg and received after the oral evidence, Professor Skinner concluded he was confident Ms Folbigg does not have CPVT and found no evidence of long QT syndrome or significant ventricular arrhythmia.[111]

MHY6

109. The MYH6 variant was present in Ms Folbigg, Caleb, Patrick and Laura Folbigg. The MYH6 variant was not present in Sarah Folbigg.

110. Professor Kirk gave evidence, with which Professor Skinner agreed:[112]

> *So, interpretation of this variant, the starting point has to be understanding the relationship between the gene and a relevant condition... Now, there is a great deal of evidence linking changes in this gene to congenital heart disease and also to cardiomyopathy. But we know that the children do not have congenital heart disease and nor does Kathleen and there is no evidence of cardiomyopathy on any of the post-mortems. In any case, it would not present at this very early age, and there's no evidence on echocardiogram in Kathleen Folbigg of a cardiomyopathy. So, those phenotypes are not really relevant.*[113]

111. Professor Vinuesa was asked whether she would defer to Professor Kirk's opinion, given their respective areas of expertise:

> *WITNESS VINUESA: Yes, with one comment. That, again, many of these cardiac problems manifest for the first time with sudden unexpected death and may be autopsy negative. So, with that, I still think we have to take into consideration variable expressivity and the potential for alternative pathogenic mechanisms.*[114]

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109 Transcript of the Inquiry, 16 April 2019 T478.20-36.

110 Transcript of the Inquiry, 16 April 2019 T474.29-41.

111 Exhibit BK, Letter from Professor Jonathan Skinner to the Inquiry (30 April 2019) p 1; Exhibit BJ, Report of Professor Jonathan Skinner (24 April 2019) pp 3-4.

112 Transcript of the Inquiry, 16 April 2019 T487.1-23; Exhibit Y, Report of Professor Jonathan Skinner (31 March 2019) p 9.

113 Transcript of the Inquiry, 16 April 2016 T483.3-12.

114 Transcript of the Inquiry, 16 April 2019 T487.28-32.

IDS

112. The Sydney Team excluded IDS from consideration primarily on the basis that there was strong evidence that Patrick, the only person with the variant, did not have the condition to which it relates, namely Hunter syndrome. In its supplementary report, the Canberra Team stated that "we would defer to a metabolic disease specialist on this matter".[115] Professor Vinuesa was asked about the statement and gave evidence, "I think it would be good to consult with a metabolic expert for sure."[116]

113. Professor Kirk, a metabolic disease specialist, provided this explanation of the variant:

> *This relates to a condition called Hunter syndrome, which is a condition in which there is abnormal storage of material in a component of the cell called the lysosome and it's one of a group of, of lysosomal storage disorders. And the effect of this progressive accumulation of material is both enlargement of the tissues that are involved, but also damage to the function of some of the organs, particularly the brain.*
>
> *This is a condition which is not always clinically obvious in the first year of life, although there may be features present as early as birth, but they are generally not the most distinctive features of the condition.*
>
> *The reason that we felt confident in excluding this from consideration is that a very – two very sensitive biochemical tests had been done which were not consistent with the diagnosis and we were aware of that information. So, we, we, deemed that it did not need further evaluation… in addition, we had post mortem evidence that it was not consistent with the diagnosis…*
>
> *And then, lastly, I would say that, as far as I can tell, none of the information I received about Patrick in any way connects this condition to the events of his life and death…*
>
> *Look I think, possibly, your Honour, I should walk back slightly on what I said. I think there is a very remote possibility that this child had Hunter syndrome. My confidence is more about whether this was the cause of his death. I'm extremely confident that this was not the cause of his death. So, I think it's very unlikely he had the condition and if he did, then it would not have been the cause of his death.*[117]

Other variants

114. Professor Vinuesa gave evidence that there were three other variants in relation to which there was a "theoretical possibility" that she was not prepared to rule out of causing the phenotype seen in the children: DMPK, ADAMT56 and SCLC12A9.[118]

115. The Sydney Team did not consider any of the variants causative of the phenotype, largely because there was no evidence linking the genes to human disease.[119]

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115 Exhibit AY, Written response from Professor Carola Vinuesa and Professor Matthew Cook to written response of Sydney genetics team (12 April 2019) p 5.

116 Transcript of the Inquiry, 16 April 2016 T489.29-30.

117 Transcript of the Inquiry, 15 April 2019 T424.41-425.11, T427.28-30; 17 April 2019 T562.41-46.

118 Transcript of the Inquiry, 16 April 2019 T499.48-500.14.

119 Transcript of the Inquiry, 16 April 2019 T499; Exhibit AX, Response from Professor Kirk and Dr Buckley to joint report of Canberra genetics team (9 April 2019).

Limitations given advances in genetics

116. The expert panel was asked about the limitations of the work they undertook given the rapidly progressing nature of the science.

> *WITNESS COLLEY: I think the likelihood, even in a decade's time that we would find something startlingly different is low, because of the Whole Genome Sequencing techniques that have been used and the quality of the data that we have been told about. Now in saying that, there is clearly going to be new technology and new interpretation, but at this stage, looking to the future as much as we can, I am not envisaging that we're going to have to redo all this in a different way…*
>
> *Now our genomes aren't going to change that much, I don't think. I mean, there is natural selection, but I don't think we're going to see a change in the genome and we've done the test hypothesis-free to interrogate the genome as much as we can.*[120]
>
> *WITNESS BUCKLEY: … this is a multifaceted test with clinical components and laboratory components and interpretive components. The clinical component is going to be the same in another five years. The features of these – of this family is of well grown, developmentally normal children who have a sudden and catastrophic event, but without many features of a genetic disorder of early childhood onset. That clinical setting, together with the power of the Whole Genome Sequencing result in combination, I think means it is very unlikely that despite the advances and we will expect that there will be new diseases, but I think that the new diseases that are discovered are not going to be relevant to this clinical situation. So I - anything is possible but in my professional opinion I think that the likelihood in this particular situation is quite low.*[121]
>
> *WITNESS KIRK: Yeah, I'd concur with my colleagues…*
>
> *From what I've seen during my career, I think I agree, that it's, it's very unlikely that we're going to identify something in the future that will, that will explain this.*[122]
>
> *WITNESS SKINNER: Yes. The principles aren't really going to change, I don't think. If you have four very young children who have a catastrophic event, then the parent, if they carry the same genetic marker, would not be expected to be alive…*
>
> *we could come back here in ten years and have this same conversation. I think - this is really up for the Court to decide but we can speculate forever about what might be and what might happen and what experiments in mice might mean for the human being. Right now all we can look at what we know now or what we have reasonable confidence in knowing now and I, I think we're going to end up in, in a circular conversation unless we agree what the endpoint is here. I, I think the ideas that are put forward by Professor Vinuesa's team are great. It's, it's, it's a good thing to think laterally and to think wisely, multigene inheritance and so on, but at this stage we just don't have enough information about that to make meaningful judgments in, with the current knowledge of phenotype genotype data.*[123]

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120 Transcript of the Inquiry, 15 April 2019 T432.46-433.2, T433.13-16.

121 Transcript of the Inquiry, 15 April 2019 T433.20-31.

122 Transcript of the Inquiry, 15 April 2019 T433.35, T433.41-43.

123 Transcript of the Inquiry, 15 April 2019 T433.48-50, T434.1; 16 April 2019 T524.9-20.

> *WITNESS BUCKLEY: … It's very unlikely that we're going to be able to retest all of these samples using a putative technology that comes along in another five years. I think the data we have are reliable. I think the very fact that our Canberra colleagues and ourselves analysed these data, using different approaches, similar, using different models, but very largely we came up with a very similar set of variants that we thought were plausible, that we were confident in, that we thought should be considered as part of this matter. I, I don't see that, that we're going to come up with a very substantially different view into the future unless there is some radical change in sequencing technologies in the next few years. We have what we have. These are the data that we are best able to explain.*
>
> *They seem to be consistent between two groups by and large and where we depart is where – it's the different weighting and interpretation that we put on those, and I think to a degree some of the analysis reflects, says more about ourselves perhaps than about the data, that it reflects our different views. I think together the data presented by Professor Vinuesa, the data presented by us, are a remarkable snapshot of the genetics of this family at this time which we are trying to understand in the light of current knowledge.*[124]
>
> *WITNESS VINUESA: I agree that in terms of technology, we will probably not come up with a substantial number of different variants, but we are only analysing 1% of the genome, we have not even considered 99% of the non-coding mutations. We know that – we have agreed that 50% of genetic conditions cannot be diagnosed today – of monogenic genetic conditions, and the expectation is that as soon as we have better tools to explore the significance of structural variants, other missense mutations in enhancers or cryptic supplies in sites throughout the genome might give us a, a whole new list of variants to look at.*
>
> *Also, we are limited by current knowledge of genes and their function. We still don't understand how at least one third of the genes in the genome work or what their function is, so I expect that over the next few years there will be more genes that will have been implicated in cardiac disease, there will be more variants. So, I think the interpretation can significantly change in a few years, not the raw data. I agree with you, the technology will not change, the raw data will not change, but we will make better sense of it in a few years.*[125]

117. Professor Vinuesa additionally raised as a limitation that the analysis undertaken by the experts did not contemplate the possibility of "digenic causes". She gave the following evidence:

> *WITNESS VINUESA: Look, I would like to make a comment. I think we are only contemplating the most simple scenario of the single gene causing disease. There is increasing evidence of digenic causes of disease. We've dealt with many, you probably have as well. When we have digenic causes, two genes coming together, first, the frequency of each of those doesn't need to be so ultra-rare, so we can cope with frequencies like the one we've just talked about for this ADAMTS6. Furthermore, there is good evidence that even common variants can substantially modify the incidence of disease, and if I may quote one, "Crotti et al have provided evidence that the common polymorphism KCNH2 (30% carrier amongst whites – 30% carrier frequency) may modify the clinical expression of latent LQT2 mutation."*

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[124] Transcript of the Inquiry, 16 April 2019 T529.7-25.

[125] Transcript of the Inquiry, 16 April 2019 T529.30-46.

> *So we can have either different genes, two that, rare variance, one common and one rare, many common. If you look at the pedigree we have in our screens we have quite a lot of variance that could be coming together to cause part of this disease, and then in those cases the frequency changes, we just can't say that because the frequency is not that rare we have to exclude a variant.*[126]

118. In response to Professor Vinuesa's view about digenic causes, Professor Skinner explained that:

> *at this stage we just don't have enough information about that to make meaningful judgments in, with the current knowledge of the phenotype genotype data.*[127]

119. Professor Kirk expressed a similar sentiment and said "we're a long way off from being able to interpret that kind of information usefully."[128]

Submissions on genetics

120. Submissions on genetics were received from counsel assisting and Ms Folbigg.

Submissions of counsel assisting

121. Counsel assisting submitted I could be satisfied that the samples obtained, the methodology used and the processes followed resulted in good quality data.[129]

122. It was also submitted I could be further satisfied that the process of filtering and prioritising the variants using:

    a. hypothesis free analyses;
    b. literature and database searches for sudden unexplained death in infancy, cardiac conditions and epilepsy;
    c. gene panel analyses on genes associated with:
        i. sudden death in infancy/childhood;
        ii. childhood neurological disorders;
        iii. immunology; and
        iv. metabolics;
    d. any variant annotated as pathogenic or likely pathogenic related to any phenotype; and
    e. chromosomal microarray analysis,

    has resulted in there being no reasonable possibility that the children had a known pathogenic or likely pathogenic variant which caused their death which was not identified by this exercise.[130]

123. Further, it was suggested I could be satisfied that the absence of a sample from Mr Folbigg, and the limited information available about other members of the Folbigg family, did not lead to uncertainty in the phenotype or about a particular variant and therefore detrimentally affect the reliability of the results.[131]

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126 Transcript of the Inquiry, 16 April 2019 T520.37-521.4.
127 Transcript of the Inquiry, 16 April 2019 T524.16-20.
128 Transcript of the Inquiry, 16 April 2019 T521.14-31.
129 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [63].
130 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [63].
131 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [64].

124. Counsel assisting noted there were three variants about which the Sydney and Canberra teams differed in their interpretation.[132] Each concerned matters within the expertise of clinical geneticists and cardiologists. It was submitted that Professor Vinuesa properly conceded Professor Skinner's expertise in relation to MHY6 and Professor Kirk's knowledge and training in IDS and deferred to them in respect of those variants. It was said the expertise of Professors Kirk and Skinner should also be accepted and preferred in the interpretation of CALM2.

125. It was submitted that the variants about which Professor Vinuesa considered a "theoretical possibility" existed of being pathogenic cannot by definition be considered to represent a reasonable possibility of causing the death of any of the children.[133]

126. Accordingly, in counsel assisting's submission, I should be satisfied that no variant was identified as being pathogenic or likely pathogenic in relation to the Folbigg children. It was suggested it follows that there is no reasonable possibility that the death of any of the Folbigg children was caused by a recognised genetic variant.[134]

127. Counsel assisting acknowledged that the testing that was carried out is necessarily defined by the data identified and the processes and knowledge currently available.[135] It was suggested that the proposition that tomorrow more may be known and therefore there is a reasonable possibility that the children died from an as yet unrecognised genetic cause must be rejected. In this regard it was noted that the phenotype will not change and the genome will remain unaltered.

Submissions of Ms Folbigg

Genetics at trial

128. Ms Folbigg's submissions suggested there were two problems with how the issue of genetics and genetic testing was approached at trial.

129. First, it was submitted there was confusion at trial "between a familial disorder covering the four children, and an individual disorder which may have triggered the death in one of the individuals".[136] In this regard, Dr Cooper's evidence was referenced. Dr Cooper was asked "what can you now say about the question of familial or genetic links?" and answered "the likelihood of a second SIDS in a family whose [sic] had one is probably no higher than in the general population".[137]

130. This evidence was said to "fail to distinguish between SIDS and sudden death". The submissions suggested, by reference to Professor Blackwell's evidence about the link between the success of the Back to Sleep campaign and the identification of infection as being a cause of death, that "the issue of cause of death is multifactorial" and accordingly "[t]here may be an interplay between genetics and infection".[138]

131. Secondly, it was submitted there was a fundamental flaw in the argument by the Crown that the genetic testing performed had excluded a genetic cause. In support of this characterisation of the Crown case, the submissions pointed specifically to Dr Wilcken's evidence that the results of the genetic testing conducted on blood spots from each child were "entirely normal", and that many genetic or metabolic disorders tested for in Patrick's urine sample could be "ruled out utterly".[139]

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132 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [65].
133 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [66].
134 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [67].
135 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 6, [68].
136 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [10]-[13], [21]-[22].
137 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [9]-[10]; 14 April 2003 T608.6-10.
138 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [12].
139 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [19]-[20].

132. Additionally referred to was the Crown prosecutor's closing address where he stated "Dr Kan's opinion excluded effective causes of death, metabolic causes of death, genetic disorders",[140] and to the trial judge's summing-up in respect of Caleb:

> *You will remember the evidence of Dr Wilcken that tests were carried out on the blood of Caleb which had been preserved, and a large number of possible natural causes of death had been excluded, so that many or all, of the likely candidates as a cause of death, by way of infection or metabolic or genetic causes, were excluded.*[141]

133. Ms Folbigg submitted that the excerpts referred to were misleading because they suggested that any doubt raised by a potential genetic link could be put to one side.[142] This was said to demonstrate a lack of understanding of the limits of genetic science at trial, and whether it was possible to positively exclude a genetic cause of death in an unqualified manner.[143]

Genetics in the Inquiry

134. Ms Folbigg submitted that the "fallacious reasoning" used at trial was also used in the Inquiry when assessing the genetic information obtained by WGS and WES.[144] It was suggested that counsel assisting's submissions conflated:

> *the absence of any recognised genetic cause (which leaves open doubt about future developments as further information comes available) with the proposition that genetic causes have been excluded*

and that such an assessment "carries a presumption of guilt rather than a presumption of innocence or even a neutral assessment".[145]

135. Ms Folbigg's submissions included only very limited discussion as to the relevance and significance of any of the genetic variants actually identified in the children and Ms Folbigg, or of the phenotype of the children and Ms Folbigg, to the assessment of cause of death. The submissions for the most part focussed on what was not in evidence in the Inquiry, and what is not yet known in the field of genetics.

136. In this regard the submissions advanced a series of criticisms which may be categorised as about the scope of genetic investigations undertaken by the Inquiry; the methodology and reasoning applied by the experts engaged by the Inquiry to interpret the available data; and the ultimate opinions proffered by those experts. These criticisms were relied on in support of the ultimate submissions that:

    a. the genetic evidence before the Inquiry is "of limited utility in excluding a reasonable doubt when this Inquiry comes to exercise its discretion";

    b. a genetic or non-genetic cardiac cause of death in one or more of the children has not been excluded;

    c. there is still incomplete medical knowledge and

> *the experts are expecting an avalanche of material that may shed new light on the association between monogenic and digenic variants and disease and the interaction between genes and infection and other exogenous causes*; and

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140 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [26].
141 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [26]-[27]; 19 May 2003 T19.
142 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [30].
143 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [32].
144 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [37].
145 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [105].

d. the lack of genetic material and cardiac functioning tests provides a further impediment to assessing the likelihood of a cardiac cause for the deaths of one or more children in the Folbigg family.[146]

137. As to the scope of investigations undertaken by the Inquiry, it was submitted:

a. "the appropriate three generation genetic assessment" was not performed as was recommended by Professor Vinuesa and the forensic pathologists. This would have involved an assessment of three generations on both sides of the family, including Ms Folbigg's natural parents and siblings and extended family, and Mr Folbigg and his extended family. The significance of this was that:

> *these children may well have a genetic disorder which is incapable of being identified until such time as a proper genetic assessment has been performed.*

It was submitted the more extensive family assessment was "the only way to have greater confidence to exclude genetic disorders";[147]

b. the geneticists did not have access to any genetic material from Mr Folbigg or his side of the family and it is clear from the ACMG Guidelines that a de novo mutation may well be pathogenic;[148] and

c. Professor Vinuesa recommended a number of alternatives tests that should have been performed in order to come to a clearer picture regarding pathogenicity including in vitro and functional studies that were not performed by the Inquiry.[149]

138. As to the methodology and reasoning applied by the experts engaged by the Inquiry to interpret the available data, it was submitted:

a. the "hypothesis-free" approach employed by Dr Buckley and the Sydney Team, applying the ACMG Guidelines, resulted in the interpretation exercise becoming constrained because it focussed upon "known" and "recognised" conditions that could cause sudden infant death. Professor Vinuesa did not agree with this approach and the difference reflected the difference between clinical practice and research;[150]

b. the approach used by Dr Buckley and the Sydney Team did not address all potential genetic disorders within a family. The preferred approach would have been to interrogate the family "to identify the common genetic abnormalities within the family that would explain the disease process within that family";[151]

c. whether a genetic anomaly is currently classified as "pathogenic", "likely pathogenic" or as a "variant of uncertain significance" "is not to the point as to whether a specific genetic anomaly is causative in the strict medical sense";[152]

d. the examination conducted by the Inquiry was limited to a monogenic cause and did not address a digenic cause, a multi-genetic cause or gonadal mosaicism. A monogenic cause will only be found in between two to 20 per cent of SIDS cases, and there is "growing evidence" of digenic causes contributing to disease;[153]

e. there was no information advanced in the Inquiry about genetic anomalies and environmental factors such as infection or pollution which could trigger sudden infant deaths;[154] and

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146 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [82], [133].
147 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [40]-[44], [47]-[49], [54]-[56].
148 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [60].
149 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [79].
150 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [50]-[51].
151 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [38]-[40], [47]-[48], [50].
152 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [57].
153 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [64], [67], [73], [76], [107].
154 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [77].

f. in determining the phenotype of the children, the geneticists assumed the children were previously normal, well children who died a sudden death. This made a significant difference to the determination of whether a variant was classified as being of uncertain significance or likely pathogenicity.[155]

139. As to the utility of the ultimate opinions offered by the experts in the Inquiry it was submitted that the understanding of genetic mutations that can cause or contribute to sudden death is not yet complete and research is continually being published. This means geneticists have to work with the information they have and the testing conducted by the Inquiry reflects the current limits of scientific understanding.[156]

140. It was further submitted there is currently limited understanding of the interaction between genetic mutations and environmental circumstances, medications, physiological processes such as dehydration and electrolyte imbalance and infections and the possible relationship with sudden unexpected death; these are possibilities that may prove in time to be extremely important factors.[157]

141. By way of example of the "massive amount of information which is being discovered… and reflects the practical limitation of today's knowledge", a 2019 report suggesting for the first time a link between the ADAMTS6 variant which was identified in each of the children and "the phenotype" was referred to.[158]

Genetics specifically regarding Patrick

142. Ms Folbigg submitted that Patrick had "Hunter syndrome genetic variants" which were capable of triggering his ALTE and death. It was noted that although Professor Kirk thought it was unlikely that Patrick had Hunter syndrome, he said that further tests would provide clarity.[159]

143. In noting that the Hunter syndrome variants were not classified as pathogenic by the Sydney Team, it was submitted that the clinical history, family information and further information from the scientific literature may "trigger reclassification" of this variant as pathogenic. In this regard it was noted that there remains an association between Hunter syndrome and cardiac arrhythmias, which was said to demonstrate the possible interaction between two diseases.[160]

144. It was also submitted that despite Associate Professor Fahey stating that "[a]ll of those with both ALTE and epilepsy have a family history" there was no study of the Folbigg family to determine whether there was a family history of ALTE, epilepsy or infantile or premature death.[161]

Genetics specifically regarding a cardiac cause of death

145. It was submitted by Ms Folbigg that a lack of "genetic material" and cardiac function tests were impediments to assessing whether there was a cardiac cause of death for one or more of the Folbigg children.[162]

146. In submitting that cardiac conditions in Ms and Mr Folbigg, and a cardiac cause of death in one or more of the children caused either by a monogenic variant, a digenic variant, or a combination of variants(s) and infection or environmental trigger, had not been eliminated by the results of the cardiac function tests on Ms Folbigg, it was noted:

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155 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [63], [70].
156 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [56], [77], [80].
157 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [57].
158 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [71].
159 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [86].
160 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [90].
161 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [95]-[96]; Exhibit AK, Report of Associate Professor Michael Fahey (30 March 2019) p 13.
162 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [133].

a. a drug provocation test "which is part of electrophysiological practice" to determine whether an abnormal heart rhythm can be triggered was not done "due to time constraints";

b. there was no cardiac function examination of Mr Folbigg;

c. in a certain number of patients, they may still suffer from long QT syndrome despite having a normal ECG (such that the variant is "non-penetrant"); and

d. the cardiological opinions offered to the Inquiry were predominantly directed to the identification of single common cardiological disease and did not assess "digenic causes of any cause associated with a known genetic variant and infection".[163]

147. Specifically, in relation to Laura, for Ms Folbigg's submissions emphasised Professor Skinner's opinion that "the ECGs available are not of a quality whereby a cardiac channelopathy can either be diagnosed or excluded."[164]

Response to submissions of counsel assisting

148. Ms Folbigg's submissions took issue with counsel assisting's characterisation of Professor Vinuesa's opinion as to the possibility of variants identified by her being pathogenic as a "theoretical possibility".

149. It was submitted Professor Vinuesa had demonstrated a valid difference of opinion on pathogenicity and raised a "demonstrated issue of interactions between genes that can demonstrate pathogenicity", by reference to her own recent experience of four deaths in one family. It was suggested I could not dismiss Professor Vinuesa's evidence without some cogent and rational analysis, or "without specialist expert opinion from experts in the same field".[165]

150. It was then submitted the following remain "real possibilities" which give rise to a reasonable doubt as to Ms Folbigg's guilt:

a. there is a single underlying genetic condition in each of the four children, that may explain four deaths in the one family;

b. in any one of the children, there is a genetic condition that could trigger sudden infant death;

c. there could be two or more gene variants that could have triggered the death or deaths; and

d. there could be an association between a genetic variant and an exogenous stressor, such as infection or environmental factors.[166]

Findings

Genetics-related evidence at the trial

151. I do not consider the evidence in the Crown case at the trial, or the Crown's reasoning in respect of that evidence, was fallacious. The evidence was clear that only a certain range of known or recognised genetic and other disorders had been tested for and confidently excluded. I note those results were confirmed in evidence before the Inquiry in 2019. The evidence at the trial was also clear that genetic testing was a rapidly evolving area of medical knowledge.

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163 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [116]-[119].
164 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [130].
165 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [110].
166 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part B, [111].

Advances since the trial

152. I find that significant advances relevant to the Inquiry's task have been made since the trial in the field of genetics. These advances permit a much broader scope of investigation than was possible in 2003. In particular, genetic sequencing technologies enable examination of a person's whole genome which is accessible, and a person's whole exome (one to two per cent of the genome).[167] As a consequence of advances in sequencing, advances have also been made in the rate of discovery of genes responsible for genetic disorders.

153. Together, these advances permit hypothesis-free study of a person's DNA, and interrogation of a person's genetic variants against the known healthy human genome and the phenotype or clinical features of a person.[168]

Genetic sequencing undertaken by the Inquiry

154. I find that the DNA samples utilised and the sequencing technology used resulted in the availability of good quality genetic data from each of Caleb, Patrick, Sarah, Laura and Ms Folbigg for the purpose of interpretation.

155. Having regard to the matters agreed by the experts during the course of the consultation meetings, I do not accept Ms Folbigg's submissions made after the close of the evidence that further testing should have been done.

156. I am satisfied no further sequencing, or other form of further testing, was required in order for the experts to ably interpret the data.

157. Having regard to the "trio" testing of child, mother and father discussed and agreed during the consultation meetings, and referred to in the ACMG Guidelines, I reject the submission on behalf of Ms Folbigg that three generation genetic assessment was appropriate. I note in this regard that neither Professor Vinuesa nor Professor Cook, nor indeed Ms Folbigg's legal representatives, raised at any stage during the consultation meetings the notion of a three generation genetic assessment.

Scope of phenotype information and investigations

158. In respect of Ms Folbigg's cardiac phenotype, I find the investigations carried out were extensive and adequate for the purpose of the interpretation of the genetic sequencing data both in respect of her and of the children.

159. I note in this regard Professor Vinuesa was asked by the Inquiry what investigations she recommended, and those which she recommended were undertaken.

160. I accept the opinions of Professor Skinner and Associate Professor Raju that no further investigations of Ms Folbigg (including a drug provocation test) are indicated either by the results of the investigations carried out, or by the genetic sequencing data itself.[169]

161. In respect of Mr Folbigg, I accept the opinions of Dr Buckley, Professor Kirk and Dr Colley that having regard to the particular genetic sequencing results, the absence of Mr Folbigg's genetic information or more detailed clinical information was not ultimately an impediment to the data interpretation process.[170] I note there was available to the experts some information about Mr Folbigg and his extended family.[171]

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167 Exhibit AB, Report of Dr Michael Buckley (25 February 2019) p 1.

168 Exhibit AA, Report of Dr Alison Colley (26 November 2018) p 2.

169 Exhibit BL, Letter from Associate Professor Hariharan Raju (18 April 2019) pp 1-2; Exhibit BK, Letter from Professor Jonathan Skinner to the Inquiry (30 April 2019) p 1.

170 Transcript of the Inquiry, 15 April 2019 T404.28-405.27.

171 Exhibit BG, Statement of Craig Folbigg (19 April 2019); Exhibit AE, Pedigree or Kathleen Folbigg (8 October 2018); Exhibit Z, Joint report of Sydney genetics team (29 March 2019) p 5 (Dr Colley also had the benefit of meeting Mr Folbigg and his sister in 1991, 1992 and 1993 following the death of Caleb and Patrick).

162. In respect of the children, I accept the opinion of Dr Colley that the phenotype was clear.[172] I find there was adequate phenotypic information for the purpose of the interpretation of the genetic sequencing data. I note also in this regard Dr Colley's evidence as to the consistency between the phenotype and the genotype of the children.[173]

Interpretation methodology adopted by the experts

163. I accept Dr Buckley's, Professor Kirk's and Dr Colley's evidence that the investigation of the sudden death of a child is intrinsically a clinical matter.[174] I note that during the course of the meetings between the experts before the results of the genetic testing were available, Professors Vinuesa and Cook agreed upon the application of the ACMG Guidelines. Only after the results became available and had been subject to interpretation did Professors Vinuesa and Cook suggest the ACMG Guidelines ought not be applied.[175]

164. I am satisfied the hypothesis-free analysis, coupled with:

    a. literature and database searches for sudden unexplained death in infancy, cardiac conditions and epilepsy;

    b. gene panel analyses on genes associated with sudden death in infancy/childhood; childhood neurological disorders; immunology and metabolism;

    c. any variant annotated as pathogenic or likely pathogenic related to any phenotype; and

    d. chromosomal microarray analysis,

    is sufficiently specific as well as broad to properly inform the Inquiry's task in considering the issue of cause of death of the Folbigg children from the perspective of the genetics as it is known and understood in 2019.

165. I find the scope of interpretation and analysis applied by the experts was adequate for the purpose of considering the relevance, to the Folbigg children, of all known or recognised genetic disorders correlating to their phenotype or clinical presentation.

166. I accept the interpretation exercise undertaken by the experts is necessarily defined and limited by the data identified, and the processes and knowledge in the field of genetics available in 2019.

Results

167. The findings of each of the teams were almost identical. Neither found variants in genes which were assessed as pathogenic or likely pathogenic in all four children so as to cause their sudden death.[176]

168. Of the three genetic variants the subject of differing conclusions as to classification between the Sydney and Canberra teams:

    a. In respect of the CALM2 variant found in Ms Folbigg, Sarah and Laura, I prefer the expertise and evidence of Professors Skinner and Kirk and Dr Buckley. Having regard to the conflict between the genetic and clinical information in respect of Ms Folbigg's cardiac presentation and in respect of the manner of the children's deaths, I find there is no reasonable possibility that this variant caused the death of Sarah or Laura.

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172 Transcript of the Inquiry, 17 April 2019 T553.37-38.

173 Transcript of the Inquiry, 17 April 2019 T554.20-26.

174 Transcript of the Inquiry, 17 April 2019 T579.7-10.

175 Exhibit CC, Transcript of second meeting with expert geneticists (4 February 2019) T10.40-50.

176 Exhibit Z, Joint report of Sydney genetics team (29 March 2019) p 8; Exhibit AF, Joint report of Canberra genetics team (29 March 2019) p 13.

b. In respect of the MHY6 variant found in Ms Folbigg, Caleb, Patrick and Laura, I prefer the expertise and evidence of Professor Skinner and Professor Kirk in relation to the clinical information and its application in the classification process. I find there is no reasonable possibility that this variant caused the death of Caleb, Patrick or Laura or caused Patrick's ALTE.

c. In respect of the IDS variant in found Patrick I prefer the expertise and evidence of Professor Kirk in relation to the clinical information and its application in the classification process. I find there is no reasonable possibility that this variant caused the death of Patrick.

169. I note that to the extent known, in relation to monogenic genetic causes of death by reason of variants in genes related to immunology, no relevant variants were identified by either the Sydney or the Canberra report. The issue of the relationship between infection and sudden infant death, including with a genetic interplay, is dealt with in Chapter 6.

170. In respect of digenic genetic causes of disease raised by Professor Vinuesa in her evidence,[177] I accept that such causes are a part of the expanding knowledge and understanding in the field of genetics. However, I find that:

a. the state of knowledge and understanding in the field of genetics in 2019 is not such as to enable digenic genetic causes interpretation of the Folbigg family genetic sequencing data and phenotype information in any meaningful sense; and

b. in any event there was no evidence before the Inquiry to suggest, on current knowledge and understanding, that a digenic genetic cause arises on the Folbigg family genetic sequencing data and the Folbigg children's phenotype.

171. In respect of the variants where Professor Vinuesa considered there was a theoretical possibility of pathogenicity, the evidence in the Inquiry went no higher than speculating about the possibility of genetic causes of death of any of the children.

172. I find that there is no reasonable possibility that any of the Folbigg children had a known or recognised genetic variant which caused their deaths.

[177] Transcript of the Inquiry, 16 April 2019 T520.37-521.8, T522.22-29.

Report of the Inquiry into the convictions of Kathleen Megan Folbigg

July 2019

Volume 1: Chapter 8

Chapter 8: The Non-Medical Evidence Including Ms Folbigg's Diaries

Introduction

The Crown case

1. The Crown's circumstantial case at the trial comprised both medical evidence as discussed in the earlier chapters, and non-medical evidence.

2. The non-medical evidence in the Crown case included:

    a. sworn oral evidence from Mr Folbigg about Ms Folbigg's mental state and attitudes and conduct towards the children over the course of their relationship;

    b. sworn oral evidence from other members of Ms Folbigg's family, as well as a number of Ms Folbigg's friends, police and other witnesses about their interactions with Ms Folbigg and the children at different points in time;

    c. various diaries authored by Ms Folbigg between 1989 and 1999; and

    d. a video and transcript of an electronically recorded interview between police and Ms Folbigg on 23 July 1999 before she was charged with any offence.

3. The Crown case relied significantly on an inculpatory interpretation of the entries in Ms Folbigg's diaries. It was the Crown case that the entries contained virtual admissions by Ms Folbigg of her guilt for the deaths of Caleb, Patrick and Sarah, as well as admissions by her that she appreciated she was at risk of causing, similarly, the death of Laura. Indeed, the Crown prosecutor submitted to the jury that the diaries were the strongest evidence the jury could possibly have for Ms Folbigg's responsibility for the deaths of the four children.[1]

4. In seeking to give this meaning to the diaries, the Crown suggested:

    a. Ms Folbigg never thought anybody would ever read them, the entries were only for herself;[2]

    b. the diaries did not have one entry of the kind one would expect from a person who had cruelly lost three children to natural causes;[3] and

    c. the diaries contained repeated ramblings about her tiredness, and her frustrations with the restrictions placed on her by having children.[4]

1 13 May 2003 T1372.54-56.
2 13 May 2003 T1366.32-34.
3 13 May 2003 T1366.53-55.
4 13 May 2003 T1367.9-14.

5. During the course of her interview with police on 23 July 1999, Ms Folbigg gave an account of the circumstances of the life and death of each child, as well as the meaning to be given to entries in her diaries of 1989 and mid 1996 to mid 1997, and her possession and disposal of her diaries.

6. It was the Crown case that the explanations Ms Folbigg gave to police during her interview on 23 July 1999, about entries in the 1996 – 1997 diary which police had at the time of the interview, were "unbelievable" and "unsatisfactory".[5]

7. The Crown prosecutor acknowledged to the jury that in parts of her diaries Ms Folbigg expressed joy at having her children. The Crown suggested that Ms Folbigg's "flashes of anger, resentment, bitterness and hatred" were not matters she thought all of the time.[6] It was suggested that Ms Folbigg had shown an unusual grief reaction following the children's deaths, consistent with ambivalence on her part, and that her reaction was one of grief, coupled with guilt for the children's deaths.[7]

8. The Crown case raised for the jury's consideration consciousness of guilt reasoning in respect of what was said to be lies told by Ms Folbigg, during the interview, about the meaning of the words "I really needed him to wake that morning and take over me", written in an entry dated 16 May 1997.[8] Ms Folbigg had accepted in the interview that the entry related to the night of Sarah's death.[9]

9. The trial judge directed the jury as to what they must be satisfied of in order to treat Ms Folbigg's answers about the entry as consciousness of her guilt. He directed the jury further that if they were satisfied she had deliberately lied they could use that finding in aid of other evidence in the Crown case.[10]

10. The Crown case also relied on Mr Folbigg's evidence about Ms Folbigg's generally deteriorating mood and patience with Sarah and Laura in the lead up to their deaths, and specifically her having lost her temper with each of them in the hours before their deaths. It also relied on Mr Folbigg's account that Ms Folbigg and Sarah had been out of the bedroom when he awoke during the evening a short time prior to Sarah's death, which Ms Folbigg had denied during her interview with police.

11. In respect of Mr Folbigg's admissions to having lied to police about aspects of Ms Folbigg's behaviour during the initial stage of the police investigation, and the impact this might have had on his credibility as a witness, it was the Crown case that his lies were readily understandable in the context of the events and his relationship with Ms Folbigg, and that there was other evidence which confirmed his account at trial, including Ms Folbigg's own answers in her interview with police.

12. The other lay witnesses in the Crown case gave evidence about their observations of Ms Folbigg as a mother, including some observations about her becoming frustrated at various times, however this evidence was not ultimately relied on significantly by the Crown.

The Defence case

13. It was the defence case in relation to the non-medical evidence that the absence of a motive on the part of Ms Folbigg, and the lack of evidence to support the picture of Ms Folbigg's behaviour which Mr Folbigg had attempted to place before the jury, would give the jury significant disquiet about the Crown case.

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5 13 May 2003 T1369.9-10, T1370.10-12; 20 May 2003 T110.
6 13 May 2003 T1376.15-24.
7 13 May 2003 T1373.26-44.
8 Exhibit AZ, Diaries tender bundle, pp 162-163; Exhibit E, ERISP of Kathleen Folbigg Q749-778.
9 19 May 2003 T84-85.
10 20 May 2003 T124-128.

14. The defence case also included non-medical evidence, namely sworn oral evidence from a number of Ms Folbigg's friends from the gym she attended at the time of Laura's death in 1999, who spoke positively of Ms Folbigg and her relationship with Laura, as well as letters written by Ms Folbigg to Mr Folbigg in the course of their relationship which demonstrated Ms Folbigg's dissatisfaction in the marital relationship, not with her children.

15. Defence counsel suggested there was no pattern of behaviour, or manifested course of conduct, or history of abuse consistent with the underlying state of mind alleged of Ms Folbigg by the Crown, by reference to her diary entries and Mr Folbigg's evidence. It was the defence case that there was instead evidence portraying Ms Folbigg in a positive light, inconsistent with Mr Folbigg's account of her behaviour.[11]

16. It was also the defence case that Mr Folbigg was motivated by revenge towards Ms Folbigg for her having left him, and that Ms Folbigg's diary entries exhibited normal reactions not only of grief, but of shame, guilt and responsibility, though not in the sense contended for by the Crown.[12]

The verdicts

17. It is apparent from the jury's verdicts that the jury rejected this aspect of the defence case and instead interpreted Ms Folbigg's diary entries in the inculpatory way the Crown contended for. An interpretation of the diary entries which treated certain entries as virtual admissions was considered by Sully J in the appeal against convictions to be not merely open and reasonable, but very persuasive:

> *These entries make chilling reading in the light of the known history of Caleb, Patrick, Sarah and Laura. The entries were clearly admissible in the Crown case. Assuming that they were authentic, which was not disputed; and that they were serious diary reflections, which was not disputed; then the probative value of the material was, in my opinion, damning. The picture painted by the diaries was one which gave terrible credibility and persuasion to the inference, suggested by the overwhelming weight of the medical evidence, that the five incidents had been anything but extraordinary coincidences unrelated to acts done by the appellant.*[13]

18. The significance of the diaries to the Crown case against Ms Folbigg was summarised by McHugh J in refusing special leave to appeal against the Court of Criminal Appeal's decision to dismiss her convictions appeals:

> *Essentially, we think that this was a case for the decision of the jury on the coincidence or tendency evidence led against the applicant in this unusual case. But apart from the coincidence evidence, there was other strong evidence, especially the diary entries made by the applicant, that was available to support the inferences that could be drawn from the tendency or coincidence evidence*.[14]

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11 14 May 2003 T1391.6-1393.4.

12 14 May 2003 T1408.45-1410.11, T390.35-51.

13 *R v Folbigg* [2005] NSWCCA 23, [132] (Sully J).

14 Transcript of Proceedings, *Folbigg v The Queen* [2005] HCATrans 657, 8 (McHugh ACJ).

The Inquiry

19. During the course of the Inquiry on 20 December 2018, Ms Folbigg indicated through her counsel that she may wish to give evidence before the Inquiry. Accordingly, I extended the scope of the Inquiry to allow Ms Folbigg to give evidence, if she wished to do so, specifically about the diary entries, possession of the diaries and her disposal of the diaries. I ruled that any evidence from her including by way of cross-examination would be restricted to those particular issues.[15] There was no challenge to this ruling at any stage.

20. On 16 March 2019, Ms Folbigg's legal representatives confirmed in writing to the Inquiry that she wished to give evidence. Between 29 May and 1 June 2019 Ms Folbigg appeared before the Inquiry to give evidence. She appeared of her own volition; she was not compelled by a summons to attend. The Commissioner of Corrective Services issued a local leave permit pursuant to s 36 of the *Crimes (Administration of Sentence) Act 1999* allowing her to be absent from the Silverwater Correctional Facility for the purpose of giving evidence.

21. Leave was granted to the DPP of Public Prosecutions and to Mr Folbigg to cross-examine Ms Folbigg. Ms Folbigg was cross-examined by Mr Maxwell QC for the DPP, Ms Cuneen SC for Mr Folbigg, and Ms Furness SC, counsel assisting. She was also examined by her own counsel, Mr Morris SC.

22. In addition to Ms Folbigg's oral evidence, the Inquiry received into evidence additional diary entries from the 1989-1999 period, additional evidence relating to Ms Folbigg's disposal of the diaries, and psychiatric assessment reports from the time of sentence in 2003 and from 2019 concerning Ms Folbigg's mental state.

23. This chapter details the non-medical evidence at trial, the further non-medical evidence in the Inquiry, the submissions on behalf of the parties and findings about that evidence as relevant to my task.

Non-medical evidence in the Crown case at trial

Mr Craig Folbigg – Ms Folbigg's husband and father of the four children

24. Mr Folbigg gave evidence about his relationship with Ms Folbigg between 1985 and 1999, about each of the children, about Ms Folbigg's relationship with each of the children, and about the circumstances of each of the children's deaths and Patrick's ALTE.

25. He explained that he met Ms Folbigg in 1985 and commenced a relationship with her after a short time. Ms Folbigg moved in with him in Newcastle in January 1986, and they were engaged in August 1986. Together they purchased a home in May 1987 and were married in September 1987.[16]

26. Mr Folbigg recalled that he and Ms Folbigg were both close with all of his family. He was one of eight children, with 22 nephews and nieces at the time of the trial.[17]

27. Mr Folbigg gave evidence that he had always been a smoker, but that Ms Folbigg never smoked. He said that when Ms Folbigg fell pregnant with Caleb she asked him not to smoke in the house. He said he then never smoked in the house again, and never smoked around any of the children in a confined space.[18]

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15 Transcript of the Inquiry, 20 December 2018 T6.6-10.

16 2 April 2003 T99.24-100.4.

17 7 April 2003 T217.43-47.

18 10 April 2003 T530.24-35.

Caleb

28. Mr Folbigg said that following Caleb's birth on 1 February 1989, Ms Folbigg and Caleb spent about five days in hospital. He gave evidence that Ms Folbigg was happy to be a mum, though was uncomfortable with breastfeeding. He said when they returned home Caleb slept in a white bassinet in the sunroom at the front of the house, with a door between his and Ms Folbigg's bedroom into that room.[19]

29. Mr Folbigg recalled discussing concerns with Ms Folbigg about Caleb making a noise while drinking from the bottle, such that he would have to break away and have a couple of breaths before starting to suck again. As a result of those discussions, Ms Folbigg took him to see Dr Springthorpe, a paediatrician.[20] This occurred while Ms Folbigg was still in hospital and Dr Springthorpe advised that Caleb had a floppy larynx.[21]

30. Mr Folbigg gave evidence that Ms Folbigg was Caleb's primary carer. He recalled Caleb was a quiet baby and seemed to be a good sleeper, but also said that he was working during Caleb's 19 days of life. He said he was himself a very heavy sleeper, such that he slept through "anything that went on during the night, whether a truck came through the wall, or a bomb fell".[22] He said he used to sleep through the night when Ms Folbigg got up to feed Caleb.[23]

31. As to Ms Folbigg's feelings and response to the role of motherhood, Mr Folbigg thought she was "pretty happy, we were happy".[24] He said "I wasn't really there much, but when I was there it seemed she was going okay".[25] In cross-examination he confirmed that there was nothing untoward about Ms Folbigg's attitude towards Caleb when he was alive, and nothing about problems or difficulties in her before he was born, that he saw.[26]

32. He added though that he observed a change in Ms Folbigg's demeanour after Caleb was born, namely that things went from her being special because she was having a baby, to Caleb being the special one.[27] Nonetheless he accepted that other than grumpiness and tiredness from lack of sleep, she appeared calm and comfortable with her new situation and that she was diligent, as shown by the diary recording his feeding and sleeping times.[28]

33. Mr Folbigg recalled that on the day before Caleb's death he, Ms Folbigg and Caleb spent the day with his brother, John, at his home. They arrived home at 8:00pm with Caleb already asleep. Ms Folbigg changed him and put him in his bassinet. At approximately 10:00 or 10:30pm Ms and Mr Folbigg went to bed, and Caleb was asleep peacefully.[29]

34. Mr Folbigg was next woken by Ms Folbigg screaming.[30] He went in and Ms Folbigg was standing at the end of the bassinet screaming. Caleb was in the bassinet, and Mr Folbigg lifted him.[31] Caleb's lips were blue, his eyes were closed and his skin was warm to touch.[32] He was not breathing.[33] Mr Folbigg attempted to resuscitate him, and told Ms Folbigg to call an ambulance.[34]

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19 2 April 2003 T100.27-44.
20 2 April 2003 T101.38-102.1.
21 2 April 2003 T102.15-21.
22 2 April 2003 T1011-17.
23 2 April 2003 T101.22-24.
24 2 April 2003 T102.23-29.
25 2 April 2003 T102.32-33.
26 7 April 2003 T220.4-8.
27 7 April 2003 T221.53-222.37.
28 7 April 2003 T224.55-225.36, T229.45-230.2, T230.42-231.50.
29 2 April 2003 T103.3-45.
30 2 April 2003 T104.3-9; 7 April 2003 T246.41-45.
31 2 April 2003 T104.11-15.
32 2 April 2003 T104.23-37.
33 2 April 2003 T104.37-41.
34 2 April 2003 T104.40-50.

35. He said both he and Ms Folbigg grieved for Caleb.[35] After Caleb's death Mr Folbigg said that he "fell to pieces" whereas it appeared to him that Ms Folbigg "pretty much basically just got on with her life".[36] He thought that she appeared to cope much better, and noted that she went out "a bit" to nightclubs and friends' places as soon as she started back at work, approximately a few months after his death.[37] He accepted in cross-examination that the outings to nightclubs were "infrequent".[38]

36. He recalled that it was he who pressed Ms Folbigg about having another baby, because he wanted to be a father.[39] In cross-examination he accepted that they both wanted to have another baby.[40]

37. He said they were introduced to a woman from the SIDS organisation, who told them about an assumed scenario for SIDS including low socio-economic status and housing issues, so they set about doing renovations to their house.[41] In cross-examination Mr Folbigg agreed that Ms Folbigg assisted with these renovations and appeared happy during her pregnancy with Patrick.[42]

Patrick – ALTE

38. Mr Folbigg recalled that at the time of Patrick's birth, both he and Ms Folbigg were euphoric.[43] He said Ms Folbigg stayed in hospital for approximately five days. She did not want to breastfeed Patrick and he was given formula in a bottle. Patrick slept in a cot in a bedroom off the dining room.[44]

39. At the start of Patrick's life Mr Folbigg said he was very nervous. He quit his job and stayed home so as to be with him and Ms Folbigg.[45] He observed that Ms Folbigg's attitude to motherhood was that she seemed to be enjoying it.[46] He noted it was still Ms Folbigg who attended to Patrick during the night, while he generally was fast asleep.[47] Mr Folbigg did not work for what he recalled was approximately three months, at which point he considered Patrick was healthy and Ms Folbigg to be going well, and he took a good job.[48]

40. On the date of Patrick's ALTE, Mr Folbigg had been back at work for three days.[49] He recalled that Ms Folbigg put Patrick to bed at about 8:30pm, and at about 10:30pm he went into Patrick's room and saw him in his cot, laying on his back, with a sheet and blanket over him. He noted he was still being fed at night.[50]

41. Next, in the early hours of the morning, Mr Folbigg was awoken by a "blood-curdling scream".[51] He ran down to Patrick's bedroom and saw Ms Folbigg standing at the end of cot, screaming.[52] He noted the covers were down towards the end of the bed, and Patrick looked like he was asleep. Mr Folbigg grabbed him out of his bed and screamed at Ms Folbigg to call the ambulance. He heard a little noise and thought he was breathing. Patrick was warm and pink. Mr Folbigg started CPR.[53]

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35 2 April 2003 T105.54-58.
36 2 April 2003 T106.13-24.
37 2 April 2003 T106.16-43.
38 7 April 2003 T250.23-31.
39 2 April 2003 T106.45-50.
40 7 April 2003 T248.40.
41 2 April 2003 T106.52-107.11.
42 7 April 2003 T249.1-11.
43 2 April 2003 T107.34-42.
44 2 April 2003 T107.44-58.
45 2 April 2003 T108.6-12.
46 2 April 2003 T108.56-109.2.
47 2 April 2003 T108.28-34.
48 2 April 2003 T108.40-50.
49 2 April 2003 T109.4-11.
50 2 April 2019 T109.20-39.
51 2 April 2003 T109.41-44.
52 2 April 2003 T109.44-49.
53 2 April 2003 T110.23-36.

42. In cross-examination Mr Folbigg agreed there was nothing in his statements to police to suggest that Ms Folbigg was not coping with Patrick prior to his ALTE. He also agreed there was nothing in his statements about the night of Patrick's ALTE being anything but normal.[54]

Patrick – death

43. Mr Folbigg recalled that over the following two months, Patrick was in and out of hospital suffering from similar types of fits and seizures. On 21 November 1990, he was diagnosed as blind.[55]

44. Mr Folbigg's evidence was that this was a very difficult period for Ms Folbigg, as she had a huge amount of things to do for Patrick, "on top of all the normal mum stuff".[56] He considered that she did not cope very well and lost her temper a bit with him and with Patrick. He said she got frustrated and cranky. He said that as a way of expressing this she used to growl, with her forearms out with her fists clenched and moving up and down.[57] He said this was a daily occurrence.[58] In cross-examination he accepted there was nothing to suggest she was in any way abusive of Patrick.[59]

45. It was Mr Folbigg's view that Ms Folbigg also showed that she was not coping by leaving Patrick with other people so that she could have some time out.[60] One of those people was Mr Folbigg's sister, Carol Newitt, and he also learned that Ms Folbigg was leaving Patrick with a neighbour named Dianne.[61]

46. Mr Folbigg recalled that at some stage after Patrick's ALTE he read a diary which Ms Folbigg kept on her bedside table. He read an entry in which she said she was not coping, that it was all too much drama, and that he and Patrick would be better off without her; that he could bring Patrick up with his family and do it better than she could.[62]

47. In cross-examination Mr Folbigg denied that Ms Folbigg ever expressed to him feelings of inadequacy about her care of Patrick or discussed with him that he needed to do more to share the load of care.[63] He agreed her reactions of stress and temper were understandable in light of the burden placed on her, and that she was doing the best she could and meeting his needs.[64]

48. Mr Folbigg said he phoned his sister and asked her to speak with Ms Folbigg. The three of them sat and discussed that Ms Folbigg could not just leave and she ought not to. Ms Folbigg agreed to stay, and Ms Newitt said she would assist her with Patrick, which she did.[65] In cross-examination he agreed that his sister's assistance had the effect of Ms Folbigg appearing to settle down a bit.[66]

49. In re-examination Mr Folbigg also said that Ms Folbigg was very upset that he had read her diary and made him promise he would never read another diary, so he never did, until May 1999, after Laura's death.[67]

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54 7 April 2003 T254.55-255.44.
55 2 April 2003 T112.7-15.
56 2 April 2003 T112.17-30.
57 2 April 2003 T112.32-54.
58 2 April 2003 T113.15-22.
59 7 April 2003 T261.44-48.
60 2 April 2003 T112.56-113.1.
61 2 April 2003 T113.3-13.
62 7 April 2003 T259.44-260.9.
63 7 April 2003 T260.37-51.
64 7 April 2003 T258.2-43.
65 2 April 2003 T114.6-27.
66 7 April 2003 T261.23-42.
67 10 April 2003 T526.33-57.

50. On the day of Patrick's death, Mr Folbigg recalled getting up at about 6:00am and getting dressed for work. He had breakfast with Patrick and left at about 7:30am. He did not recall that he noticed anything unusual that morning but did not think he could say that he took a huge amount of notice.[68] In cross-examination he also agreed he had not observed any stress in Ms Folbigg, or had any arguments with her, during the days before his death.[69]

51. He said that at 10:00am that morning he received a phone call at work from Ms Folbigg, who screamed down the phone "It's happened again" and "I need you. Come home".[70] He drove home quickly and upon running into the house saw Ms Newitt and Ms Folbigg there. He raced into Patrick's room and saw he was laying in his cot. He scooped Patrick up, put him on the lounge and commenced CPR. Patrick was floppy, warm, with blue lips. The ambulance officers then took over.[71]

52. Mr Folbigg said he asked Ms Folbigg a couple of days later what had happened, to which she replied that she just went in to check on him and found him how he was. He said they did not really discuss any further, and that "Kathy had a way of just cutting conversations off".[72]

53. Mr Folbigg recalled that he and Ms Folbigg had not been happy with the explanation given by Dr Wilkinson, that he thought Patrick may have died of an epileptic fit that he had not overcome, because after Christmas Patrick had been going "really, really well. Hadn't missed a beat". [73]

54. He said Patrick's death devastated both him and Ms Folbigg, though there were significant differences in the ways they grieved which was a cause for constant arguments. He said that for him the world pretty much stopped and he lost his job, whereas Ms Folbigg "went back to being herself, happy go lucky".[74]

55. He said that he and Ms Folbigg socialised together with friends and went to nightclubs and bought a new house.[75] In cross-examination he accepted that she was overcome with grief at the hospital and agreed there was no socialising by her other than with him.[76]

56. He recalled that in late 1991 Ms Folbigg said to him that she wanted to have another baby. He said he did not want to, and Ms Folbigg responded there was no point in being married if they were not having children. She said she would leave him if they were not going to have a family and gave him a week to think about it.[77] In cross-examination he agreed he had never said anything to Ms Folbigg against having another child because of concerns she was not a good mother.[78]

Sarah

57. Mr Folbigg recalled that at Sarah's birth in October 1992 both he and Ms Newitt were present. Ms Folbigg stayed in the hospital for a few days, and Sarah was fed formula. At home she slept in a crib in Ms and Mr Folbigg's bedroom, next to their bed. He recalled they were loaned an apnoea blanket from the SIDS organisation about two days after Sarah was born. This blanket did not have any attachments to the baby.[79]

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68 2 April 2003 T114.33-115.1.
69 7 April 2003 T276.24-30.
70 2 April 2003 T115.3-21.
71 2 April 2003 T115.23-116.31.
72 2 April 2003 T116.42-53.
73 2 April 2003 T116.55-117.12.
74 2 April 2003 T117.14-118.2.
75 2 April 2003 T118.4-18.
76 7 April 2003 T276.44-277.13, T279.4-23.
77 2 April 2003 T118.20-52.
78 7 April 2003 T283.6-284.2.
79 2 April 2003 T119.19-120.4.

58. His evidence was that Sarah snored but slept well and the snoring never caused any concern. He recalled the apnoea blanket sounded an alarm if the blanket failed to detect any movement for a set period, and it went off regularly, nearly every night. He said he did not know if the blanket was used during the day because he was at work, but that it was used at night. He said during the night sometimes he heard the alarm and sometimes he got up to respond, and at other times Ms Folbigg did.[80]

59. Mr Folbigg recalled that hearing the alarm caused anxiety, though on no occasion was there a cause for concern as they were ultimately all false alarms.[81] He said Ms Folbigg hated the blanket and wanted to throw it out since the start.[82] In cross-examination he accepted that the alarm going off caused stress to Ms Folbigg in particular because she got up and responded to the alarm, and was home in the day, which added extra pressure on her.[83]

60. It was Mr Folbigg's evidence that Ms Folbigg at times enjoyed motherhood with Sarah, but that there were things about it that she did not enjoy.[84] He said she went back to work when Sarah was about two and a half months old, because she was "sick of being broke, sick of being stuck at home".[85]

61. He described Ms Folbigg as a "very rigid, regimented type of person", particularly in respect of times for Sarah going to bed, and that "she just got sort of like harder about things".[86] He recalled that Ms Folbigg got very frustrated with Sarah and growled from time to time, becoming very domineering towards her.[87] In cross-examination he said arguments over this "8:30 affair", being the time Ms Folbigg wanted Sarah to go to sleep, happened a lot.[88]

62. It was Mr Folbigg's view also that it did not seem to bother Ms Folbigg having Sarah cared for by other people, and that she made arrangements for this "a huge amount of the time".[89] He said Ms Folbigg had ceased work in about mid 1993 after his sister's husband castigated her over missing much of Sarah's life.[90]

63. Mr Folbigg recalled that in the days prior to Sarah's death she had been suffering from a runny nose or cold. During the hours before Sarah's death, Mr Folbigg's evidence was that Sarah was wound up from the day, and after he ran her a bath and put her into her pyjamas Ms Folbigg took her to put her to sleep in bed. He said at this point "it all went pretty ordinary".[91]

64. He was in the loungeroom and could hear Ms Folbigg in the bedroom with Sarah, who was crying and grumbling. He heard Ms Folbigg growl while trying to comfort her. He went into the bedroom and saw Ms Folbigg standing with Sarah in a one arm bear hug, patting her bottom hard with the other hand.[92] Ms Folbigg told him to "fuck off" and said that everything was under control.[93] He recalled saying:

> *well, you know, for Christ's sake it's WWIII between the two of yous every time this kid's got to go to the bedroom. If she doesn't want to go to sleep, why make her go to sleep?* [94]

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80 2 April 2003 T120.6-44.
81 2 April 2003 T121.4-23.
82 2 April 2003 T121.23-33.
83 7 April 2003 T288.25-289.16.
84 2 April 2003 T123.3-7.
85 2 April 2003 T122.10-20; 7 April 2003 T290.25-33.
86 2 April 2003 T123.11-19.
87 2 April 2003 T123.21-29.
88 7 April 2003 T291.45-292.17.
89 2 April 2003 T123.30-35.
90 2 April 2003 T123.37-43.
91 2 April 2003 T125.8-126.7.
92 2 April 2003 T126.9-30.
93 2 April 2003 T127.26-35.
94 2 April 2003 T127.26-35.

He said Ms Folbigg told him to get out and that Sarah would sleep if she said she would. [95]

65. He went back into the loungeroom and could still hear that Sarah was upset. He then heard footsteps coming down the hallway. He said Ms Folbigg stopped two or three steps short of him and "threw" Sarah at him saying "you fucking deal with her" before storming off back to the bedroom. He said he had never seen Ms Folbigg do something like that before.[96]

66. Mr Folbigg said that Sarah fell asleep with him on the lounge, and he put her into her own bed at about 10:30 or 11:00pm. He put her down on her back with a blanket and a sheet.[97]

67. At this point, the sleep apnoea blanket had not been used for about two or three days. Mr Folbigg said they stopped using it because Ms Folbigg was reluctant to keep using it, on the basis that Sarah was fine and there was a possibility that the mattress of her new bed would not feed information down to the monitor.[98]

68. The next thing Mr Folbigg recalled was waking up at 1:10am which he read on a red digital electric clock which was lit up. He looked around half-asleep, halfawake, and saw that Ms Folbigg was not in the bed, and Sarah was not in her bed. He looked to the door and saw it was closed but could see light around the door. He said he was able to see reasonably well because the bedroom had light coming in from an outside streetlight, which came in through venetian blinds. He assumed Ms Folbigg must have been out of the room attending to Sarah, who was still being fed at night by Ms Folbigg. There was nothing unusual to him about Ms Folbigg and Sarah being out of the room during the night. He went back to sleep.[99]

69. Mr Folbigg then was awoken by Ms Folbigg's scream. The light was on in the room and Ms Folbigg was standing at the door. Sarah was laying on her bed, on her back, with her legs and arms straight alongside her, which he thought was different to how she ordinarily slept, "crunched up". He grabbed her off the bed and saw she was all floppy. She was warm and not breathing. He started to do CPR and screamed at Ms Folbigg to call an ambulance. [100] Ms Folbigg was sitting in the hallway just outside the door, screaming and crying with her knees up underneath her chin.[101]

70. Mr Folbigg gave evidence that he tried to speak with Ms Folbigg about what happened on the morning of Sarah's death, including the fact they were not in the room when he woke up. He recalled she said "I got up and went to the toilet. I came back, turned the light on, found her. That was that. The rest, you know". He said they did not talk much further about it.[102]

71. Mr Folbigg described that after Sarah's death his and Ms Folbigg's relationship "fell to pieces". He said he was not paying much attention to her and was still grieving about two years later. She told him he needed to go and see a grief counsellor and if he did not then she would leave him. They separated for about six to eight weeks before she returned home at his insistence, though they had more than one separation.[103]

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95 2 April 2003 T127.38-40.
96 2 April 2003 T127.42-128.3.
97 2 April 2003 T128.6-20.
98 2 April 2003 T126.54-127.6.
99 2 April 2003 T128.28-129.24, T130.44-131.30.
100 2 April 2003 T131.32-56; 3 April 2003 T150.46-52, T150.17-52.
101 2 April 2003 T131.35-45.
102 2 April 2003 T135.10-36.
103 2 April 2003 T136.5-137.2

72. Mr Folbigg was cross-examined at length about the detail that he was able to recall in relation to the events on the evening before Sarah's death. He agreed his first statement to police in 1999 did not include any reference to Ms Folbigg slapping Sarah's bottom. He maintained he had told the truth about what he remembered on this issue, and said:

> *You remember things more clearly when you're given more time and less stressful situations to remember them. So I'm sorry I didn't say it that day, but that was a horrible thing and day, and over the period of time since then I remembered that being the case.*[104]

73. As to his account in oral evidence about Ms Folbigg throwing Sarah, he said he had originally said "threw" to police but then changed it in order to "soften" and "lessen the blow", but still "impart the importance of what had happened".[105] He maintained that he told police she threw the baby at him, and in respect of his recorded conversation with Ms Folbigg on the listening device in which he denied this, said he was "covering my arse what I was telling her".[106] When it was suggested to him that it did not make sense that he was editing in order to "soften the blow", he said he had already made the "threw" statement to police and that it was apparent to him that they would still carry on investigating Laura's and the other children's deaths, and so it did make sense.[107]

74. In relation to his account in oral evidence that Sarah and Ms Folbigg were not in their beds when he woke at 1:10am, he said that in his original statement to police on 19 May 1999 he said this, but because of the "ramifications" for Ms Folbigg which became apparent to him between 19 and 23 May 1999, in circumstances where he was still in love with her, he then qualified his position with uncertainty to Detective Senior Constable Ryan.[108] He also said he never thought to bring up that information to anybody sooner because he never had any suspicions as to Ms Folbigg's involvement in anything until after Laura died.[109]

Laura

75. Mr Folbigg recalled that in May 1996, Ms Folbigg told him that while everything in their life was wonderful, having another baby would round it off. His reaction was one of shock, as he thought they had already agreed to not having more children. He initially said no, and she continued to bring it up a few times per week.[110]

76. He said he overlooked at the time something that she had said, namely "we were more mature, more patient people, and, like, older and wiser". He said he told her to wake up to herself, querying what that had to do with it as nobody had been able to say what happened to the children other than in one instance SIDS but nobody could tell them what SIDS was.[111]

77. He recalled he telephoned Professor Hilton and was put in contact with Dr Seton who welcomed them into the sleep study and assistance program and told them about the corometrics sleep monitor.[112]

78. Mr Folbigg recalled that upon her arrival in August 1997, Laura was breastfed as the hospital was "fairly stringent on that". He said she slept in a bassinet in the bedroom next to their bed and was taken to Westmead for three to four days as an inpatient at about one week for extensive tests and sleep studies.[113]

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104 7 April 2003 T295.10-15.
105 8 April 2003 T302.50-303.26.
106 8 April 2003 T324.11-32.
107 8 April 2003 T303.18-32.
108 8 April 2003 T342.50-347.17.
109 8 April 2003 T376.20-31.
110 3 April 2003 T151.15-152.45.
111 3 April 2003 T153.3-21.
112 3 April 2003 T153.24-52.
113 3 April 2003 T154.40-58.

79. In relation to the corometrics monitor, Mr Folbigg's evidence was that he and Ms Folbigg were told to plug the sensors onto Laura's chest whenever she was asleep, day or night. He said the monitor was used every time Laura went to sleep when he was home at night. He said it came to his attention after about two to three months that Ms Folbigg was not using the monitor through the day time. When he asked her about this, she said "I keep my eye on her and she'll be fine". He said that was not what they had been told, to which she replied that it was not him at home putting up with the machine, which he acknowledged emitted false alarms, and that she just wanted to have a normal baby.[114]

80. In cross-examination Mr Folbigg accepted dealing with the false alarms was stressful for Ms Folbigg. He also acknowledged that at times he did not feel he had done as much as he should have in assisting in the day-to-day care of Laura.[115]

81. Mr Folbigg said that by March 1998, Ms Folbigg's non-use of the monitor during the day had not changed. He felt he could not talk to Ms Folbigg about it so he wrote a letter to Dr Seton. That letter, in fact addressed to Margaret Tanner, a nurse in Dr Seton's clinic, was tendered and made Exhibit E at trial. In it, Mr Folbigg wrote "I feel that Kathy finds it all tedious and frustrating and would probably rather not use it at all, merely entrusting Laura's survival to fate".[116]

82. As to Ms Folbigg's attitude to motherhood with Laura, Mr Folbigg thought that she was happy being a mum, but that she would also get frustrated and cranky every day. This was directed at him, and at Laura for not doing what she was told. He said this started to get worse when Laura started walking, around 11 months old.[117]

83. He said at this stage his and Ms Folbigg's relationship had also "fairly much packed it in" such that they were mostly sleeping in separate rooms.[118] He described the relationship as cordial and polite if he kept his mouth shut and did not aggravate or intimidate Ms Folbigg.[119] He said that towards the end of Laura's life Ms Folbigg was going out with her girlfriends nearly weekly, and going to the gym daily and whichever nights she could.[120] In cross-examination he accepted that the only time when Ms Folbigg left Laura with others was when she went to the gym in the evening, and the usual arrangement of him returning home in time from work was not possible.[121]

84. Mr Folbigg recalled that about a fortnight before Laura's death Ms Folbigg had written him a letter in which she said she wanted to break up the marriage. [122] In cross-examination Mr Folbigg accepted that in the letter Ms Folbigg described him as an oppressive and depressing person and talked about leaving him and taking Laura with her.[123]

85. He gave evidence that they talked about things and she agreed to give it another go. He agreed in cross-examination that he said he would do more to assist her if she stayed.[124] He said he used to get cranky with Ms Folbigg for leaving Laura places when she went to the gym, and Ms Folbigg said to him that part of her problem was she never had enough time. It was at this point that Laura's sleeping arrangement changed and she started sleeping in a single bed in her own room.[125]

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114 3 April 2003 T155.11-157.9.
115 3 April 2003 T398.10-56.
116 3 April 2003 T158.37-159.22.
117 3 April 2003 T162.1-22.
118 3 April 2003 T162.24-31.
119 3 April 2003 T162.33-43.
120 3 April 2003 T162.45-58.
121 9 April 2003 T416.3-34.
122 9 April 2003 T163.9-11, T164.2-5.
123 9 April 2003 T400.45-50, T405.19-21.
124 9 April 2003 T404.49-58.
125 3 April 2003 T165.11-21.

86. Mr Folbigg recalled that on the Friday evening before Laura's death on the Monday, Ms Folbigg went out on a girls' night.[126] In re-examination a diary entry to that effect was tendered as Exhibit S.[127] On the Saturday, he went out and did various things but Ms Folbigg did not attend and did not want Laura to attend because she would become wound up. When he arrived home that night Laura was already in bed, a bit before her usual bedtime. Ms Folbigg warned him against waking her up.[128]

87. The next day, Sunday, they had friends over for a barbeque. He recalled that Laura was "full of beans", running around and swimming in the pool. He noticed something was off between Laura and Ms Folbigg, as it appeared they were avoiding contact. He asked Ms Folbigg about it, and said that she told him "Oh, she's got the shits with me… It's probably over what I did to her last night… I lost it with her."[129] She said that just before he had gotten home the night before she had spun around to tell Laura to stop whinging and moaning, and (inadvertently) knocked her down and screamed at her. On the Sunday night, both Mr and Ms Folbigg played with Laura and Ms Folbigg put her to bed.[130]

88. On the Monday morning, the day of Laura's death, Mr Folbigg's evidence was that he got up with her around 6:20am and then Ms Folbigg got up around 6:45am. Laura was "clingy, very subdued, whinging". She had picked up that Mr Folbigg was going to work and became very agitated and upset. He observed that Ms Folbigg was losing patience, and heard her growl from another room. He walked down the hallway and saw Ms Folbigg with Laura in the highchair, with both of Laura's hands pinned down while Ms Folbigg tried to feed her cereal.[131]

89. Ms Folbigg told him to "fuck off" and said "she's only like this when you're around. You do this to her. You mollycoddle her and sook her up too much". He said Ms Folbigg grabbed Laura and pulled her out of the chair, plonking her on the ground and saying "go to your fucking father".[132] She screamed "I can't handle her when she's like this". By this point Laura was "hysterical, shaking and sobbing". Mr Folbigg took Laura into the bedroom, then Ms Folbigg came in and said "Give me that baby… You give me that baby and get ready for work. Get out. You do this. This is your fault." He left for work and Laura was sitting in the family room watching television.[133]

90. It was Mr Folbigg's evidence that, like with Sarah, in the months prior to Laura's death Ms Folbigg was growling on a daily basis.[134] He said the causes of this were frustrations at Laura not having dinner at the right time, or going to bed when Ms Folbigg wanted her to, as well as things that he did and his attitude.[135]

91. At about 8:30am at work Mr Folbigg received a telephone call from Ms Folbigg. She sounded "very chipper" and wanted to apologise for having lost her temper that morning. She said she wanted to talk about their different parenting methods. She said Laura was fine, but agreed to come and have morning tea with him and did so around 10:30am after attending the gym. At about 11:30am, he recalled Ms Folbigg said "I better get buggerlugs home. She's due for a sleep", and that Laura was resistant to leaving with her.[136]

126 3 April 2003 T167.42-52.
127 10 April 2003 T529.1-33.
128 3 April 2003 T168.21-169.22.
129 3 April 2003 T170.28-171.25.
130 3 April 2003 T171.28-50.
131 3 April 2003 T171.52-172.36.
132 3 April 2003 T172.51-58.
133 3 April 2003 T173.5-34.
134 3 April 2003 T173.41-50.
135 3 April 2003 T174.1-5.
136 3 April 2003 T174.13-175.52.

92. Mr Folbigg recalled that at about 12:00pm he was on the phone when a staff member bolted in to his office and virtually screamed at him to hang up and get to the hospital because there was something wrong with Laura. At the hospital he met Ms Folbigg. When he asked her what happened she replied "I just went in and she was just laying there".[137] She went on to say that Laura had fallen asleep while driving home. She took her out of the car, walked up the hallway and took her shoes off before laying her down on her bed. She then went out to play with the dog, cleaned up the verandah and put the washing out. She said she heard Laura cough and splutter via the monitor but did not check straight away. She finished what she was doing and then went in to check on her and found her. She said it was between five to 10 minutes after hearing the coughing that she went to check.[138]

93. Mr Folbigg said that when he arrived home from the hospital he noticed that the hand piece for the monitor which could be carried around was plugged into the wall in the family room. He said one could not have heard the monitor in that position from the yard where the clothesline was. He said the wall position was where the monitor was either being charged or unused.[139]

94. In cross-examination it was suggested to Mr Folbigg that his version of events intentionally sought to put a negative light on Ms Folbigg's behaviour when what in fact occurred were normal domestic situations. It was pointed out to him that he had not told police of the detail of the last morning of Laura's life until December 2002. He responded by saying that he had not been given an opportunity to talk in terms like that since May 1999.[140] He denied attempting to paint Ms Folbigg in a sinister manner and said he "merely wanted everybody to understand [her] aggression; [her] gruff nature".[141]

After Laura's death

95. It was Mr Folbigg's evidence that Ms Folbigg packed away every photo of every child on the night of Laura's death.[142] He said their relationship deteriorated further thereafter. He was taking anti-depressants. About six weeks after Laura's death Ms Folbigg moved into a flat, saying she could not deal with her own grief and was not prepared to carry him and let him pull her down. She said she just wanted to be concerned for herself.[143] In cross-examination Mr Folbigg described that Ms Folbigg "never let anything out"[144] and "never told you much at all".[145]

96. When being cross-examined about an instance after Laura's death when Ms Folbigg was in the bath crying, Mr Folbigg said he thought it had been about Laura but that later Ms Folbigg told him "I was crying because I'm trapped here, cause I don't want to be here and I'm trapped here."[146]

97. In early May 1999 during the weeks after Ms Folbigg left the home, Mr Folbigg decided to tidy up and located a range of personal items belonging to Ms Folbigg. He asked her what to do with her things, and she told him to throw them in the bin as she did not want them.[147]

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137 3 April 2003 T176.8-56.
138 3 April 2004 T177.5-23.
139 3 April 2004 T177.45-178.23.
140 9 April 2003 T405.45-407.47.
141 9 April 2003 T430.20-25.
142 3 April 2003 T178.25-48
143 3 April 2003 T179.12-48.
144 7 April 2003 T248.6-7.
145 7 April 2003 T249.54-250.1.
146 8 April 2003 T311.34-50.
147 3 April 2003 T179.50-180.9.

98. He said he came across a diary in one of Ms Folbigg's bedside tables. He read some of the entries and what he read made him want to vomit. He said that prior to reading that diary he "had the odd suspicion", particularly by reference to Ms Folbigg and Sarah being out of the room prior to her death, and his observation of the monitor on the wall and Laura's shoes on the futon on the day of Laura's death. But he said he had nowhere to go with it and could not get his head around it.[148]

99. Mr Folbigg's evidence was that he felt sick reading the diary and did not know what to do with it, so he rang Detective Senior Constable Ryan and asked to meet him.[149] In re-examination Mr Folbigg clarified that it was the entries about Ms Folbigg's attitudes towards the children that had upset him. He said he had never seen before in Ms Folbigg, or experienced or witnessed, those attitudes prior to reading the diary entries.[150]

100. Upon meeting Detective Senior Constable Ryan he told him about the diary, but Detective Senior Constable Ryan would not take it unless he delivered it to the station. He said he took the diary to the police on 19 May 1999 when he attended the police station and spoke with police further.[151]

101. Mr Folbigg's evidence at the trial was that when he met with police on 19 May 1999 he gave an account orally about the circumstances of Sarah's death, and told the truth in that account. He said when he later returned on 23 May 1999 he changed some things about the account concerning Sarah.[152]

102. His explanation for this was that after speaking to police on 19 May he had been to see Ms Folbigg at her flat and told her that he had been to police. He mentioned the diary and told her that he had read some horrible things and given it to police. He said that from the next day onwards they started to have friendlier contact, and then about a month later in mid June she returned to the matrimonial home.[153] They separated on a final basis about 12 months later in June 2000.[154]

103. In cross-examination Mr Folbigg said that in his first discussion with police he tried as best he could to give as much detail as possible.[155] He said:

> *after I had read those diary entries, certain things about my life in some ways made sense to me then and I expressed all that to the police.*[156]

104. In cross-examination it was put to him that Ms Folbigg never told him to do anything but go and tell the truth to police. He said he understood this to mean "her truth", that she was a good, loving mother, and that the children were always neat and tidy and clean and fed.[157]

105. Mr Folbigg denied any revenge motivation for going to the police in May 1999 while separated from Ms Folbigg. He said he was "devastated" that she had left him. He said he lied to the police in his signed statement dated 23 May 1999 out of "concern"[158], and to suit his objective of life with her, with peace and harmony at home. He said he was in love with her and was blind.[159]

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148 3 April 2003 T180.17-53, T181.29-32.
149 3 April 2003 T181.10-11, T181.16-18.
150 10 April 2003 T524.5-18.
151 3 April 2003 T181.20-23, T181.48-58.
152 3 April 2003 T184.19-42, T521.24-522.7.
153 3 April 2003 T182.41-183.20.
154 3 April 2003 T184.4-9.
155 7 April 2003 T215.44-216.34.
156 7 April 2003 T216.40-41.
157 8 April 2003 T355.17-20, T355.57-356.24.
158 7 April 2003 T240.40-58.
159 7 April 2003 T245.11-15, T304.1-3.

106. In re-examination he said further that when he spoke with Ms Folbigg between 19 and 23 May, he had made an accusation towards her and felt "like a mongrel" afterwards because she had said to him:

> *How could you say those things about me. You know I loved them. And, you know, you saw how much I loved those babies... You've got to tell the truth... You know I loved those kids.*[160]

107. He said he had seen how she loved them, so he went back to Detective Senior Constable Ryan and asked him to "rewind back through his machine" so he could change things.[161]

108. On the voir dire, Mr Folbigg confirmed he had previously assisted Ms Folbigg to make inquiries about her natural parents, and she found out that her father had murdered her mother by stabbing her 27 times. He confirmed he had read the diary entry with the words "I'm my father's daughter"[162] and had asked Ms Folbigg about this. He said Ms Folbigg told him her father was, in her eyes, a loser, and she was as well.[163]

109. When a listening device conversation dated 26 July 1999 was put to Mr Folbigg in cross-examination, in which he said that Detective Ryan had "come and planted some bullshit in my head when I was at me lowest point when Kath had left me".[164] Mr Folbigg explained:

> *Detective Ryan came to see me at that time in my life and expressed to me the possibilities of what my wife could possibly have done, because it was evident to him I guess that I couldn't accept what she may have done and through what Detective Ryan said to me, helped me to come to grasp with those possibilities… by the time this conversation took place [Ms Folbigg] and I were back together… I had made the decision to myself that, as long as she didn't know that I was just spending whatever time she had left in the house getting to know who she was… hence conversations like that arose with people who were very good friends of hers.*[165]

Carol Newitt – Mr Folbigg's sister

110. Ms Newitt gave evidence that she had very close contact with Mr Folbigg during the early years of his marriage to Ms Folbigg, including during the years in which the children were born and died.[166]

111. She gave evidence about the assistance she provided to Ms Folbigg in caring for Patrick after his ALTE in October 1990. She said she spoke to Ms Folbigg and offered assistance after Mr Folbigg told her something about Ms Folbigg's intentions, and that Ms Folbigg agreed to stay with him as a result. She said at times Ms Folbigg would come over to her home in the daytime and walk in and give Patrick to her saying "you look after him. He is good for you and I can't get him to shut up". [167]

112. She considered that both Ms and Mr Folbigg were very dedicated parents to Patrick, and that it got Ms Folbigg "down like it would get anybody else down", but she thought her difficulties appeared to pass.[168]

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160 10 April 2003 T521.53-522.19.
161 10 April 2003 T522.16-19.
162 Exhibit AZ, Diaries tender bundle, p 178.
163 3 April 2003 T190.52-191.30.
164 8 April 2003 T304.14-23.
165 8 April 2003 T304.33-46.
166 23 April 2003 T891.48-892.10.
167 23 April 2003 T893.1-35.
168 23 April 2003 T898.54-900.18.

113. Ms Newitt described her attendance on the day of Patrick's death. She said she received a phone call from Ms Folbigg at about 10:00am saying "it had happened again". It was a five to eight minute drive from her home to Ms and Mr Folbigg's home.[169]

114. When she arrived, Ms Folbigg was sitting on the lounge directly in front of the door with her elbows on her knees, her head down, crying.[170] Ms Newitt saw Patrick was in his cot in another room, with the sides of the cot up. She recalled Ms Folbigg said to her "Don't pick him up" but clarified in cross-examination that all Ms Folbigg in fact said was "no".[171]

115. Ms Newitt gave evidence that none of the children showed any signs of failing to thrive, and none appeared to have constantly recurring infections. She was herself a mother of four children.[172]

116. Ms Newitt said she observed Ms Folbigg to be crying and very upset at both Caleb's and Patrick's deaths. She said she grew apart from Ms and Mr Folbigg somewhat after Sarah died so did not make close observations after that, and also that they lived further apart when Laura was alive.[173]

Lea Bown – Ms Folbigg's foster sister

117. Ms Bown gave evidence of a very close relationship with Ms Folbigg, having known her since she was three years old and maintained a lot of contact as adults. She said she had more contact with Laura than the other children and regarded Laura as a granddaughter.[174]

118. She recalled Christmas in 1998 when Mr and Ms Folbigg brought Laura to Melbourne at age 17 months. She said Ms Folbigg was not getting much sleep and had lost her temper with Laura in the high chair by pulling her out by her arm, which Ms Bown thought was uncalled for. She recalled also on Christmas Eve that Ms Folbigg had shown "over the top" anger when Laura did not want to go to sleep. She said she was surprised because she had not seen this side of Ms Folbigg before.[175]

119. Ms Bown also recalled an earlier occasion when Laura was five months old and she was visiting at the Folbigg home. She was inside with a migraine while all other adults were outside, and heard an alarm go off. She said she told Ms Folbigg who said it was probably the baby monitor and just shrugged her shoulders.[176]

120. When challenged that her police statement about these events was tempered compared with her oral evidence, she said she thought at the time of giving the statement that police were conducting a "witch hunt" and she was very angry at them for considering Ms Folbigg capable of what was alleged.[177]

121. Ms Bown agreed that Ms Folbigg was very happy to have each of her children and was devastated by their deaths. She agreed in cross-examination that Ms Folbigg had reported to her that each of the children (except Caleb) had been a "good sleeper" and a "good eater".[178]

122. Ms Bown denied any contact with Mr Folbigg in the two years before the trial and denied any discussion with him about statements to police.[179]

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169 23 April 2003 T894.1-24.
170 23 April 2003 T901.40-49.
171 23 April 2003 T900.38-50.
172 23 April 2003 T897.3-12.
173 23 April 2003 T898.31-43, T903.39-904.8.
174 16 April 2003 T764.4-768.38.
175 16 April 2003 T768.43-T769.57, T770.35-58, T771.1-12.
176 16 April 2003 T774.10-58, T775.1-26.
177 16 April 2003 T792.6-793.32.
178 16 April 2003 T783.23-38.
179 16 April 2003 T801.14-22.

Senior Constable Stephen Saunders – police officer who spoke with Ms and Mr Folbigg on occasion of Sarah's death

123. Senior Constable Stephen Saunders worked as a police officer and attended Ms and Mr Folbigg's home at approximately 2:45am on the date of Sarah's death. He noted both parents appeared distressed and the father was nursing the child. He said he was told that a sleep monitor had been used until the previous week, and that the child had not displayed any signs of illness other than having suffered a cold or flu-type virus. He was told this was treated by Dr Marley, with medication prescribed but the course not completed.[180]

124. Senior Constable Saunders gave evidence that the father, Mr Folbigg, told him that the child had been put to sleep at about 9:00pm in a single bed in the parents' bedroom, that parents went to sleep and the mother woke at 12:00 or 12:30am and heard the child turn over in its sleep, and that the mother again woke about 1:00am to go to toilet and on her return to the bedroom was unable to hear the child breathing. He read from his statement in which he had written that the mother had woken at 1:00am to go to the toilet.[181]

125. Senior Constable Saunders was asked about his statement in the report to the Coroner which he prepared, which stated the mother got up to go to the toilet at 1:30am. He was unable to explain the discrepancy between the report and his statement but considered it more likely to be 1:00am given that information was provided to him by the attending ambulance officer.[182]

Margaret Tanner – clinical nurse who met with Ms and Mr Folbigg during Laura's life for sleep monitor use

126. Ms Tanner worked as a clinical nurse at the Sleep Disorders Unit at the Royal Alexandria Hospital where Ms and Mr Folbigg attended with Laura for sleep management shortly after she was born.[183]

127. Ms Tanner made a number of observations about Ms Folbigg during the early attendances at the unit. She considered that Ms Folbigg seemed detached from Laura, as though she did not want to get close to her, and did not consider her to be overprotective.[184]

128. Ms Tanner also made observations about the use of the sleep monitor for Laura in subsequent months. She gave evidence that the instructions provided to Ms and Mr Folbigg about the monitor was that it was to be used for all sleep periods for about 12 months (until August 1998), with reassessment to be performed over time and use to be discontinued if the sleep studies were normal after 12 months.[185]

129. Ms Tanner attended to the periodic downloading of the monitor data at the unit. She observed that initially the monitor was used fairly well for the first two months after Laura's birth (September – October 1998). She said it then "dwindled off", being used at night time but not being used routinely in the day time. She noted that it was only used on the occasional Sunday or Monday during the day, and additionally for about an eight week period during December 1997 – February 1998.[186]

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180 11 April 2003 T527.35-574.28.
181 11 April 2003 T574.50-575.2.
182 11 April 2003 T579.39-580.7.
183 15 April 2003 T671.16-30, T674.29-33.
184 15 April 2003 T676.10-30.
185 15 April 2003 T677.13-16, T685.53-58.
186 15 April 2003 T678.22-55, T679.1-3, T679.16-49.

Deborah Grace – neighbour to Ms and Mr Folbigg

130. Ms Grace was a neighbour to Ms and Mr Folbigg during Laura's lifetime. She gave evidence of an event approximately eight days before Laura's death, when Ms Folbigg brought Laura to her home. She observed that Laura was in a fine mood, not being silly or naughty, but Ms Folbigg said "You're being silly. There's no nonsense like that, we are going home", and then walked straight out the door with Laura. She recalled attending the Folbigg home the day after Laura's death and observing "no emotion whatsoever" in Ms Folbigg.[187]

Melissa Smith – neighbour to Ms and Mr Folbigg, who cared at times for Laura

131. Ms Smith was a neighbour to Ms and Mr Folbigg during Laura's lifetime. Ms Folbigg had told her after Laura's birth that she would be placed on a sleep monitor. She started to babysit Laura when she was between 10 weeks and three months old. She recalled doing so twice before Laura was 11 months old. She was not provided with a monitor and recalled babysitting Laura for periods of about two hours, during which Laura went to sleep being nursed in her arms as she would not put her in the bedroom and did not like to leave her anywhere.[188]

132. Ms Smith recalled that when Laura was 11 months old and Ms Folbigg asked her to look after Laura while she went to the gym, she (Ms Smith) asked if she could mind her at Ms Folbigg's home so she could be placed on the monitor, which was done. She said she did not observe any breathing difficulties with Laura and observed a healthy and happy child who appeared to sleep well. She said she saw Ms Folbigg subsequent to Laura's death and thought she did not appear to be affected by Laura's death.[189]

Barbara Unicomb – neighbour to Ms Smith

133. Ms Unicomb was a direct neighbour to Ms Smith. She first remembered attending Ms Smith's home and seeing Laura there in her care when she was about three months old. She said there was no apnoea blanket attached to Laura, and no such device left at Ms Smith's home. She said she saw Laura in Ms Folbigg's company sometimes and thought she was a good mother.[190]

Karen Hall – friend of Ms and Mr Folbigg, who cared at times for Laura

134. Ms Hall was a friend to both Ms and Mr Folbigg, having met Mr Folbigg in 1994 while working at the same car dealership. She gave evidence that she started sometimes looking after Laura when she was about two months old, both in her own mobile home and at the Folbigg home. She said she was aware of the three prior children's deaths and was concerned to keep an eye on Laura.[191]

135. The monitor was not able to be used at Ms Hall's home, and she said there was a mutual decision for Ms Hall not to have care of Laura when she needed to sleep until she was a bit older. When she minded Laura at the Folbigg home the monitor was on, which she clarified was always at night. She said later Laura always slept on the lounge when with her, and occasionally she nursed her when she went to sleep.[192]

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187 16 April 2003 T803.51-804.16, T805.1-7.
188 16 April 2003 T811.57-812.56.
189 16 April 2003 T813.3-814.2, T817.15-18.
190 16 April 2003 T808.56-809.34, T810.10-16.
191 23 April 2003 T885.10-886.1, T886.13-32.
192 23 April 2003 T886.39-887.13.

136. Ms Hall recalled a time when Laura was about 12 months old and went to sleep on the lounge. She was out of the room for two to three minutes and returned to find she could not hear or feel Laura breathing. She scooped her up and put her on the floor, at which point Laura startled awake immediately. She said when she told Ms Folbigg, she responded not to worry too much because Laura slept deeply and it was probably just sleeping, though she promised she would put the monitor on when she went home. She generally observed Ms Folbigg to be alert to Laura.[193]

Kerrie Anderson – gym *crèche* worker at time of Laura's death

137. Ms Anderson gave evidence that she first met Ms Folbigg when Laura was only a few weeks old. She recalled that Ms Folbigg came to the gym frequently over the next 18 months or so, usually for an hour or two, during which time Ms Anderson cared for Laura in the ***crèche***. She said that as Laura became older, she started coming in daily. She also described that when Laura was younger, Ms Folbigg would check on her during the class, then as she got older she would just stay in the class. Ms Folbigg had told Ms Anderson that she had lost previous children.[194]

138. Ms Anderson recalled that on the day of Laura's death, towards the end of the class Laura appeared to be getting tired. She heard Ms Folbigg say to Laura that she would not be staying for a coffee because she (Laura) just ran around. She also heard her say that she wanted to take Laura home for a sleep. She thought Ms Folbigg seemed like her normal, friendly self.[195]

Detective Senior Constable Bernard Ryan – detective in charge of investigation

139. Detective Senior Constable Ryan gave evidence about his conduct of the police investigation after Laura's death.[196]

140. He said he attended the hospital shortly after Laura's death and took an account from Ms Folbigg which he recounted as follows:

> *She woke up at 6:20am this morning. She was in a bad mood. Mr Folbigg went to work and we had breakfast. We went to the gym and then we went to see Mr Folbigg at work for morning tea. She went to sleep in the car on the way home, so I put her in bed when we got home. I heard her coughing and did not think much of it. I went to check on her about five minutes later and saw that she wasn't breathing. I took her to the breakfast bar and did CPR and rang 000.*[197]

141. He said he asked Ms Folbigg why Laura was in a bad mood, to which she replied that she had had a cold for about a week. Ms Folbigg told him she left the gym at 10:30am and arrived home at about 11:00am, after attending Mr Folbigg's work. She told him she heard Laura coughing about half an hour after putting her down to bed and then found her laying on her back, face white.[198]

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193 23 April 2003 T887.25-888.47, T889.19-33, T890.44-46.
194 24 April 2003 T918.57-919.36, T921.51-922.2.
195 24 April 2003 T920.30-39, T920.57-921.14, T923.35-39, T924.15-17.
196 28 April 2003 T955-967; 1 May 2003 T1084-1096.
197 28 April 2003 T1353.50-1354.1.
198 28 April 2003 T956.8-20.

142. Later on the day of Laura's death Detective Senior Constable Ryan went to the Folbigg house and searched and photographed the room. He saw and took photographs of pillows on the bed, which had four small circular stains.[199] He observed in the lounge room on the lounge white Teletubby sandals and a baby's bottle, and a baby monitor nearby.[200]

143. Detective Senior Constable Ryan then gave evidence about his meetings with Mr Folbigg some months later. He gave evidence that he met with Mr Folbigg on 14 May 1999 at Mr Folbigg's home, at which time he understood that Ms Folbigg had moved out. Mr Folbigg told him about the existence of a diary, but did not give it to him at that point.[201]

144. Then on 19 May 1999 Mr Folbigg attended the detectives' office at Singleton Police station and brought with him two diaries (a 1989 diary and 1996 to 1997 diary), together with a number of other documents, including handwritten letters. Detective Senior Constable Ryan said that on that day he commenced taking a type-written statement from Mr Folbigg. He said he asked a series of questions in an attempt to allow a free account, with clarifying questions also asked. He said he did not complete the statement on that day and arranged for Mr Folbigg to come back on 23 May 1999.[202]

145. Detective Senior Constable Ryan's evidence was that on 23 May 1999 Mr Folbigg returned to the police station and told him he had not told the truth in relation to a number of issues on 14 and 19 May 1999. Mr Folbigg said he had resumed his relationship with Ms Folbigg and supported her. The interview continued and the statement, with changes, was concluded.[203]

146. The Detective Senior Constable then read the contents of the two diaries provided by Mr Folbigg very carefully. He said that as a result of what he read, and what he was told by Mr Folbigg, on 23 July 1999 he returned to the Folbigg home. There he met Ms Folbigg, who agreed voluntarily to an interview with him.[204]

147. Ms Folbigg drove herself to the police station and was seen by the Detective Senior Constable speaking on her mobile phone. Detective Senior Constable Ryan passed the nearby motor dealership where Mr Folbigg worked and saw him standing on the footpath speaking on a mobile phone. A short time later he arrived at the police station and saw both Mr and Ms Folbigg there. He explained that Mr Folbigg could not be present during the interview, and he left. Ms Folbigg agreed to be interviewed and declined to have anyone else attend.[205]

148. After the interview concluded, Detective Senior Constable Ryan informed Ms Folbigg police were going to execute a search warrant at the flat where she had been living. He said "we are looking for other diaries which relate to the death of your children. Do you have any more diaries?" She replied, "I've just started a new diary and it's up at the house". At approximately 6:30pm, the flat was searched and police took possession of a number of items (including the 1992 diary).[206]

149. Then at 7:15pm that evening, Detective Senior Constable Ryan attended Ms and Mr Folbigg's home at Millard Close to execute a search warrant there. He gave evidence that he said to Ms Folbigg "what we are actually doing here is that we are here to look for diaries, like we did at the last flat. Are there any diaries here?", to which he said she replied, "yeah, one that I bought yesterday". He observed her then walk into main bedroom, remove a personal diary (the 1999 diary) and hand it to another officer.[207]

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199 Exhibit E, trial Exhibit AE, Photographs of Laura's bedroom and stained pillow; Exhibit E, trial Exhibit AF, Report of Virginia Friedman (29 November 1999).

200 28 April 2003 T958.51-959.42.

201 28 April 2003 T960.8-49.

202 28 April 2003 T961.1-962.3.

203 28 April 2003 T962.10-44.

204 28 April 2003 T962.49-963.16.

205 28 April 2003 T963.25-58, T964.1-11.

206 28 April 2003 T965.7-28

207 28 April 2003 T965.30-58, T966.1-10.

150. The Detective Sergeant gave evidence that a short time later Sergeant Gralton found another personal diary in the main bedroom. He took Ms Folbigg into the bedroom and asked her if she'd like to make a comment about it. She said "I didn't know it was here. I thought it was gone".[208]

151. On 19 April 2001, Detective Senior Constable Ryan went to Mr Folbigg's workplace address and arrested him for the offence of hindering an investigation. Mr Folbigg then participated in an electronically recorded interview (the contents of which was not before the jury, and he was released without charge). Later that day he went to another address in Singleton and arrested Ms Folbigg in relation to the murder of her four children.[209]

The diaries

152. Tendered in the Crown case were five notebooks and calendars containing diary entries ranging from 1989 to 1999. There was no dispute at the trial (or in the Inquiry) that the author of the entries was Ms Folbigg.

153. Two diaries were initially provided to police voluntarily by Mr Folbigg on 19 May 1999. This occurred a few days after he first approached police on 14 May 1999.[210] The two diaries provided to police by Mr Folbigg consisted of entries between 1 February – 1 March 1989 (tendered in the trial as Exhibit L and one single entry from 20 February 1989 as Exhibit AK) and 4 June 1996 to 5 June 1997 (tendered in the trial as part of Exhibit J).[211]

154. A further diary containing entries between June 1997 – April 1998 was located by police inside Mr and Ms Folbigg's bedroom wardrobe at the time police executed a search warrant at the home on the evening of 23 July 1999, after Ms Folbigg's interview with police (tendered in the trial as part of Exhibit J).[212]

155. In June 2001, Mr Folbigg located inside a bread tin, which he and Ms Folbigg had used to keep important documents, a one page calendar sheet of August 1993 from a 1993 calendar (this became Exhibit H in the trial).[213] On the date of Sarah's death, 30 August 1993, was recorded "Sarah left us. 1:00am".[214] He recognised the writing as Ms Folbigg's.[215]

156. In October 2002, shortly before the trial commenced, Mr Folbigg located inside an old brief case a further diary from 1990, during Patrick's lifetime (this diary became Exhibit G in the trial).[216]

157. The table below sets out the calendars and notebooks containing diary entries which were tendered in the Crown case at trial. Each was also tendered in the Inquiry.

208 28 April 2003 T966.20-35.
209 1 May 2003 T1086.53-58, T1087.1-25.
210 28 April 2003 T960.36-49.
211 3 April 2003 T201.55-56, T1361.7-24, T202.40-44; 1 May 2003 T1086.50-51.
212 3 April 2003 T201.38-56.
213 3 April 2003 T200.58-201.9.
214 Exhibit AZ, Diaries tender bundle, p 62.
215 3 April 2003 T201.6-8.
216 3 April 2003 T199.10-45.

Year	Event	Diary	Trial exhibit reference	Inquiry exhibit reference
1989	• Caleb born 1 February 1989 • Caleb died 20 February 1989	1989 calendar diary *Covers period of Caleb's life*	Exhibit L	Exhibit AZ, Diaries tender bundle, pp 1 – 29
1990	• Patrick born 3 June 1990 • ALTE occurred on 18 October 1990	1990 calendar diary *Covers period from 1 February – 5 October 1990 (ends before Patrick's ALTE occurred)*	Exhibit G	Exhibit AZ, Diaries tender bundle, pp 32 – 61
1991	• Patrick died 13 February 1991	No diary entries tendered		
1992	• Sarah born 14 October 1992	No diary entries tendered		
1993	• Sarah died 30 August 1993	Calendar page of August 1993 *Records death of Sarah – "Sarah left us 1:00am"*	Exhibit H	Exhibit AZ, Diaries tender bundle, p 62
1994		No diary entries tendered		
1995		No diary entries tendered		
1996		Notebook diary from June 1996 – June 1997	Exhibit J	Exhibit AZ, Diaries tender bundle, pp 63 – 175
1997	• Laura born 7 August 1997	Notebook diary from June 1996 – June 1997 Notebook diary from June 1997 – April 1998 *Records pregnancy and birth of Laura*	Exhibit J	Exhibit AZ, Diaries tender bundle, pp 63 – 175 Exhibit AZ, Diaries tender bundle, pp 179 – 272
1998		Notebook diary from June 1997 – April 1998	Exhibit J	Exhibit AZ, Diaries tender bundle, pp 179 – 272
1999		Diary entry of 26 February 1999 from 1999 diary	Exhibit S	Exhibit E, trial Exhibit S

158. The table below sets out the contents of entries from the tendered diaries which were relied upon by the Crown at trial as containing virtual admissions by Ms Folbigg as to her guilt for Caleb's, Patrick's and Sarah's deaths, and admissions as to an appreciation of her risk of causing the death of Laura.

159. Listed beside each entry is a reference to Ms Folbigg's account interpreting the entry, either in her interview with police on 23 July 1999 or in her subsequent sworn oral evidence in the Inquiry.

160. The Schedule to this chapter is a table setting out the contents of other entries which were otherwise referred to either by the Crown or Defence during the course of the trial or at sentence, or which Ms Folbigg was asked to provide interpretation of during her evidence in the Inquiry.

Diary Entry	Ms Folbigg interpretation reference
1989 diary	
19 February 1989	
120 mls. Wind okay. 1 o'clock. Put back to sleep? 1.30: A bit restless [In the margin is written what looks like "wind"] 2am: Finally asleep!![217]	Exhibit E, ERISP of Kathleen Folbigg Q405-440 Transcript of the Inquiry, 29 April 2019 T676.29-677.43
1990 diary	
3 June 1990	
This was the day that Patrick Allan David Folbigg was born. I had mixed feelings this day wether [sic] or not I was going to cope as a mother or wether [sic] I was going to get stressed out like I did last time. I often regret Caleb & Patrick, only because your life changes so much, and maybe I'm not a person that likes change. But we will see?[218]	Transcript of the Inquiry, 29 April 2019 T640.16-642.13, T678.3-680.28
August 1993 calendar sheet	
30 August 1993 – Sarah died (10 months, 16 days)	
Sarah left us. 1am[219]	Transcript of the Inquiry, 30 April 2019 T733.45-50

[217] Exhibit AZ, Diaries tender bundle, p 19.
[218] Exhibit AZ, Diaries tender bundle, p 40.
[219] Exhibit AZ, Diaries tender bundle, p 62.

June 1996 – June 1997 diary

18 June 1996 Tues 10:21am

Baby plans still on the go. Could be preggy now. Won't find out til next month though. Back could create problem if my bodies [sic] not in peak condition- it may not accept pregnancy. Must have sex more if wanting to get pregnant. I'm ready this time. And I'll have help & support this time. When I think I'm going to loose [sic] control like last times, I'll just hand baby over to someone else. Not feel so totally alone Getting back into my exercise after will help my state of mind & sleeping whenever possible as well. I have learnt my lesson this time.[220]

Exhibit E, ERISP of Kathleen Folbigg Q590-599

Transcript of the Inquiry, 29 April 2019 T642.39-645.30, T642.26-37, T682.47-686.22

22 June 1996 – Sat

I watched a movie today about Shizophenia [sic], wonder if I have mild curse of that. I change moods really quickly. In my most dangerous mood, I'm not nice to be around & always want to be anywhere, but where I am. As long as it has music & men to show off too. Then there are times I wish to be more of a home body & please my hubby. Am I strange or is this behaviour normal. Guess I'll never know.[221]

Transcript of the Inquiry, 29 April 2019 T645.32-45, T688.8-37

21 July 1996 – Sunday 10:43pm

Depressed a little now. Probably because it will be a couple of more months before I'm pregnant. Pretty sure I'm not now, had or having what I think is a period. God I hope so or these tablets will cause brain damage. Probably would be just desserts for me considering. But not fair for Craig at all. I would feel like failure and wouldn't cope at all. Can't be dwelling on what ifs. I truly deserve anything life throws at me so my philosophy is whatever happens happens & it's the way it shall be. I'm going to try my hardest this time. If anything does happen, I'll just leave & try to let Craig go in peace and start again. No, I wouldn't. I'm not that brave - Really I depend on people & other people's help too much[222]

Exhibit E, ERISP of Kathleen Folbigg Q600-604

Transcript of the Inquiry, 29 April 2019 T688.39-692.19

26 August 1996 – 9:30am Monday

Have been spring cleaning lately- cupboard curtains etc. Must be pregnant. Well I hope so. I feel the time would be right now for us.

…

I suppose my heart isn't really in it. Because I want a baby. Yes I actually do want one!! Went to Clairvoant [sic] last week – so did Craig. I always believed there is more going on than just human nature. I seem content now because I now know that even though I'm responsible. It's alright. She accepts & is happy their [sic]. I've always felt her strongly. And now know why. She is with me. I think my mother is too.[223]

Exhibit E, ERISP of Kathleen Folbigg Q605-628

Transcript of the Inquiry, 29 April 2019 T645.47-647.20, T693.6-694.49, T647.22-648.37

220 Exhibit AZ, Diaries tender bundle, p 70.
221 Exhibit AZ, Diaries tender bundle, pp 71-72.
222 Exhibit AZ, Diaries tender bundle, pp 82-83.
223 Exhibit AZ, Diaries tender bundle, pp 92-93.

11 September 1996 – Tuesday nite 8:30pm

Transcript of the Inquiry, 30 April 2019 T703.24-705.30

Feeling inferior doesn't help. Feeling inadequate because I'm not pregnant yet. Feel as though its [sic] my fault. Think its [sic] deserved, after everything that's happened.
I suppose I deserve to never have kids again.
I am just so depressed don't know what to do.
Feel like taking the rest of the week off. But know my pay will be grossly affected if I do.[224]

14 October 1996 – Monday 9:00am

Transcript of the Inquiry, 29 April 2019 T670.8-670.28, T672.19-674.3; 30 April 2019 T708.50-710.48

Children thing still isn't happening. Thinking of forgetting the idea. Nature, fate & the man upstairs have decided I don't get the 4th chance. And rightly so I suppose. I would like to make all my mistakes & terrible thinking be corrected and mean something though. Plus, I'm ready to continue my family time now. *[REDACTED FROM TRIAL VERSION PURSUANT TO PRE-TRIAL RULING: Obviously, I'm my father's daughter.]* But I think losingmy temper stage & being frustrated with everything has passed. I now just let things happen & go with the flow. An attitude I should have had with all my children if given the chance. I'll have it with the next one.[225]

30 October 1996 – 5am

Exhibit E, ERISP of Kathleen Folbigg Q660-667

Transcript of the Inquiry, 30 April 2019 T710.50-712.24

I worry that my next child will suffer my physicological [sic] mood swings like the others did. I pray I'm prepared & ready mind wise for this next one. Maybe nature has decided I will never be & 'it will' never happen.[226]

4 December 1996 – Thurs 4:30am

Exhibit E, ERISP of Kathleen Folbigg Q 668-697

Transcript of the Inquiry, 29 April 2019 T648.39-651.25, T651.27-654.5; 30 April 2019 T712.26-719.1, T719.5-27

I'm ready this time. But have already decided if I get any feelings of jealousy or anger to [sic] much I will leave Craig & baby, rather than answer being as before. Silly but will be the only way I will cope. I think support & not being afraid to ask for it will be a major plus. Also - I have & will change my attitude & try earnestly not to let anything stress me to the max. I will do things to pamper myself regularly & just deal with things. If I have a clingy baby, then so be it. A cat napper so be it. That will be when I will ask help & sleep whenever I can. To keep myself in a decent mood. I know now that battling wills & sleep depravaision [sic] were the causes last time. Fish I've got help they are relaxing to watch its quite funny.[227]

224 Exhibit AZ, Diaries tender bundle, p 97.
225 Exhibit AZ, Diaries tender bundle, pp 177-178.
226 Exhibit AZ, Diaries tender bundle, p 107.
227 Exhibit AZ, Diaries tender bundle, pp 111-112.

1 January 1997 – 9:30pm Wednesday

Another year gone & what a year to come. I have a baby on the way, which means major personal sacrifice for both of us. But I feel confident about it all going well this time. I am going to call for help this time & not attempt to do everything myself anymore. I know that that was the main reason for all my stress before & stress made me do terrible things. Had a talk to Craig while in the bath tonight. Our favourite talking spot. Haven't really cleared anything, just told him how I feel and what vibes I'm receiving from him.[228]

Exhibit E, ERISP of Kathleen Folbigg Q698-711

Transcript of the Inquiry, 29 April 2019 T654.7-655.16; 30 April 2019 T719.29-722.49

14 January 1997 – Tuesday morn 3:00am

Well, best go. Time to return to bed & see if I can get some sleep. I'm sure this is training for when the baby arrives. That's OK, I'm pretty sure this time I'll handle it better. Hope so.[229]

Exhibit E, ERISP of Kathleen Folbigg Q712-716

Transcript of the Inquiry, 30 April 2019 T723.1-8

4 February 1997 – Tuesday morn 3:30am

Still can't sleep. Seem to be thinking of Patrick & Sarah & Caleb. Makes me seriously wonder wether [sic] I'm stupid or doing the right thing by having this baby. My guilt of how responsible I feel for them all, haunts me, my fear of it happening again, haunts me. My fear of Craig & I surviving it if it did, haunts me as well. I wonder wether [sic] having this one, wasn't just a determination on my behalf to get it right & not be defeated by me [sic] total inadequate feelings about myself. What sort of mother am I, have I been – a terrible one, that's what it boils down too [sic]- that's how I feel & that is what I think I am trying to conquer with this baby. To prove that there is nothing rong [sic] with me, if other women can do it, so can I. Is that a wrong reason to have a baby. Yes I think so, but it's too late to realise now. I'm sure with the support I'm going to ask for I'll get through. What scares me most will be when I'm alone with the baby. How do I overcome that? Defeat that?[230]

Exhibit E, ERISP of Kathleen Folbigg Q717-733

Transcript of the Inquiry, 29 April 2019 T655.18-656.38; 30 April 2019 T724.4- 727.44

17 February 1997 – Monday 9:50am

Wasn't all good news for me, But now I know what to do & say to keep him happy with me & everything else. Found out he's jealous already of Bub. He says he only has 6mths left to be with me and for me. Hopefully I've explained, thats not true, he should be for me forever, just because a baby is entering our life makes no difference really. One day it will leave. The others did, but this one's not going in the same fashion. This time I'm prepared and know what signals to watch out for in myself. Changes in mood etc. Help I will get if need be. I also know that my lethargy & tiredness & continued rejection of him had a bad effect.[231]

Exhibit E, ERISP of Kathleen Folbigg Q734-748

Transcript of the Inquiry, 30 April 2019 T727.46-729.21

228 Exhibit AZ, Diaries tender bundle, pp 114-115.
229 Exhibit AZ, Diaries tender bundle, p 118.
230 Exhibit AZ, Diaries tender bundle, p 126.
231 Exhibit AZ, Diaries tender bundle, p 127.

28 April 1997 – Monday morn 9.15

Transcript of the Inquiry, 30 April 2019 T729.23-730.21

Me, well, piling on wight [sic] now, what I eat doesn't help. Also all the books say that baby piles on stacs [sic] of fat from now on as well.

Hope that my labour isn't too different or I'm not sure I'll cope to [sic] well but made up my mind- will attempt breast feeding at least for the first 4-6 weeks then will swap to a bottle. I think this baby deserves everything I can give her. Concidering [sic] I really gave nothing to the others. I think even my feelings towards this one are already deeper. Shame, but thats [sic] the way it is. I think its [sic] because I'm 30 now & time to settle & bring up a child. Obviously I wasn't ready before at all.[232]

16 May 1997 – Friday morn 2:00am

Exhibit E, ERISP of Kathleen Folbigg Q749-778

Transcript of the Inquiry, 29 April 2019 T656.40-658.32; 30 April 2019 T730.39-735.29, T732.17-31

I think that she will be a great help in preventing me from stressing out as much as I've done in the past.

Night time & early morning such as these will be the worst for me, that's when wishing someone else was awake with me will happen.
Purely because of what happened before.

Craig says he will stress & worry but he still seems to sleep OK every night & did with Sarah. I really needed him to wake up that morning and take over from me. This time I've already decided if I ever feel that way again I'm going to wake him up.

I am glad I don't have to stay down in Sydney by myself. That prospect was really nerve racking [sic]. I would of [sic] felt so vulnerable & exposed. Relying on total strangers all the time.

… I also have selfish reasons. I'de like my last amount of private own time to myself. To just do what I want.[233]

232 Exhibit AZ, Diaries tender bundle, p 156.

233 Exhibit AZ, Diaries tender bundle, pp 162-163.

June 1997 – April 1998 diary

6 July 1997 - fri nite 9:30pm [Error made - should be 6 June]

Transcript of the Inquiry, 29 April 2019 T658.34-659.15; 30 April 2019 T736.6-739.22

From now on though I'm sure his attention & focus will change from me to his child & so it should.

I couldn't see that before. I was very selfish when it came to Craig's attention.

Hopefully this time we have both learned how to share it but still manage to keep a little something aside for just each other. We will see.

I hope I can say to him hey, this is our personal time just you & me, baby not included maybe if I remember to Pamper him, he'll remember me eg, put bub to sleep & have dinner with him, do a bath for him & I etc. Always hug & kiss him with love & intent affection, Cards, notes etc. listen to him. Not let baby totally dominneer [sic] my emotions & feelings. Maybe then he will see when, stress of it is getting to be too much & save me from ever feeling like I did before, during my dark moods. Hopefully preparing myself will mean the end of my dark moods, or at least the ability to see it coming & say to him or someone hey, help I'm getting overwhelmed, help me out. That will be the key to this babies [sic] survival. It surley [sic] will.

But, enough dwelling, things are different this time. It will all work out for sure.[234]

11 June 1997 – Wednesday nite 9:45pm

Transcript of the Inquiry, 30 April 2019 T739.24-740.41; 1 May 2019 T784.40-786.25

Sad news for Craig & his dad & family, His grandmother Gam died tonight.

Even though craig says, she wasn't very special to him I'm sure he feels sadness of some kind. He says that he has been thinking about her lately for some reason. I think he has slight ability to foresee whats happened or going to happen. Wonder if he'll predict our coming birth in the same way, interesting.

I know its a selfish thought, but why is at that something always seems to happen so that my birthday pales to non existence & becomes less important. I'm 30 this year, another milestone age, and again I feel depressed, lonely & unaknowledged [sic].

I think its because, Mum & Dad now no longer aknowledge me on my birthdays it hurts. On my birthdays I have no family of my own that really cares about when I was born into this world. Except for craig & this year its now been marred by Gams death. It will be interesting to see who rembers [sic] me & who doesn't.

[234] Exhibit AZ, Diaries tender bundle, p 182.

None of craigs family will except Dad & Mary & they may still get to [sic] upset over Gams funeral to remember anyway, Mel might remember, work will probably let It slide.

If it wasn't for my baby coming soon I'de [sic] sit & wonder again what I was put on this earth for, what contribution have I made to anyones life. Only person I think Ive made a difference too is craig. And times like this I can't do anything for him so I fail there as well.

30 years, first 5 I don't really remember, rest I don't choose to remember last 10-11 have been filled with trauma, Tragedy, happiness, mixed emotions of all designs. Maybe from now on I'll be able to settle a little. But no Immediate future brings. Turmoil, happiness, Sad memories, Happy ones, depression, great pride & it goes on. Life sux [sic], you can never figure it out is anyone meant too [sic].

Don't think I'll suffer alzimers [sic] disease, my brain has too much happening, unstored and unrecalled memories just waiting. Heaven help the day they surface & I recall. That will be the day to lock me up & throw away the key.

Something I'm sure will happen one day.

My problem is Im feeling like an obscurity of non existana [sic].
And it happens every birthday- Damn why have them.[235]

26 June 1997 – Thursday nite 9:30pm

Transcript of the Inquiry, 30 April 2019 T740.43-742.2

This time Im positive with support from Friends etc & Craig this time everything will work out fine & the sight & visions of the future I've been having will come true this time. With the other 3 I never bothered to think of school & teenage years. Maybe because I always knew they'd never get there - but this one I see myself taking her to school & Craig doing homework etc with her. Therefore I assume I'm actually ready for the "Family Life" now where I wasn't before. Feeling secure, loved successful & wanted by Craig has helped me. And to a degree the fact that I don't wish to die with no one really knowing I was here. At least now I know my son or daughter will. If God or that elusive higher power doesn't take them away from me once they are older to punish me. I'm trying to do right this I hope that is received & understood.

Most of my life has been turmoil, sadness, anger etc. I think now I might of [sic] actually realised it was mostly of my own making & stupidity that made it that way. Now I understand truly that your life & how it turns out is in your control, no one elses.[236]

235 Exhibit AZ, Diaries tender bundle, pp 188-189.
236 Exhibit AZ, Diaries tender bundle, pp 196-197.

25 August 1997 – Monday nite 8:30pm

Transcript of the Inquiry, 30 April 2019 T747.9-748.14

Scary feelings, I've realised I actually love her & have bonded with her, wish to protect her etc. Maternal instinct, is what they call it. I now know I never had it with the others. Monitor is a good idea. Nothing can happen without the monitor knowing & since I'm not game enough to not plug it in, because they'd want to know why I hadn't. Everything will be fine this time.[237]

20 September 1997 – Sat morn 3:15am

Transcript of the Inquiry, 30 April 2019 T748.16-749.12

Sleep, who needs it. Yes I'm getting a little irritable now.

This is my punishment for the others to be continually woken up, because this time we know that we have a child with a sleeping disorder. Even though I'm sure they are all false alarms, the thought is still scary. Must admit the only thing this has taught me is how to go to sleep myself quickly. Except for this morning. Every little sound is disturbing me. Wonder if this could lead to me becoming an insomniac.

Am getting very stressed, because I can't depend on Craig for any real help or support. He doesn't hear her or the alarms & I can't even trust & depend on him to look after her properly. He refuses to bother to learn anything about her. He doesn't pay attention when feeding her, hasn't changed a nappy, doesn't do washing or ironing only wakes up once in a while. His life continues as normal. Work, come home & I look after him, he doesn't even cook tea every now & then unless I ask him to. And then it is begrudgingly.

What do I do. The only break I get is when I go to aerobics – 3½ hours a week. But there one times, is not enough. I know my feelings are normal. I'm just venting. But at the moment, I sometimes wish I hadn't made the decision to have her but then all I have to do is look at her and all that melts away.

Well I just pissed Craig off, he's up and out of there now. Complaining he can't sleep. I have to keep disturbing him because he snores & grinds teeth badly.

[Craig] How dare he complain to me about lack of sleep — what the fuck would he know. Think he'll have to sleep in other room. Just so He's not disturbed- selfish prick. Well now I know where I stand. Craig is refusing to help & hasn't even attempted to in any way. Just wants me to bear all the stress, so he can keep selling his cars & making money. I suppose the stress of having to provide for us is real, but its nothing compared to this.[238]

237 Exhibit AZ, Diaries tender bundle, p 220.

238 Exhibit AZ, Diaries tender bundle, pp 220-222.

23 October 1997 – Thursday nite 9:30pm

Transcript of the Inquiry, 30 April 2019 T749.35-47

[Laura] She sleeps pretty good during the night too. Hell of a lot better than Sarah ever did. I think that's why I seem to be coping better this time.

Sure Im really tired by the evening But not too bad during the day. Also exersising [sic] is helping me too release stress & energises me through the week, I was fat & lazy last time I really not ready like I am this time.[239]

25 October 1997 – 10 pm Sat nite

Transcript of the Inquiry, 29 April 2019 T635.9-638.50; 30 April 2019 T749.49-751.48, T751.50-752.37, T752.39-49

Just watched video of Sarah, little upsetting, but she did some funny things. Made us laugh, think John was a little upset but he hid it well. I looked at it, but have to be honest & say I cherish Laura more, I miss her yes, but am not sad that Laura is here & she isn't. Is that a bad way to think, don't know.

I think I am more patient with Laura. I take the time to figure what is rong [sic] now instead of just snapping my cog. Also she is a far more agreeable child & easily flows most of the time.

Not sure how Craig feels about Sarah now. Know that even though he tried, he loves Laura just as hard - wasn't prepared for that. Thought he could remain stand offish, but couldn't. I think Laura is beautiful compared to Sarah — she was cute but Laura has a special look about her. Her slight difference in looks gives her a beautiful face, not just pretty, cute & cuddly, gorgeous & beautiful. Well so far anyway.

Looking at the video, Sarah was boyish looking. Laura has definite feminine features, they are chalk & cheese. And truthfully just as well. Wouldn't of handled another one like Sarah. She saved her life by being different.[240]

29 October 1997 – Wed nite 9.48pm

Transcript of the Inquiry, 29 April 2019 T639.1-640.14; 30 April 2019 T753.1-754.50

Felt a little angry towards Laura today. It was because I am & was very tired. That's why I decided to have an easy day, except for a walk - no gym. Obviously my body decided enough to the week. Might go Friday. Will see, if so, just for the ½ hr step class not the rest. Too long a day & too much [sic] (illegible word).

Wonder if Craig was serious about trip to Melbourne. Would be great to see Lea's face, but also scary, because it's a little to similar to what we did with Sarah. But Laura's different. Totally she doesn't push my Button anywhere near the extent she did. Which is good for her is all I can say.[241]

239 Exhibit AZ, Diaries tender bundle, p 225.
240 Exhibit AZ, Diaries tender bundle, p 226.
241 Exhibit AZ, Diaries tender bundle, pp 228-229.

3 November 1997 – Monday avo – 6pm

Transcript of the Inquiry, 30 April 2019 T755.1-759.15

Why is it when Im so tired Im feeling sick — shitty I cant [sic] sleep. Very depressed with myself at the moment. Feeling deprived of my freedom. I know that's the price that you pay for having a baby, but Ide [sic] not be human if it didnt get me down a little every now and then. It's because my release & enjoyment of the gyms been taken away I have to take her with me most times now, which means I can't enjoy myself & turn off like I usually do because she's there & I worry about her.

…

Someone's awake got to go. Lost it with her earlier. Left her crying in our bedroom & had to walk out- that feeling was happening. And I think it was because I had to clear my head & priotise [sic]. As I've done in here now. I love her I really do I don't want anything to happen.[242]

9 November 1997 – Sunday nite 8.45pm

Transcript of the Inquiry, 29 April 2019 T660.19-663.18; 30 April 2019 T756.42-759.15

Craig was pretty drunk Friday nite; In his drunken stupor he admitted that he's not really happy. There's a problem with his security level with me & he has a morbid fear about Laura- me well I know theres nothing wrong with her. Nothing out of ordinary any way. Because it was me not them.

Think I handle her fits of crying better than I did with Sarah-I've learnt to (illegible word) getting to me, to walk away & breath in for a while myself. It helps me cope & figure out how to help her. With Sarah all I wanted was her to shut up. And one day she did.[243]

8 November 1997 – Monday night 10pm [Error made - should be 8 December]

Transcript of the Inquiry, 30 April 2019 T759.17-762.8

Had a bad day today. Lost it with Laura a couple of times. She cried most of the day. Why do I do that. I must learn to read her better. She's pretty straight forward. She either wants to sleep or doesn't. Got to stop placing so much importance on myself.

…

Much [sic] try to release my stress somehow. I'm starting to take it out on her. Bad move. Bad things & thoughts happen when that happen. It will never happen again.[244]

15 December 1997 – Mon nite 10:14pm

Transcript of the Inquiry, 30 April 2019 T762.44-764.27

Caz sent a beautiful Angel & Tedy for Laura. Both her & Craig are convinced that Lauras soul is not her own, by the looks of it. Me well Im sure she met everyone & they've told her, don't be a bad or sickly kid. Mum may you know crack it. They've warned her — good. But she's still her own little person & will always be — must stop calling her Sarah. She's most definitely not her.[245]

242 Exhibit AZ, Diaries tender bundle, pp 229-230.
243 Exhibit AZ, Diaries tender bundle, p 231.
244 Exhibit AZ, Diaries tender bundle, p 238.
245 Exhibit AZ, Diaries tender bundle, p 242.

17 December 1997 – Wed nite 10:30

Tell you what don't think anyone could read this & find out all my secrets, I write like a 5 yr old, Disgusting to look.[246]

Transcript of the Inquiry, 30 April 2019 T764.29-765.17

31 December 1997 – New Years Eve– 11pm

Getting Laura to be next year ought to be fun, She'll realise a Party is going on. And that will be it. Wonder if the battle of the wills will start with her & I then. We'll actually get to see. She's a fairly good natured baby-Thank goodness, it has saved her from the fate of her siblings. I think she was warned.[247]

Transcript of the Inquiry, 29 April 2019 T663.20-66529; 30 April 2019 T765.19-766.32

4 January 1998 – 10:49pm

Sarah's missed. We watched her video. Made me realise how much I love Laura & cherish her like I never did the others. I don't take her for granted. I think with age has come a lot more patience & resignation that I can't fix or change things eg If she doesn't sleep all night then so be it. Sure it shits me & makes me a little grumpy. But I sort of just catch up during the day some time.[248]

Transcript of the Inquiry, 29 April 2019 T665.31-666.5; 30 April 2019 T766.34-767.43

28 January 1998 – Wednesday 5:30pm

Very depressed with myself, angry & upset.
I've done it. I lost it with her. I yelled at her so angrily that it scared her, she hasn't stopped crying. Got so bad I nearly purposely dropped her on the floor & left her. I restrained enough to put her on the floor & walk away. Went to my room & left her to cry. Was gone probably only 5 mins but it seemed like a lifetime.

I feel like the worst mother on this earth. Scared that she'll leave me now. Like Sarah did. I knew I was short tempered & cruel sometimes to her & she left. With a bit of help.
I don't want that to ever happen again. I actually seem to have a bond with Laura. It can't happen again. I'm ashamed of myself. I can't tell Craig about it because he'll worry about leaving her with me. Only seems to happen if I'm too tired her moaning, bored, wingy sound, drives me up the wall. I truly can't wait until she's old enough to tell me what she wants.[249]

Transcript of the Inquiry, 29 April 2019 T666.7-670.6; 30 April 2019 T767.45-771.27

6 March 1998 - Friday nite 10pm

Laura not well, really got on my nerves today, snapped & got really angry, but not nearly as bad as I used to get.[250]

Transcript of the Inquiry, 30 April 2019 T771.30-772.26

246 Exhibit AZ, Diaries tender bundle, p 243.
247 Exhibit AZ, Diaries tender bundle, p 246.
248 Exhibit AZ, Diaries tender bundle, p 250.
249 Exhibit AZ, Diaries tender bundle, p 258.
250 Exhibit AZ, Diaries tender bundle, p 264.

Ms Folbigg's interview with police on 23 July 1999

161. Following a ruling of the trial judge determining that answers given by Ms Folbigg about the diary entry dated 14 October 1996 in which she said "I'm my father's daughter" were not to be admitted into evidence, the Crown indicated it did not propose to lead the interview in the Crown case.[251] Ultimately however, on the basis of unfairness because this was a change in position from before the ruling, the Crown did lead the interview, edited to remove those particular answers.[252]

162. Detective Senior Constable Ryan gave evidence about the circumstances leading to the interview. He said that he approached Ms Folbigg after reading and considering the contents of the diaries provided to him by Mr Folbigg in May 1999. He attended Ms Folbigg's home in the morning of 23 July 1999.[253]

163. Ms Folbigg agreed to attend the police station for an interview and drove herself there. When Detective Senior Constable Ryan arrived at the police station, Mr Folbigg was present also. She and Mr Folbigg had reconciled following their separation after Laura's death, during which time Mr Folbigg had read her diary and provided it to police. Ms Folbigg was aware that he had done so. Detective Senior Constable Ryan explained that Mr Folbigg could not be present during the interview and he left. Ms Folbigg again agreed to be interviewed and declined to have anyone else attend.[254]

164. The interview commenced at 9:26am and concluded at 5:40pm, with breaks throughout the day. Ms Folbigg was informed that police were making inquiries in relation to the death of Caleb, Patrick, Sarah and Laura Folbigg.[255] She was informed she was not under arrest and was free to come and go from the interview at any time.[256] She agreed that at her home that morning Detective Senior Constable Ryan asked her to come to the police station "to be interviewed about the deaths of [her] children".[257]

165. The interview commenced with Detective Senior Constable Ryan asking open questions about each child's birth and the circumstances of their death. In response Ms Folbigg gave very lengthy answers taking up multiple pages of transcript, with few interruptions by police.[258]

166. Police then asked Ms Folbigg a series of questions about Mr Folbigg's statement to police regarding each of the children's deaths and Ms Folbigg's relationship with the children.[259] Ms Folbigg said she was aware of the statement and that Mr Folbigg had spoken to her about it "in bits and pieces". She said he had not said exactly what was in the statement.[260]

167. Before asking Ms Folbigg's questions from Mr Folbigg's statement, she was told she was not obliged to answer any questions unless she wished to do so and that if she did the answers may be used in evidence (cautioned).[261]

168. Police generally read Mr Folbigg's statement to her and asked for her response and asked follow up questions pointing out apparent inconsistencies between his account and her account given during the course of the interview.

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251 1 April 2003 T22.39-23.25; 28 April 2003 T968.1-29.
252 17 April 2003 T829.28-45; 28 April2003 T968.3-29.
253 28 April 2003 T962.49-963.16.
254 28 April 2003 T963.25-964.15.
255 Exhibit E, ERISP of Kathleen Folbigg Q11-12.
256 Exhibit E, ERISP of Kathleen Folbigg Q17-18.
257 Exhibit E, ERISP of Kathleen Folbigg Q30.
258 Exhibit E, ERISP of Kathleen Folbigg Q31-140 (Caleb); Q141-259 (Patrick); Q260-327 (Sarah); Q328-379 (Laura).
259 Exhibit E, ERISP of Kathleen Folbigg Q380-440 (Caleb); Q441-512 (Patrick); Q513-784 (Sarah); Q785-878 (Laura); Q879-914 (all children).
260 Exhibit E, ERISP of Kathleen Folbigg Q385.
261 Exhibit E, ERISP of Kathleen Folbigg Q383.

Caleb

169. In relation to Caleb, Ms Folbigg said generally that she did not remember that much and could not say that anything was out of the ordinary on the day of his death. She said she did not tend to hang on to dates, times, places, people's names. She said that all she remembered was walking into the bedroom and doing a check like she used to, following his early morning feed which had been a bit difficult and taken about half an hour.[262]

170. She said she remembered waking up for no particular reason, because she did not need to go to the toilet. She thought to herself, why am I awake, I better check Caleb and see if anything's up with him, even though he was not making any noises.[263] She said that she "scooped" Caleb up upon finding him not breathing, and that after that it was all a blur.[264]

171. She said she could not remember what time she had last put Caleb to bed on the night of his death,[265] and said that given the time of year if she had put any bedding on him it would have only been something light like a sheet.[266] She said that Caleb did not have sniffles or colds, but noted he had a feeding problem.[267]

172. Ms Folbigg said that Mr Folbigg did not get up and tend to Caleb at all, and that "you could let a bomb off under Craig and he would stay asleep".[268] She agreed she was very tired and that it was not an easy time.[269]

173. Ms Folbigg observed that she tried to have a sort of routine with Caleb, as with all the children.[270] She later denied that she tried to have him, or any of the other children, settled in bed by a particular time at night.[271]

174. When asked questions about Mr Folbigg's statement regarding Caleb, Ms Folbigg said he could be right that it was him not her who picked up Caleb, and that she did not specifically remember at the time.[272]

175. When asked about the 19 February 1989 diary entry in which she wrote "Finally asleep!!", she said this might have meant Caleb was a bit restless. When asked about the time entry of 2:00am, she agreed it appeared that he was having trouble going to sleep that night at that time, and that she seemed to have her times out with the 1:00am time she told the police.[273]

176. She said the only significance of the exclamation marks was that if he was having trouble and she was pleased he was asleep it meant that she could go to bed, but she said she did not recall him having the trouble that seemed to be presented in the diary entry.[274]

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262 Exhibit E, ERISP of Kathleen Folbigg Q37, 103, 104, 124, 132-133.
263 Exhibit E, ERISP of Kathleen Folbigg Q130.
264 Exhibit E, ERISP of Kathleen Folbigg Q104, 107-108.
265 Exhibit E, ERISP of Kathleen Folbigg Q85.
266 Exhibit E, ERISP of Kathleen Folbigg Q96.
267 Exhibit E, ERISP of Kathleen Folbigg Q38.
268 Exhibit E, ERISP of Kathleen Folbigg Q100.
269 Exhibit E, ERISP of Kathleen Folbigg Q127.
270 Exhibit E, ERISP of Kathleen Folbigg Q37.
271 Exhibit E, ERISP of Kathleen Folbigg Q117.
272 Exhibit E, ERISP of Kathleen Folbigg Q403-404.
273 Exhibit E, ERISP of Kathleen Folbigg Q412, 415-416, 424, 426; Exhibit AZ, Diaries tender bundle, p 19.
274 Exhibit E, ERISP of Kathleen Folbigg Q435-437.

Patrick – ALTE

177. Ms Folbigg said that Patrick was not anticipated but she and Mr Folbigg were extremely happy.[275] She said that Mr Folbigg was a bit more involved with Patrick.[276] She said by the time he reached close to three months old he was only waking up probably once, had gotten into a routine sleeping, and had no problems with breathing or with health in general.[277]

178. Ms Folbigg said that on the night of Patrick's ALTE she thought she had done his feed around midnight or 1:00am for about three quarters of an hour, everything went well and she put him back to bed and went back to bed herself. She said it was as with Caleb, a case of her finding herself awake for some reason or other, and she got up and thought "I need to go to the toilet, I'll check on him on my way past."[278] She said she was listening for his breathing and noticed that it was laboured. She said she flung the light on and went into action from there. [279]

179. She said she grabbed and scooped Patrick up, and had him in her arms by the time Mr Folbigg woke up.[280] She could not recall whether she rang the ambulance or Mr Folbigg did.[281]

180. She said that from October through to Christmas time she and Mr Folbigg were in and out of hospital with Patrick, trying to control his fits and then going through physiotherapy and trying to teach him how to keep up with normal development. She said the follow-up appointments were attended by her.[282] She said that on Mr Folbigg's birthday around 21 November 1990 they were told that Patrick was blind, which meant the physiotherapy and appointments all took another turn.[283]

181. Ms Folbigg said it was hard work, but that she and Mr Folbigg were just so relieved that he had survived.[284] She described herself as being "on auto-pilot", and said she received a lot of family support including from Mr Folbigg's sister.[285] She said she and Mr Folbigg started discussing special schooling for Patrick, and were being optimistic and thinking about the future.[286]

182. When asked questions about Mr Folbigg's statement regarding Patrick, Ms Folbigg said she thought she picked him up, but again accepted it was a possibility that it was Mr Folbigg.[287]

183. She agreed with Mr Folbigg's statement that the marriage was strained somewhat after the ALTE. She said 99 per cent of her time was looking after Patrick, and even the animals probably were ahead of Mr Folbigg on the ladder.[288] She considered that he was the sort of man that required someone to devote most of their time to him.[289]

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275 Exhibit E, ERISP of Kathleen Folbigg Q142.
276 Exhibit E, ERISP of Kathleen Folbigg Q142.
277 Exhibit E, ERISP of Kathleen Folbigg Q142.
278 Exhibit E, ERISP of Kathleen Folbigg Q149.
279 Exhibit E, ERISP of Kathleen Folbigg Q149, 172, 186, 189.
280 Exhibit E, ERISP of Kathleen Folbigg Q191, 195-198.
281 Exhibit E, ERISP of Kathleen Folbigg Q149.
282 Exhibit E, ERISP of Kathleen Folbigg Q149.
283 Exhibit E, ERISP of Kathleen Folbigg Q149.
284 Exhibit E, ERISP of Kathleen Folbigg Q149.
285 Exhibit E, ERISP of Kathleen Folbigg Q205-206.
286 Exhibit E, ERISP of Kathleen Folbigg Q156.
287 Exhibit E, ERISP of Kathleen Folbigg Q445.
288 Exhibit E, ERISP of Kathleen Folbigg Q454.
289 Exhibit E, ERISP of Kathleen Folbigg Q455.

Patrick – death

184. Ms Folbigg said that on the day of Patrick's death he woke up around 5:30 or 6:00am and had breakfast with Mr Folbigg.[290] She said she preferred to give him breakfast a little later but Mr Folbigg liked to spend time with him so eventually that was the routine and she had to do that.[291]

185. She said Mr Folbigg left for work, and that nothing struck her "that the day was any sort of different to any other".[292] She recalled putting him down for a bit of a morning nap at about 10:30am. She said she would usually hang out the washing during this time, but she did not really remember. She said she recalled walking into the room but could not recall if this was to see if he was alright or to put washing away. She said he was flat on his back which made her look twice because she used to always lay him on his side and he stayed there. She said her first thought when she saw him was that he was having another fit.[293]

186. Ms Folbigg said in respect of Patrick's death and that day that she might have "just blocked it". At that point she asked to take a break in the interview.[294] After the break, she went on to say that on that day Patrick was "the same as he always was", "a pretty happy sort of kid" who had not registered he had a problem.[295] She said he was happy 90 per cent of the time, and was a "very determined baby".[296] She said that he used to "run out of energy" at around 10:00am and would start getting a little on the grumpy side, so he would have a feed and go off to bed.[297]

187. When asked what she could tell police about Mr Folbigg finding a diary of hers sometime after Patrick was diagnosed as blind in November 1990, she said that she used to write in diaries as a sort of vent or release. She said she had learned not to do this, because she and Mr Folbigg had an altercation after she sprung him reading one of her diaries, which she took as an invasion of her space, even though she thought there was not anything in it that should have upset him too much.[298] It was at that point that she went on to tell police that she had disposed of three diaries on Mother's Day that year, being 9 May 1999.[299]

188. When asked again about an entry read by Mr Folbigg after Patrick was diagnosed as blind, referring to her considering leaving them both, Ms Folbigg said she was sort of suffering, as she had no time for herself, and her reaction was to want to "do a runner" from the situation. She said this came from spending so much time looking after Patrick and not being able to spend any time looking after Mr Folbigg or herself.[300] She said leaving would have been a fleeting thought.[301]

189. When asked about Mr Folbigg's description of her as suffering from depression while caring for Patrick, Ms Folbigg did not agree. She said she recalled being in a "down sort of mood" but contrasted that with the moods she experienced around the time of the interview which she would "class as a depression". She said Mr Folbigg had "a tendency to over analyse severely".[302]

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290 Exhibit E, ERISP of Kathleen Folbigg Q218.
291 Exhibit E, ERISP of Kathleen Folbigg Q218.
292 Exhibit E, ERISP of Kathleen Folbigg Q219, 222.
293 Exhibit E, ERISP of Kathleen Folbigg Q223.
294 Exhibit E, ERISP of Kathleen Folbigg Q223.
295 Exhibit E, ERISP of Kathleen Folbigg Q237.
296 Exhibit E, ERISP of Kathleen Folbigg Q239.
297 Exhibit E, ERISP of Kathleen Folbigg Q242.
298 Exhibit E, ERISP of Kathleen Folbigg Q459.
299 Exhibit E, ERISP of Kathleen Folbigg Q459-462.
300 Exhibit E, ERISP of Kathleen Folbigg Q466.
301 Exhibit E, ERISP of Kathleen Folbigg Q473-474.
302 Exhibit E, ERISP of Kathleen Folbigg Q468.

190. When asked if there were times that she got angry or frustrated after Patrick's ALTE she said it was just part of parenting, but that there was not an increase in the frustration level. She said when she did get frustrated it was usually towards Mr Folbigg because she felt she was doing everything herself and she did not get frustrated with Patrick.[303]

Sarah

191. Ms Folbigg told police that Sarah was "planned", following lots of discussions between her and Mr Folbigg. She said the doctors assured them they would help, and keep an eye on her.[304] She said she did not really remember day to day with Sarah, except that she was "a fairly good kid", and got nicknamed the "catnapper" because she would not sleep for any longer than 15-20 minutes at a time, and three hours during the night if lucky.[305] She thought this was when she developed "a real bad broken sleep habit, from her".[306] She said she was a healthy baby.[307]

192. Ms Folbigg described that she used to think that partly the reason why Sarah would not go to sleep happily and easily was that Mr Folbigg used to rev her up that much that she decided she wanted to keep playing and not go to bed.[308] When asked how this made her feel, she said it used to annoy her a little because Sarah would probably be grouchy the next day if she did not get sleep.[309]

193. She said at the stage of Sarah's death they had just decided to put her sleeping in a single bed rather than a cot, given she slept best in their bed.[310] She said they decided to put the bed in their room as a way of monitoring her, because they could not figure out a way to have the sleep apnoea mat on the bed.[311] She said the night of Sarah's death was her first night without the mat.[312]

194. When asked whether she had received any advice as to how long to use the sleeping mat, she responded that they had actually had it longer than what was recommended, which she said was six months.[313]

195. On the night of Sarah's death, Ms Folbigg said she had been trying to get Sarah to go to bed between 6:00 and 8:00pm, even for a bit of a sleep knowing full well she would not have stayed there.[314] She said she put Sarah to bed and she actually slept for a couple of hours, before Sarah "decided to get up and have a bit more of a party". At that stage Ms Folbigg went to bed herself and left Mr Folbigg with Sarah. She said she slept for a while, and Mr Folbigg put Sarah to bed, or she went out and said "that's enough, it's time for bed".[315]

196. Ms Folbigg said from there the only thing she remembered was actually finding Sarah. She said she got up to go to the toilet and glanced over and saw her lump in the bed. When she walked back in she took another look and she had not moved. She said what caught her attention was that she was flat on her back and one of her arms was hanging out, so she went over to make sure she was not cold. At that point she noticed that she was not hearing any breathing sounds. She said she woke Mr Folbigg up and there just seemed to be light in the room.[316]

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303 Exhibit E, ERISP of Kathleen Folbigg Q481-484.
304 Exhibit E, ERISP of Kathleen Folbigg Q261.
305 Exhibit E, ERISP of Kathleen Folbigg Q261.
306 Exhibit E, ERISP of Kathleen Folbigg Q261.
307 Exhibit E, ERISP of Kathleen Folbigg Q261.
308 Exhibit E, ERISP of Kathleen Folbigg Q261, 285.
309 Exhibit E, ERISP of Kathleen Folbigg Q287.
310 Exhibit E, ERISP of Kathleen Folbigg Q265.
311 Exhibit E, ERISP of Kathleen Folbigg Q321.
312 Exhibit E, ERISP of Kathleen Folbigg Q321.
313 Exhibit E, ERISP of Kathleen Folbigg Q327.
314 Exhibit E, ERISP of Kathleen Folbigg Q265.
315 Exhibit E, ERISP of Kathleen Folbigg Q268.
316 Exhibit E, ERISP of Kathleen Folbigg Q269, 308-317.

197. She said she could not remember if she had her or if Mr Folbigg had her, but they ended up in the loungeroom and Mr Folbigg attempted CPR. She could not remember if Sarah was pronounced dead at the house or the hospital and said the time after that was "just pretty much a blur".[317] She said the only vivid thing which stuck in her mind was "the fantastic day we had the day before". She said the only other thing was that she had a cold or flu with a runny nose and the sniffles, as was normal around that age.[318]

198. Ms Folbigg reflected that "because of her age, we weren't relaxing, we were still paranoid, but because she was older than what Patrick was we thought we were gunna manage to keep her."[319]

199. When asked about Mr Folbigg's statement to police that she had approached him to be a mother again, she agreed she had, and said:

> *Sure, having Sarah wasn't an attempt to replace or anything like that, it was determination probably on my part to succeed. At that particular time in life I was feeling like I'd failed, so the suggestion… was me wanting to have another child and succeed at being a mother sort of thing. It wasn't, I don't think there was anything else behind it.*[320]

200. When told that police had spoken to Mr Folbigg "at length about the four children, but in particular the death of Sarah", and that he said she was stressed constantly caring for all the children, Ms Folbigg said she recalled "becomin' probably a little bit more stressed with Sarah because she was such a short sleeper".[321] She also said that with Patrick there were reasons as to why he was needing constant care, whereas with Sarah there was really nothing wrong with her other than she did not like to go to sleep and wanted to play all the time.[322]

201. When asked about Mr Folbigg's statement that the night before Sarah died they had words about Sarah, she first said she did not recall this. She then said it was "the usual" – "a battle of the wills" as described to her by Mr Folbigg once. She said she used to get a bit stressed and a bit on the snappy side and then sort of give up and go to bed to leave Sarah to do what she wanted. She said there were "probably" battling wills that night, as it was a regular thing.[323] She said she "probably" made an angry growling noise that night as suggested by Mr Folbigg.[324]

202. She said further though that she would "become frustrated but never angry at her".[325] She said she would always end up reasoning to herself as she went to bed herself that Sarah was a baby and it was illogical to her.[326]

203. She said she did not recall having a conversation with Mr Folbigg as she was trying to put Sarah to bed.[327] When Mr Folbigg's version of the conversation was put to her, she again said that she did not recall it, and specifically said she did not recall saying that Sarah would go to sleep when she said so.[328] She denied Mr Folbigg's version that she "threw" Sarah at him, but recalled "giving" Sarah to him and saying she was going to bed.[329]

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317 Exhibit E, ERISP of Kathleen Folbigg Q269.
318 Exhibit E, ERISP of Kathleen Folbigg Q270, 280.
319 Exhibit E, ERISP of Kathleen Folbigg Q270.
320 Exhibit E, ERISP of Kathleen Folbigg Q516-517.
321 Exhibit E, ERISP of Kathleen Folbigg Q520-521.
322 Exhibit E, ERISP of Kathleen Folbigg Q521.
323 Exhibit E, ERISP of Kathleen Folbigg Q524-525.
324 Exhibit E, ERISP of Kathleen Folbigg Q527.
325 Exhibit E, ERISP of Kathleen Folbigg Q525.
326 Exhibit E, ERISP of Kathleen Folbigg Q525.
327 Exhibit E, ERISP of Kathleen Folbigg Q528-529.
328 Exhibit E, ERISP of Kathleen Folbigg Q533.
329 Exhibit E, ERISP of Kathleen Folbigg Q535-536.

204. Ms Folbigg said she could not explain the inconsistency between Mr Folbigg's version and her recall of the night until his version had been read to her, which was of not having too much trouble with Sarah.[330] She also said that with her children she chose to remember "all the good bits", not the "difficulty or the hard bits". She said specifically that sitting in the interview trying to recall whether or not the children gave her a hard time the night before they died, it was probably a case of her choosing not to remember it.[331]

205. When Mr Folbigg's account that he woke up at 1:00am on the morning Sarah died and saw that neither of them were in bed, the door was closed and there was a light on somewhere else inside the house was put to Ms Folbigg, she emphatically denied this saying "That is incorrect. He actually, no that's incorrect. Sarah never left the bedroom, she was in the bedroom the whole time".[332]

206. When police asked whether she understood the significance of Mr Folbigg's account, she said:

> *Yep, but that's not how it was. She never left the bed, she was in the bed, and I did shut the door, yes, but I didn't turn any lights on… As I said, I just remember that when I finally went over to the bed to uncover and find her and I've yelled at Craig the lights were on.*[333]

207. She agreed it was normal practice for her to wake up with Sarah if she was stirring in the night, shut the bedroom door and take her into another part of the house with the light on.[334] She maintained that on the night of her death, Sarah "stayed in bed".[335]

208. Later in the course of the interview police referred to Ms Folbigg's language of "I went to find her and uncover her" and asked whether she knew what she was going to find. She replied, "No. I just don't know why I used the word find."[336]

Laura

209. Ms Folbigg said that it was not an easy decision to have Laura and she and Mr Folbigg spent 12 months trying to figure out what support was available and whether they would proceed.[337]

210. Following her birth, she slept in a bassinette beside the bed, and spent an overnight stay in the hospital for sleep studies. She said she was "a good, sound sleeper in between her feeds", and they thought "there was no sort of dramas and she was very, more of a sedate, relaxed baby".[338]

211. She said the monitor that was provided by Dr Seton had electrodes to be plugged in, and "there was no question about that, it was on every time she slept, even if it was only for a 15 minute nap". She recalled the alarm was quite sensitive and ear-piercing.[339] She said she did not think they started to lessen the use of the monitor until Ms Tanner and Dr Seton said they could, around the six month mark. She said she started talking over with Mr Folbigg not using the monitor in the day. She said this was more her decision than his, a form of reluctant agreement, because she was the one that was always jumping up to the alarms.[340]

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330 Exhibit E, ERISP of Kathleen Folbigg Q539.
331 Exhibit E, ERISP of Kathleen Folbigg Q539.
332 Exhibit E, ERISP of Kathleen Folbigg Q543.
333 Exhibit E, ERISP of Kathleen Folbigg Q546.
334 Exhibit E, ERISP of Kathleen Folbigg Q549-551.
335 Exhibit E, ERISP of Kathleen Folbigg Q552.
336 Exhibit E, ERISP of Kathleen Folbigg Q781-782.
337 Exhibit E, ERISP of Kathleen Folbigg Q329.
338 Exhibit E, ERISP of Kathleen Folbigg Q333.
339 Exhibit E, ERISP of Kathleen Folbigg Q333.
340 Exhibit E, ERISP of Kathleen Folbigg Q333.

212. Ms Folbigg described that even from birth, Laura would only wake up every three or four hours, so she was not a catnapper, and she would not feed every hour, so she was "a really good, good baby. There was no, no sort of trouble with her at all".[341]

213. She said during the first 12 months there had been no problems other than sniffles a couple of times in the first cold season, but nothing ever serious and it did not last very long.[342] She said that "always with her, if she had the sniffles of any kind or even coughed just once Mr Folbigg and I would go to the doctors".[343]

214. Ms Folbigg said she thought they had become a bit complacent towards Laura's second birthday. She said she remembered the day of Laura's death a lot better than the others, probably because the bonding between her and Laura was far better than with the other three children. She said they were really enjoying getting out of the baby bit that was so frightening and into more of the toddler child sort of thing, where she was semi-talking and could ask for what she wanted.[344]

215. Ms Folbigg said on the morning of Laura's death they went to the gym as they usually did, and that probably by 11:00am Laura was asleep because she had had a big morning. She said she usually hung washing out at that point, and out of habit always checked on her. She said she checked every 15 or 20 minutes usually, and did so that morning. She recalled that Laura was flat on her back, whereas she always placed Laura down on her sides.[345] She said she did not pay attention to anything else other than she may have been a bit pale, grabbed her up and ran out to the kitchen breakfast bar and started CPR.[346]

216. She added that after the gym class she said to her friends "I won't stay for coffee today she's a bit, a bit on the feral side". She said Laura had fallen asleep in the car at about 11:00am on the way home, and that she used to take her shoes off while walking up the hallway when carrying her asleep from the car into bed, but otherwise leave her with what she had on.[347]

217. She also added that after she had put Laura to bed she had gone outside to check on the dog and thought that she left the monitor piece in the kitchen because sometimes having it on her was annoying. She said after she located the dog she went back inside and walked up the hallway, thinking she would check on Laura.[348] She said it took her about 10 minutes to locate the dog and 15 or 20 minutes between leaving and returning to Laura.[349]

218. Later in the interview, after having been through the various 1996 – 1997 diary entries, police asked Ms Folbigg questions about Mr Folbigg's statement concerning Laura. She agreed with his description that in February, the month before her death, their marriage was going through a fairly rough patch.[350]

219. Police showed Ms Folbigg a letter written by her which Mr Folbigg said she handed to him in the middle of February 1999, about two weeks before Laura died. Ms Folbigg said she sort of recalled handing it to him, and that she had started to think that maybe he and Laura would be better off if she was not on the scene, as she had with Patrick. She agreed that at that time she was considering leaving the home and not taking Laura with her.[351] She explained it was not to do with Laura, but rather because of issues between her and Mr Folbigg.[352]

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341 Exhibit E, ERISP of Kathleen Folbigg Q333.
342 Exhibit E, ERISP of Kathleen Folbigg Q333.
343 Exhibit E, ERISP of Kathleen Folbigg Q333.
344 Exhibit E, ERISP of Kathleen Folbigg Q333.
345 Exhibit E, ERISP of Kathleen Folbigg Q333.
346 Exhibit E, ERISP of Kathleen Folbigg Q333.
347 Exhibit E, ERISP of Kathleen Folbigg Q357, 359.
348 Exhibit E, ERISP of Kathleen Folbigg Q365.
349 Exhibit E, ERISP of Kathleen Folbigg Q376.
350 Exhibit E, ERISP of Kathleen Folbigg Q788.
351 Exhibit E, ERISP of Kathleen Folbigg Q793-975.
352 Exhibit E, ERISP of Kathleen Folbigg Q796.

220. When Mr Folbigg's version of the argument between them on the morning of Laura's death was put to her, Ms Folbigg agreed that she had said the words "She knows that she can get away with anything with you. You sook her up too much," and "Well go to your bloody father then". She said she was more angry at Mr Folbigg than angry at her.[353] She said Laura was crying because she would not let her out of the high chair, because she really wanted her to have breakfast.[354]

221. She said she thought she had said to Mr Folbigg "I can't handle it when she's crying all the time", but said the crying all the time rarely lasted that long and it was not a thing she did often, but only ever did so when Mr Folbigg was around.[355] She said she did not remember any other part of the argument, but said Laura definitely would have been crying as "she always used to cry more if I got upset and both of us were havin' bad vibes or whatever between the two of us".[356]

222. Ms Folbigg then said "I remember the argument. I remember getting upset and it was all purely with Mr Folbigg. I wasn't upset with Laura". She appeared to accept that Laura was "visibly scared" as described by Mr Folbigg:

> *probably because I was a bit more vocal than usual. If I had screamed, which I don't remember doing, if I'd screamed it would have been the first time she'd ever heard me doing that.*[357]

223. Police also asked Ms Folbigg about inconsistencies between her version to them on that day, and the version she provided to Detective Senior Constable Ryan on the day of Laura's death.

224. As to her omission during the interview about attending Mr Folbigg's work in the morning, she said "it just slipped my mind that I went there".[358]

225. As to her omission on the date of Laura's death that she had to feed the dogs and hang the washing out between the time Laura was asleep and the time she checked on her, she said:

> *when you asked me what I do of a morning my general response to that was it would have been housework and then I have remembered that I went out to find my, find my dog.*[359]

226. As to her omission during the interview that when she put Laura to bed she heard her coughing she said, "I had forgotten about her coughing and just forgot that she had a cold".[360]

227. As to Mr Folbigg's account of her report to him of Laura's death that she went to feed the dog and heard Laura coughing on the monitor, she said she did not remember a conversation with him.[361]

Interpretation of the diary entries

228. During the course of asking questions in relation to Caleb and Sarah, police also read to Ms Folbigg diary entries from the two diaries provided to them by Mr Folbigg in May 1999. The two diaries were the 1989 diary relating to Caleb and the 1996 – 1997 diary written during the period of Ms Folbigg's pregnancy with Laura. At the time of the interview police did not yet have the 1997-1998 diary written during the course of Laura's life.

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353 Exhibit E, ERISP of Kathleen Folbigg Q799-801.
354 Exhibit E, ERISP of Kathleen Folbigg Q801.
355 Exhibit E, ERISP of Kathleen Folbigg Q804.
356 Exhibit E, ERISP of Kathleen Folbigg Q807.
357 Exhibit E, ERISP of Kathleen Folbigg Q811-813.
358 Exhibit E, ERISP of Kathleen Folbigg Q822-824.
359 Exhibit E, ERISP of Kathleen Folbigg Q834.
360 Exhibit E, ERISP of Kathleen Folbigg Q835-836.
361 Exhibit E, ERISP of Kathleen Folbigg Q842.

229. Police also read to Ms Folbigg handwritten letters from her to Mr Folbigg on various dates, which he had also provided to police. Police asked Ms Folbigg, generally in an open manner, what she meant by the various diary entries and statements in her letters.

230. Ms Folbigg agreed that Mr Folbigg had told her about him bringing the 1996 – 1997 diary to police in May 1999. She said she was not happy about it, but he told her there were a few passages in the diary that he did not understand and asked her a few questions about them, which she answered. She told police he had not brought the topic up again since.[362]

231. Ms Folbigg gave the following explanations of the thinking and sentiments behind the words and phrases which appeared in her entries and, on a plain reading, appeared to indicate she was responsible for their deaths because she had lost control of her temper and done something physically to end their lives:

> "Ready this time":[363] *I was more confident that this time it would have been, being ready this time meant that I could get myself in the right frame of mind and be able to learn to accept all the help that people were gunna offer and probably handle it a bit better.*[364]
>
> "Lose control like last times":[365] *The frustration that I felt with Sarah every now and then, the frustration that I felt with Patrick. These were never frustrations that was detrimental to the kids in any way, it was usually directed at myself or Mr Folbigg. And I meant keep in control as in not keeping control as such, but sort of keeping control and learning to voice an objection or voice if I've got a problem. Instead of trying to handle everything myself.*[366]
>
> "learnt my lesson this time":[367] *As in the frustration area I was probably thinking of more there. Not to let the frustration get the better of me and learning to actually talk and communicate more.*[368]
>
> "if anything does happen":[369] *I was referring to if fate was cruel and it happened again.*[370]
>
> "try my hardest this time":[371] *Because I felt the last few times that, I had the opinion that I obviously hadn't tried hard enough. There was something that I could have done or hadn't done and should have, so that statement was that I felt I needed just to try to pay more attention and not miss anything.*[372]

362 Exhibit E, ERISP of Kathleen Folbigg Q587.
363 Exhibit AZ, Diaries tender bundle, p 70.
364 Exhibit E, ERISP of Kathleen Folbigg Q596.
365 Exhibit AZ, Diaries tender bundle, p 70.
366 Exhibit E, ERISP of Kathleen Folbigg Q597.
367 Exhibit AZ, Diaries tender bundle, p 70.
368 Exhibit E, ERISP of Kathleen Folbigg Q599.
369 Exhibit AZ, Diaries tender bundle, pp 82-83.
370 Exhibit E, ERISP of Kathleen Folbigg Q603.
371 Exhibit AZ, Diaries tender bundle, pp 82-83.
372 Exhibit E, ERISP of Kathleen Folbigg Q604.

"Even though I am responsible it's all right":[373] *I was still carrying around the thought that I could have done more or should have done more. So the word responsible in there sort of refers to that, my thoughts of I didn't try enough or didn't do something, I should have done something.*[374]

"my mistakes and terrible thinking":[375] *Just the frustrations that I might have felt with Pat, and the occasional battles of will that I would have had with Sarah. To me that, looking back at the time I thought that was a terrible way of thinking. I kept telling myself that that shouldn't have happened.*[376]

"I think losing my temper stage and being frustrated with everything has passed":[377] *to me losing your temper is well, totally losin' it. I'd regard losing your temper as sort of smashing things or throwing things or doin' something.*[378]

"I worry that my next child will suffer my psychological mood swings like the others did":[379] *Just as in a mood swing like I used to get, and mood swings still referring to something that by this stage I'd just decided that it shouldn't happen, and that was the frustration levels and the stress levels… and probably partly blamin' myself because the fact that I should have just been really mellow and just them do what they want. I started to think maybe I should have just been one of these mothers that let them do whatever they please and go from there… you know, your child picks up when you're upset about something or not happy about something… I sort of meant when they get upset in return.*[380]

"Rather than answer being as before":[381] *I don't think I really meant it as that, I sort of think I mean I would have found it easier if the frustrations have cropped back in the same as they had with Patrick or Sarah.*[382]

"Battling wills and sleep deprivation were the causes last time":[383] *one of the things I always wondered but never got any answers either way for was whether trying to get her to go to bed and trying to have some sort of normal thing and battling wills with her every now and then wasn't responsible for tiring her out to the point where she decided not to bother to wake up one morning.*[384]

"Stress made me do terrible things":[385] *Yeah, as in have an angry thought here or there… So I sort of decided that stress must have been the trigger for all that and we sort of knew all this stress was coming, we were pre-prepared for all the stress that come with Laura, so it was a case of being prepared for all that and hopefully I wouldn't you know, get the odd angry thought or be frustrated and go from there.*[386]

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373 Exhibit AZ, Diaries tender bundle, p 93.
374 Exhibit E, ERISP of Kathleen Folbigg Q610.
375 Exhibit AZ, Diaries tender bundle, p 103.
376 Exhibit E, ERISP of Kathleen Folbigg Q642.
377 Exhibit AZ, Diaries tender bundle, pp 177-178.
378 Exhibit E, ERISP of Kathleen Folbigg Q657.
379 Exhibit AZ, Diaries tender bundle, p 107.
380 Exhibit E, ERISP of Kathleen Folbigg Q662.
381 Exhibit AZ, Diaries tender bundle, p 111.
382 Exhibit E, ERISP of Kathleen Folbigg Q675.
383 Exhibit AZ, Diaries tender bundle, p 112.
384 Exhibit E, ERISP of Kathleen Folbigg Q684.
385 Exhibit AZ, Diaries tender bundle, p 114.
386 Exhibit E, ERISP of Kathleen Folbigg Q702.

> "scared of being alone with the baby":[387] *I had pretty much decided that since these terrible things kept happening when I was by myself I didn't want to be by myself.*[388]

232. Explanations of this nature were repeated in the same or similar terms as Ms Folbigg was taken through various entries from the 1996 – 1997 diary.

233. In relation to her explanation of wondering whether battling wills was not responsible for tiring Sarah out to the point of her not waking up one morning, Ms Folbigg said she thought she and Mr Folbigg had brought this up with Dr Seton, but she could not remember and she then said that she could be getting herself confused.[389]

234. When asked whether she really believed that attempting to get Sarah into some sort of routine caused her death she said, "I did for a while yeah, because you start to doubt everything that you've done".[390]

235. Ms Folbigg was asked about an entry where she wrote: "I really needed him to wake up and take over from me that night. This time I have already decided that if I ever feel that way again I'm going to wake him up".[391] She first explained:

> *Probably because I had so much trouble waking him up sometimes. It was, you know, third yell or whatever before he would sort of wake up. I don't think I was referring to any particular one of the three kids, just that his general lack of stirring.*[392]

236. She then agreed that entry was referring to the night of Sarah's death, and said:

> *cause I might have been feelin' a little bit stressed and a little bit tired that mornin' and it probably would have been good if… I'm not reading that and perceiving it as two linked sentences.*[393]
>
> She then said:
>
> *I might have meant just in general mothering care or whatever. Why couldn't he have woken up and gone to the toilet and checked on her himself, you know… I can't explain the take over.*[394]

237. Near the conclusion of the interview Ms Folbigg was asked a series of questions about her attendance at a clairvoyant, and about her beliefs in things such as destiny.

238. She said the clairvoyant referred to in her diary, whom she attended on before Laura was born and who referred to Sarah having "accepted", was at a "sort of fair thing on every now and then" with "clairvoyants from one end of the place to the other".[395] She said she used to believe that everybody had a destiny, but not anymore.[396]

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387 Exhibit AZ, Diaries tender bundle, p 126.
388 Exhibit E, ERISP of Kathleen Folbigg Q726.
389 Exhibit E, ERISP of Kathleen Folbigg Q693-694.
390 Exhibit E, ERISP of Kathleen Folbigg Q695.
391 Exhibit E, ERISP of Kathleen Folbigg Q751.
392 Exhibit E, ERISP of Kathleen Folbigg Q755.
393 Exhibit E, ERISP of Kathleen Folbigg Q762.
394 Exhibit E, ERISP of Kathleen Folbigg Q794.
395 Exhibit E, ERISP of Kathleen Folbigg Q876.
396 Exhibit E, ERISP of Kathleen Folbigg Q878.

239. Ms Folbigg said she could not explain the deaths of her four children. She said:

> *So I'm not religious, I don't think of it like that. Fate and how you [sic] life runs and destinies and all that sort of business, occasionally I wonder whether, whether there is a possibility of those sort of things. It probably correlates with me going to clairvoyants and that sort of thing. Trying to get some sort of sense as to whether there is somethin' else goin' on.*[397]

Non-medical evidence in defence case at trial

Jan Bull – Ms Folbigg's gym instructor at time of Laura's death

240. Ms Bull gave evidence that she met Ms Folbigg at the gym when Laura had just started walking. She said a lot of people in the morning classes at the gym had children, and there was a ***crèche*** to care for them during the classes. She observed that Laura and Ms Folbigg appeared to have a good bond, and she thought Ms Folbigg was a good mother. She confirmed she had never seen Ms Folbigg and Laura together outside of the gym environment.[398]

241. Ms Bull recalled that on the morning of Laura's death when they were at the gym she remarked to Ms Folbigg that Laura did not seem herself, in that she did not seem as bright and bubbly as normal. Ms Folbigg told her she was going to take her home and put her down for a nap.[399]

242. She gave evidence that she attended Ms Folbigg's home the day after Laura's death, and attended the funeral, and observed Ms Folbigg to be very upset on both occasions. Ms Bull said that together with others, she encouraged Ms Folbigg to go back to the gym quickly after Laura's death.[400]

243. When asked whether Ms Folbigg had told her about her interactions with Laura the night before and the morning of her death, Ms Bull said no and that that behaviour did not sound like anything she had ever seen Ms Folbigg do.[401]

Debbie Goodchild – Ms Folbigg's gym friend at time of Laura's death

244. Ms Goodchild gave evidence that she met Ms Folbigg at the gym when Laura was about 12 months old. She said she and Ms Folbigg saw each other three to four times per week at the classes. Ms Goodchild also had a child, aged two, who attended the ***crèche*** at the gym.[402]

245. Ms Goodchild recalled that on occasions she went to Ms Folbigg's house. Her impression was that Ms Folbigg and Laura had a good relationship. She said she never saw any demonstration of fear in Laura, or of Ms Folbigg losing control of her temper. She gave evidence that when she attended Ms Folbigg's home in the days and week or so after Laura's death she was always very upset.[403]

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397 Exhibit E, ERISP of Kathleen Folbigg Q879.
398 7 May 2003 T1184.23-1185.15, T1187.57-1188.2.
399 7 May 2003 T1185.37-54.
400 7 May 2003 T1186.12-29.
401 7 May 2003 T1192.32-1193.28.
402 8 May 2003 T1218.25-1282.10.
403 8 May 2003 T1282.15-1284.24.

246. She was cross-examined extensively about whether Ms Folbigg reported to her any of the kinds of things recorded in her diaries. She said Ms Folbigg had never reported such things to her, and said she had no knowledge of any stress or problems Ms Folbigg had.[404]

Judith Patterson – Ms Folbigg's gym friend at time of Laura's death

247. Ms Patterson gave evidence that she had gotten to know Ms Folbigg at the gym when Laura was a few months old. She said she and Ms Folbigg attended at each other's homes, as well as other gym friends' homes. She observed that Laura was a happy little girl who got along fine with Ms Folbigg.[405]

248. Ms Patterson said she babysat Laura when she was more than 12 months old and Ms Folbigg went back to work. She said that Laura never slept at her house while she babysat her. She attended on the day of Laura's wake and observed Ms Folbigg to be variously hysterical, looking sedated, and later smiling when reminiscing about Laura. Ms Patterson also gave evidence that Ms Folbigg did not discuss any emotional issues with her, such as her marriage, or her frustrations with Laura, or being stressed.[406]

[404] 8 May 2003 T1285.22-1287.14.
[405] 8 May 2003 T1287.27-1288.40.
[406] 8 May 2003 T1289.15-22, T1290.7-45, T1291.4-8, T1291.13-16, T1291.33-57.

Further non-medical evidence in the Inquiry

249. In line with my ruling extending the scope of the Inquiry's hearings to include Ms Folbigg's diaries, the Inquiry received the following further evidence relating to the diaries.

Further diary entries

250. Tendered into evidence in the Inquiry were additional diary entries which were available at the time of the trial but had not been tendered before the jury.

Year	Event	Diary	Inquiry exhibit reference
1989	• Caleb born 1 February 1989 • Caleb died 20 February 1989	1989 calendar diary *Additional pages extending beyond Caleb's life*	Exhibit AZ, Diaries tender bundle, pp 1 – 29
1992	• Sarah born 14 October 1992	1992 calendar diary "May Gibbs" *Records pregnancy and birth of Sarah*	Exhibit AZ, Diaries tender bundle, pp 564 – 615
1996		14 October 1996 entry "my father's daughter"	Exhibit AZ, Diaries tender bundle, pp 177 – 178
1999	• Laura Died 1 March 1999	*Prior to Laura's death* Single diary entries of 1 and 3 January 1999 *After Laura's death* Single diary entry of: 19 June 1999 and July 1999	Exhibit AZ, Diaries tender bundle, pp 274 – 276; pp 281 – 288; and pp 289 – 294

251. The 1992 diary had been located by police during the execution of a search warrant at the flat which Ms Folbigg had rented when she separated from Mr Folbigg after Laura's death. The Inquiry was unable to ascertain how the January and June 1999 entries, which were produced to the Inquiry by the Office of the DPP as single pages, had been located.

252. The July 1999 diary had been handed to police by Ms Folbigg at the start of the execution of the search warrant at the matrimonial home, when police asked her whether she had any diaries in the house.

253. The contents of these additional diary entries which Ms Folbigg was asked about in her evidence in the Inquiry are set out in the table below:

Diary Entry	Inquiry transcript reference
1989 diary	
24 May 1989 16 weeks (4 months) Make appointment Dr Leeder Booster for Caleb.[407]	Transcript of the Inquiry, 1 May 2019 T778.43-779.1
19 July 1989 24 weeks (6 months) Booster for Caleb[408]	Transcript of the Inquiry, 1 May 2019 T779.1-T780.36
1996 diary	
14 October 1996 – Monday 9:00am Children thing still isn't happening. Thinking of forgetting the idea. Nature, fate and the man upstairs have decided I don't get the 4th chance. And rightly so I suppose. I would like to make all my mistakes & terrible thinking be converted and mean something though. Plus, I'm ready to continue my family time now. *[REDACTED FROM TRIAL VERSION PURSUANT TO PRE-TRIAL RULING BUT BEFORE THE INQUIRY: Obviously, I'm my father's daughter.]* But I think losing my temper stage & being frustrated with everything has passed. I now just let things happen and go with the flow. An attitude I should have had with all my children if given the chance. I'll have it with the next one.[409]	Transcript of the Inquiry, 29 April 2019 T670.8-T670.28, T672.19-T674.3; 30 April 2019 T708.50-T710.48
1999 diaries	
Jan 1st 1999 12.01pm Friday Hope I feel more satisfied with myself & life this year Resolutions (1) More tolerance where Laura is concerned (2) More acceptance of my life and lack of youth (3) Try to keep friends Ive developed (4) More effort with my marriage (5) Get on with it.[410]	Transcript of the Inquiry, 30 April 2019 T773.12-34

407 Exhibit BN, Diary entries relating to Caleb Folbigg (24 May 1989, 18 July 1989).
408 Exhibit BN, Diary entries relating to Caleb Folbigg (24 May 1989, 18 July 1989).
409 Exhibit AZ, Diaries tender bundle, pp 177-178.
410 Exhibit AZ, Diaries tender bundle, p 274.

3 January 1999 – Sunday 9pm

Transcript of the Inquiry, 30 April 2019 T773.36-T774.21

Well, another day. Was okay. Have had unsatisfied feelings about my marriage. Can't do anything though, not until Laura is much older. For her I'll stay. It's gotten to the stage that a word to describe how I feel about things would be "comfortable". Craig I can take or leave. I suppose it's how we are supposed to feel after 12 yrs? Is it. Isn't there more? Or is that just in the movies & all that. Maybe I'm just fantasising. Don't know anymore. Sometimes I feel I could just drain the bank accounts and leave. Leave Singleton, Craig & Laura behind. Start new, somewhere. But I'm not that brave a person. Could go to Queensland, but what would they say. Would they attempt to send me back or get Craig to come. Will never know. Haven't even had a happy new year from them. Although did get an xmas card. Wow. I'm always wanting to run away. Maybe I have more mum in me that I give her credit for. Scary thought. I suppose I'm happy enough. I think this is all that there is supposed to be now. Watching Laura grow up and hopefully turn into a decent human being. We'll see?

…

1/2 my problems are a bad self-image. Only I can change that.[411]

19 June 1999 – 4am

Transcript of the Inquiry, 29 April 2019 T625.41-T627.1; 30 April 2019 T705.32-T707.36, T725.30-T727.44

Can't sleep. Tossing, turning. So much going through my mind. And none of it pleasant.
Another year has passed for me, each one is getting tougher. Trying to understand why the hell I'm even on this planet.
So many things point to the fact that I'm not meant to be.
Unwanted at birth. A father who was so selfish, unthoughtful that he took my mother from me & ruined my life from that one action.

…

I know I love him. He has shown me what love is. I just have so much trouble justifying to myself that I deserve it all. I just want to hide or watch the world go by it would be so much easier. But I know that Craig's life, wellbeing, happiness, security and mental state seem to depend on me. Is that a good thing? I think so, its preventing me from just dying inside. Which once my Laura left is whats happened to a great extent anyway. I just want to cry all day and night. No one see's this but I think its all just getting a bit too much. I vowed Ide [sic] never write any of my feelings down on paper again. But it's the only way I know how to release.

… It depresses & saddens me to realise that none of them will ever reach this stage in life. Only comfort I have is that where they are now they definitely have no stress or decisions to make and eternity can be spent carefree and loved always. At least that's what I have to believe or sanity I cling on to wouldn't last very long.

…

[411] Exhibit AZ, Diaries tender bundle, pp 275-276.

I think what has stirred all these emotions up is what I found out on Monday. Information is finally coming out & more & more I discover that they all, everyone of them are responsible for my predicament that I'm in now 30 years later.

…

I can't help but feel my life would have been so different & how it was meant to be if only Tom hadn't made a stupid mistake one night & "the family" hadn't interfered in the way that they did.
I believe that each person is here for a reason. Paths of life are chosen. So me having to adjust & alter mine so drastically has upset things. Because I can't believe that if there is a higher power that selects these (?) paths for people. How could he choose this one for me.[412]

July 1999 – 10pm Thursday night

Transcript of the Inquiry, 29 April 2019 T627.3-T628.35; 30 April 2019 T698.48-T701.16

Decided to start writing in a diary again. Have missed being able to vent regularly. I just pray it doesn't come back to bite me like my 97 one has.

But this time I'm not going to use it as a means to avoid communicating with him (Craig). I did before my journal became my friend and confident [sic].

Also looking forward to becoming a jewel princess hopefully for the Mayoral Ball on the 7th. The day will comemorate [sic] a loss for us, but the thought of pampering ourselves and formally dressing up and parading, stroking our egos for the evening will help me cope. Can't talk for Craig. To remember something beautiful, well, it was beautiful, but you know what I mean. Will never forget her, as I haven't all the others. But, new memories will eventually help heal the pain and shape our lives to come. [413]

412 Exhibit AZ, Diaries tender bundle, pp 281-288.
413 Exhibit AZ, Diaries tender bundle, pp 290, 292.

Ms Folbigg's sworn evidence about the interpretation of the diary entries

254. In her sworn evidence in the Inquiry, Ms Folbigg answered further questions about the 1989 and 1996 – 1997 diaries which she had discussed during the police interview, and about the 1990, 1997 – 1998, and 1999 diaries which police were unaware of at the time of interview. Ms Folbigg also answered questions concerning her possession and dispossession of the diaries, based on evidence which police similarly did not have at the time of the interview (discussed separately below).

The purpose of the diaries

255. Ms Folbigg gave evidence that she kept diaries on and off her whole life since she was a teenager.[414] While she said she did not write in them "all the time regularly",[415] she agreed she had probably kept a diary, or at least part of a diary, most years.[416]

256. Ms Folbigg said she considered her diaries an intimate friend and confidant, and that she was sincere in what she wrote in them.[417] She explained:

> *A. My, my diaries are a pouring out of every fear, every thought – negative, positive – every emotion, anything that was concerning me, anything at all, they were all poured into this diary. They were a way for me – as has been explained, I used those diaries as a friend and a confidant and, if you're having a discussion with your friend and confidant, are you expecting that friend and confidant to then go around telling everybody about it? You don't. So, I'm not expecting that people are going to be reading my diaries…*
>
> *Q. And because it was your confidant, you felt free to divulge the deepest secrets of your mind?*
>
> *A. I felt alone, I felt lonely, I had limited people to talk to – or I felt that I had limited people to talk to. These diaries were something that I could empty my head, get rid of emotions, try to figure out what was going on, how to figure out where I was going with life and they are - that's all they are. There's--*
>
> *Q. Put it down on paper and get rid of the guilt in your head?*
>
> *A. Put it down on paper and get rid of all the emotions and thoughts that were rallying around in my head, yes.*[418]

Interpretation of the diaries

257. The interpretation which Ms Folbigg says in 2019 should be applied to the diary entries may be summarised as follows.

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414 Transcript of the Inquiry, 29 April 2019 T622.18; 1 May 2019 T806.30.
415 Transcript of the Inquiry, 29 April 2019 T622.26.
416 Transcript of the Inquiry, 1 May 2019 T798.32-37.
417 Transcript of the Inquiry, 30 April 2019 T699.50-700.13.
418 Transcript of the Inquiry, 30 April 2019 T727.5-44.

258. Firstly, her diaries were an expression of her searching for answers to explain the children's deaths:

> *Q. You've referred to "battling wills and sleep deprivation" that were the causes last time. Is that right? Is that what you said in your diary entry, "battling wills and sleep deprivation were the causes last time"?* [419]
>
> *…*
>
> *Q. So causes of what?*
>
> *A. Causes of the deaths. I'm, I'm reaching and searching for any reason or cause of death.*[420]
>
> ** * **
>
> *Q. Well, you knew it, didn't you? You knew it was your dreadful moods, rather than keeping in a decent mood, that caused the deaths?*
>
> *A. No, the – these diaries are me continually searching and asking and questioning. Statements such as those in them are me grasping and grappling with answers that I'm – that I'm trying to get. And when you take it from the point of view that I'm constantly blaming myself, yes, I had in my head a belief that my moods affected everything, they affected my children, my children then died and decided they didn't wish to be with me anymore. It was quite a warped view as to how I was thinking and is evident in the diaries and to how I'm writing them.*
>
> *Q. Because you know, don't you, that children, babies, don't decide whether or not to live?*
>
> *A. At that stage in my life, I did not know that. At that stage in my life, I was doubting everything and highly stressed, and worried that here I am about to – you know, I'm going off on another tangent and I'm having another child, and my concerns were always – almost paranoia in the point, that I'm, I'm trying to make sure that Laura survives, Laura is successful, we're a family unit and my life goes the way I wish it to go.*[421]

259. Secondly, she did not do anything to the children physically to bring about their deaths, but held herself responsible for their deaths, because she was their mother. She always considered that she was a failure of a mother, because each had died in her care:

> *Q. In that particular line, "I know that battling wills and sleep deprivation were the causes last time", when you used the word "the causes" what did you mean, the causes of what?*
>
> *A. My belief at the time, your Honour, was it was all linked and related, my frustration and my inability to be a successful parent, I had belief that, you know, it was a wrong belief and a warped belief, but I had a belief that my children had decided they weren't staying with me anymore, and I did not understand why.*

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[419] Transcript of the Inquiry, 29 April 2019 T650.18-20.
[420] Transcript of the Inquiry, 29 April 2019 T650.35-36.
[421] Transcript of the Inquiry, 30 April 2019 T716.28-46.

Q. So when you say "were the causes last time", the causes of?

A. The causes of their – yeah, the causes of them dying. They woke up and decided to never wake up again.

Q. So--

A. Like they'd sleep and decided never to wake up again.

Q. --it would read then on that basis, "I know now that battling wills and sleep deprivation were the causes of their deaths"?

A. In some respect to me, yes that's what I believed at the time.[422]

* * *

Q. May we move on to 9 November please, on 9 November 97 you're talking about Craig, you say "There's a problem with his security level with me and he has a morbid fear about Laura", so far have I read it properly?

A. Yes, security level as in our relationship and our marriage.

Q. And you're saying there that Craig has a morbid fear about Laura, does that mean that Craig is constantly worried about Laura's health and wellbeing, in view of what has happened with other children?

A. Yes, same as me yes.

Q. But you say "me, well I know there's nothing wrong with her", that's right isn't it?

A. (No verbal reply)

Q. "Me, well I know there's nothing wrong with her."

A. Well I did know at the time there was nothing wrong with her, she was a very healthy baby.

Q. "Nothing out of ordinary anyway" and then you say "Because it was me not them"?

A. Because again I always constantly blamed myself for everything and took the responsibility and onus of responsibility on the fact that I'd lost the last three as my inability and failure as a mother.[423]

* * *

Q. "It can't happen again. I'm ashamed of myself. I can't tell Craig about it because he'll worry about leaving her with me."

A. Yes. It can't happen again. I don't want anything like that to happen again. I was ashamed of myself as in being a failure as a mother and not thinking I was good enough at this job.[424]

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422 Transcript of the Inquiry, 30 April 2019 T719.7-25.
423 Transcript of the Inquiry, 30 April 2019 T756.42-757.14.
424 Transcript of the Inquiry, 30 April 2019 T771.1-6.

260. Thirdly, she attributed her causal role to the effect of her mood on the children. The nature of that mood was a depressive state. Her expressions of losing it, snapping her cog, becoming angry, and snapping referred to thoughts and emotions of a depressive nature which she felt inside. Those expressions did not refer to any external response, including physical actions towards the children:

> *Q. What I'm putting to you is that, you knew you'd got angry with your children, the three children that died, before you wrote this. What do you say?*
>
> *A. I believed and felt that my moods at any given time affected my children, yes. I believed, as far out there as it is, that whatever bad mood I might have been in was a negative thing I was putting onto my children and I didn't like it.*
>
> *Q. And because you were in a bad mood, in some way that led to their death, but you can't really say how or why?*
>
> *A. That's right, I'm always searching for why. It never stops.*[425]
>
> * * *
>
> *A. Your Honour my own upbringing was I, even as a child, if my mood affected my mother there was a response to that, there was a consequence to that, so I therefore grew up believing that I had to always keep any negative or emotions or moods in check because I didn't wish to inflict on other people and I never wanted to burden other people and I had learned that if I did so there was always a consequence of some sort to that.*[426]
>
> * * *
>
> *Q. So just when you'd finally - did you ever experience this with Sarah during her life, that just when you'd finally got off to sleep she'd wake up again?*
>
> *A. Yes, quite often.*
>
> *Q. It put you under enormous pressure?*
>
> *A. Yes, I would say so.*
>
> *Q. Enormous stress?*
>
> *A. Yes.*
>
> *Q. Such that you snapped?*
>
> *A. No.*

[425] Transcript of the Inquiry, 29 April 2019 T663.50-664.14, T665.6-14.
[426] Transcript of the Inquiry, 1 May 2019 T789.20-25.

Q. Such that you snapped your cog?

A. No.

Q. This "snap the cog" you say has got nothing to do with losing control and doing something, is that right?

A. Not to my child, no.[427]

* * *

Q. You say, "I take the time to figure out what is wrong now, instead of just snapping my cog"?

A. Yes.

Q. What did you mean by "snapping" your "cog"?

A. "Snapping my cog", to me, could have been simply as even showing a slight frustration.

Q. Well, could it have been more than "a slight frustration"?

A. No.[428]

* * *

Q. Sleep deprivation, that's something that you suffered from, particularly with Sarah, correct?

A. Yes.

Q. It can put or would put or did put you under enormous pressure?

A. At times, yes.

Q. Such that you could snap your cog?

A. Not at Sarah.

Q. Not at Sarah?

A. Not at Sarah, no.

Q. Even though you were battling wills with Sarah, is that right?

A. My frustration levels and snapping cogs is the word you like to keep using, was more directed at my feelings of not having enough support at the time, that I felt like I was doing everything myself and deprivation, my sleep deprivation wasn't helping that situation. It was exasperating it.

Q. But you accept that sleep deprivation for an extended period with a child who you've said that you battled wills with at times, correct?

A. Yes.

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427 Transcript of the Inquiry, 30 April 2019 T649.21-39.

428 Transcript of the Inquiry, 30 April 2019 T636.19-28.

Q. Could put you under enormous pressure and did, is that right?

A. Yes, I've never denied that.

Q. Such that you might lose control of your actions and hurt the child?

A. No.

Q. Did you ever have any feelings like that, even close to that?

A. No, no, not at all.

Q. I suggest that what the concept or idea that you are describing in that entry is being placed under such stress that you snapped and were a danger to your children. What do you say?

A. I say no to that.[429]

* * *

Q. So, at the times that you were having a battle of wills with Sarah, were those the times when you "snapped your cog"?

A. No.

Q. Not--

A. No, not all, no.

Q. Not at all?

A. I wouldn't say not at all but, no.

Q. Was there ever a time when you were sleep-deprived and battling wills with Sarah that you "snapped your cog" as you refer to?

A. As in, being frustrated with her lack of sleep of routine, then, yes.

Q. Well, see, frustration is something that probably goes on in your mind, isn't it? It's a state of mind, is it not?

A. Frustration? Yes, yeah.

Q. "Snapping your cog" suggests some kind of action, doesn't it?

A. Not to me.

Q. Doesn't it suggest losing control?

A. It's a loss of control, yes. "Frustration", "loss of control", "anger", "snapping cog", all these references I don't differentiate between.

Q. But they're not one in the same concept, are they?

A. I believed at the time they were.

Q. You see, what we're talking about is the meaning of your use of words in these diaries, isn't it?

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[429] Transcript of the Inquiry, 29 April 2019 T650.38-651.23.

A. If you like, yes.

Q. And what I'm suggesting to you is that there's a distinction between feeling frustrated and snapping your cog. What do you say about that? They're different concepts, is the point I'm trying to put to you. What do you say about that?

A. At the time, I didn't believe – I didn't differentiate between them. If I was slightly frustrated, that equalled me being out of control in some fashion, which equalled me snapping my cog. There was no differentiation for me.

Q. I suggest to you that you used the term "snapping your cog" as a mitigating term for something that you had done to Sarah in order to stop her living. What do you say about that?

A. No, I won't agree with that at all.[430]

* * *

Q. "She'll leave me now like Sarah did." And there you, "I knew I was short-tempered and cruel sometimes to her and she left with a bit of help."

A. Because I thought, as I stated, I've already stated short-tempered and cruel could mean a variety of different things.

Q. I was going to ask you that; what did it mean with Sarah? How did you express your short-temperedness and cruelty to Sarah?

A. In being frustrated with her and not handling why she was crying or not being able to go to sleep.

Q. What cruel things did you say or do to her?

A. Not cruel as in inflicting harm in any way. I'm not talking by, cruel as in leaving them alone to, so you'd walk away or cruel as in not meeting their needs or cruel as in not understanding how to fix something and it, to me it all meant the same thing.

Q. "Cruel" is a very strong word for you isn't it?

A. Yes. It's a strong word I use in there yes.

Q. What did you do to Sarah?

A. I didn't do anything to Sarah.

Q. When you were short-tempered and cruel with her?

A. I didn't do anything to her.

Q. Well, you say, "I knew I was short-tempered and cruel sometimes to her." "To her"?

A. Yes, I was short-tempered. I was irritable. I was cranky.

Q. And cruel?

A. Not as in an action. No.[431]

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[430] Transcript of the Inquiry, 29 April 2019 T638.7-50.

[431] Transcript of the Inquiry, 30 April 2019 T769.17-47.

261. Fourthly, she believed that she could avoid subsequent deaths, namely of Laura, by better anticipating and managing her depressive mood. She believed she could do so by asking for more help, from Mr Folbigg and others:

> *Q. And the dark moods were what led to the killing of your children?*
>
> *A. My dark mood is as I've stated before, when I say dark mood I'm referring to when I'm depressed.*
>
> *Q. And well didn't these dark moods come on you without warning, with very little warning and even surprise you?*
>
> *A. Occasionally yes.*
>
> *Q. And they shocked you and you were shocked at what they brought about after they had passed, isn't that right?*
>
> *A. No I'm not going to agree with that, that they shocked me.*
>
> *Q. Well you go on to say, "Hopefully preparing myself will mean the end of my dark moods or at least the ability to see it coming and say to him or someone, hey help I'm getting overwhelmed here, help me out"?*
>
> *A. Yes, as in I'm getting depressed and that I will require help at that time.*
>
> *Q. You don't say depressed, even though you do say depressed a lot of times in the journal, you don't say that here do you?*
>
> *A. My reference to a dark mood is a reference to depression.*
>
> *Q. This is worse than that I'd suggest to you, this is a dark and murderous mood?*
>
> *A. No.*
>
> *Q. Because then you say, "That will be the key to this baby's survival, it surely will"?*
>
> *A. As in reference to me being, if I'm not as depressed I can give 120% to my child and her needs, then yes that is what that's referring to.*
>
> *Q. The key to survival, the one thing that keeps baby alive that means doesn't it?*
>
> *A. It's an extreme way of thinking about it but my diary is full of extreme thoughts, they're not, at times not even making sense to myself, so--*
>
> *Q. Madam you always made sense to yourself in these journals didn't you?*
>
> *A. Not all the time no.*
>
> *Q. There was no point--*
>
> *A. There's many a time I've said in my journal, that's rather silly or along those lines, realising and accepting that I've just said something that is totally silly.*
>
> *Q. You may have looked back in hindsight years before and thought that things were silly, in hindsight, that may have happened, correct?*
>
> *A. Yes, of course.*
>
> *Q. But when you were writing these entries, you were being very sincere with yourself I'd suggest, for how you felt at that time?*
>
> *A. Yes okay.*

Q. And you knew that the key to the baby's survival, the one thing that it was essential to do, was to have someone help you see the dark moods coming, someone to be with you to identify that, what do you say?

A. Someone to help me at the time when I'm feeling depressed and lonely and unsure of myself, then yes I would've liked that.

Q. But there's nothing indefinite about this, "That will be the key to this baby's survival, it surely will", is there?

A. I'm convincing myself there that whatever I'm doing in my preparation for this is going to work and Laura will survive and everything will be well.[432]

262. Fifthly, when she referred to a fear of being alone with the child, and things happening again, she was not meaning that she was afraid of losing control and hurting a child when alone with it. Rather, she was meaning she was afraid of experiencing a depressive mood again, and of that mood impacting on the child such that a "spiritual", "mystical", "metaphysical" force or "higher power" of some kind would take the child away, or the child would decide to leave her and die. She was additionally or alternatively referring to being afraid of being alone while finding a dead child:

Q. So you're expressing this idea or thought that because you were short tempered and cruel to her, to Sarah, that she left this world?

A. Yes, that's how weird my belief had gotten.

Q. What, she decided that she wanted to go into another world because she couldn't put up with your short temperedness and cruelty towards her, is that it?

A. Cruelty, the word cruelty, I need to also clarify that to me that's like if you leave your child to cry for too long, I figured that and deemed that as being cruel. I'm not talking cruel as in a cruel physical action on anything. Short tempered, yes, it goes with being frustrated. If you're frustrated you get a little short tempered.

Q. So you weren't using those terms in any - just a sort of minimal kind of way in the way you've described--

A. Yes.

Q. --short tempered and cruel?

A. Yes.

Q. But it was enough in your mind for her to get the idea, "Look, I don't want to be around this mother anymore, I'll simply die." Is that what you're trying to say?

A. Because by the time, by the time Laura came around that's where most of my thoughts were. They were dark and they weren't very pleasant…

Q. Then you say "with a bit of help", do you see that?

A. Yes.

Q. So you're saying there that you were that bit of help?

[432] Transcript of the Inquiry, 30 April 2019 T737.21-738.36.

A. No, I'm saying that God, higher power, or another decision, or even my children Sarah deciding that she didn't want to say [sic] was the bit of help, not me.[433]

* * *

Q. --what do you mean by "metaphysical"?

A. I mean, the next sentence in that is "I think she was warned", which I – you're – I'm gathering you're going to ask about.

Q. Well, let's just explore that.

A. It's all connected.

Q. Hang on, do you believe in the metaphysical?

A. I've always believed that there is a spiritual or something else going on, fate, karma, destiny.

Q. So, you're saying there then that, she's good because some higher being has warned her that if she's not good she'll lose her life?

A. No, I'm, I'm merely thinking to myself that, you know, this goes back to – right back to the, the clairvoyant sort of thing, where I'm thinking, did the other children have little chats to her, was she – you know, there was a discussion about whether Laura's soul was even her own, at one point, between us and other family members.

Q. So, did the other dead children have a discussion with the live one, Laura? Is that what you just said then?

A. It's, it's a thought of which I had, yes.

Q. And did you believe it?

A. At the time, yes.

Q. And so, what, they told her to be good and she'd live, is that how one interprets that?

A. No, I, I, take that as though they told her if, if she was good, mum would be okay and mum wouldn't stress as much.

Q. Mum wouldn't get angry and snap her cog--

A. No--

Q. --is that what you meant?

A. No, I'm generalising, it's – no.[434]

[433] Transcript of the Inquiry, 29 April 2019 T669.13-50.

[434] Transcript of the Inquiry, 30 April 2019 T664.191-665.4.

* * *

Q. Can you explain to me what you were saying?

A. That sentence your Honour is where I switched from, I've been talking about comparing my children; how easy one is to the other and then I switched and said "she saved her life by being different" as in the thought of my beliefs when it came to a spiritual belief or clairvoyant sort of belief and that the children would have spoken to each other and, and all that sort of thing and but yes, Sarah, Laura being a bit different could, could save her life. I'm meaning could save her life, not that it did save her life because it didn't.

Q. I'm sorry, I'm not familiar with the clairvoyant beliefs or what you're talking about. Can you explain it to me in some way?

A. At the time, when writing these because I was searching for questions so hard and always wanting to know why I had a belief that fate, karma, God, a spiritual thing going on that there was another reason as to why all this was happening. And when I went to a clairvoyant, which was mentioned in the last 24 hours, that clairvoyant gave me the peace that my children and Sarah were happy and it was a belief that just was ingrained in me; that there was other things going on beyond my control and all the answers that I was seeking all the time, "she saved her life by being different" is my hope and dream that Laura being different would have saved her life but in the end it didn't.

Q. Are you saying to me that you believe that there was some supernatural power that took the other three children away from you and you were concerned that that same supernatural power would take Laura away from you, and that she saved her life by being different?

A. Yes.

Q. On that basis?

A. Yes, along those lines, yes your Honour.[435]

* * *

Q. Is that the view that you hold today, that some supernatural power took your first three children away?

A. Yes.

Q. You hold that view today?

A. Yes, I had no answers as to why my - I have survived my children and outlived my children, I was constantly trying to search for that answer.

Q. But you can understand that there is a difference between not having any answers and coming upon as an answer, a supernatural power?

[435] Transcript of the Inquiry, 30 April 2019 T752.7-35.

A. As, as your Honour was trying to, I thought your Honour was trying to understand the meaning of what I was trying to get across, the word supernatural I'm certainly not saying some ghost or entity or whatever came down and took my children, I'm saying that it follows along as a basis of trying to put together all the mystical and spiritual beliefs I had at the time and I still have now.

Q. So you still hold the view that some supernatural power took all of your children, or just the first three?
A. No, all of them.[436]

* * *

Q. Is it the case that the time that you were the most scared about was when you were alone with the baby?

A. Yes. And that's purely because when I found the children I was always alone.

Q. Were you?

A. I felt I was.

Q. But were you?

A. Technically no.

Q. Physically were you alone?

A. A couple of times Craig was there yes.

Q. Two out of the four babies' deaths, Craig was asleep in the house as well with you was he not?

A. Asleep, so therefore alone.

Q. You had been asleep as well had you not?

A. Yes.

Q. When the baby died?

A. Yes.

Q. Because you weren't awake when the baby died were you?

A. No.

Q. You were in the same position as Craig?

A. Yes, but--

Q. Both of you asleep?

A. He wasn't the one that found them. I was.

Q. What you are expressing in that entry is great fear of being awake and alone with the baby, what do you say about that?

A. I agree with that purely on the point that I was the one that always found them.

---

[436] Transcript of the Inquiry, 1 May 2019 T805.9-28.

Q. What you are saying is that the time of greatest danger to the baby is when you were with them. That's what you're saying isn't it?

A. No.

Q. That's what scared you the most. Is that right?

A. No.

Q. Because you'd snap a cog?

A. No.

Q. And because that's what you'd done before when you were alone with the babies that had already died, isn't that correct?

A. No.

Q. Isn't that the thought you're expressing in that diary at that point?

A. No. I'm expressing my fear. That's all it is.

Q. You were expressing a fear of being alone with the baby. That's what you're frightened of?

A. I'm expressing the fear that I was scared to death of finding my child not alive.[437]

263. Sixthly, her diaries at certain points contained separate and unconnected thoughts in the same paragraphs:

Q. Here is perhaps the clearest expression of the truth of what happened to your first three babies in any event, because Laura was still living at this stage, you are saying there that you know that there is nothing wrong with Laura and that Craig really doesn't need to have this fear about her, aren't you?

A. No, I would never presume to think that I should tell Craig he shouldn't have a feeling of any sort.

Q. You're saying, we didn't say, we have a morbid fear about Laura, you said "He has a morbid fear about Laura"?

A. An observation yes.

Q. So he on the one hand has a morbid fear, "Me" meaning yourself, "Well I know there's nothing wrong with her"?

A. In an attempt to try and ease his fear, yes.

Q. No, you are making the distinction, like the chalk and cheese distinction, that Craig, because he thinks his children are liable to die suddenly of natural causes, has a morbid fear about his fourth child, you on the other hand know there's nothing wrong with her, that's what you're saying isn't it?

A. Because it's what I desperately wanted, for there to be nothing wrong with her.

Q. You're saying there's nothing wrong with her because it was "me not them"?

A. Two separate thoughts, one--

---

[437] Transcript of the Inquiry, 29 April 2019 T655.30-656.38.

Q. In the same paragraph?

A. I do it all the time yes in the same paragraph, one is where I'm noting there is nothing wrong with my child, at that particular time she's very healthy, I had hope for a future with her, the second thought I'm always blaming myself for everything, so if anything was to go wrong I would've instantly blamed myself anyway because I was her mother.

Q. But that paragraph says "Craig is worried about Laura dying suddenly, I, on the other hand, know that she won't, because" in relation to the other children, it was me who killed them not them, that died?

A. No.

Q. That's what that means, very plainly isn't it?

A. No it's not very plain at all, no.

Q. It is very clear in the context of these journals isn't it?

A. No.

Q. "It was me, not them"?

A. I always thought it was me, always blamed myself.[438]

* * *

Q. Just while we're dealing with that particular passage, in the diary entry of 4 December 1996, you've written, "rather than answer being as before", could you explain to me what was the answer before that you're referring to?

A. "Rather than answer to before", is a separate thought again and it is a very dark thought and it's I didn't want to answer as in being the one to discover my children, I didn't want to do that again your Honour.[439]

* * *

Q. That wasn't the answer, that wasn't an answer because when you're saying "Rather than answer being as before", you're talking about how you answered your feelings of jealousy and anger before aren't you?

A. No, my diaries are quite - as I've said before, they can be random and I can turn from one thought to another in a split second. I can be discussing how well - I'm having coffee and a relationship with Craig in one sentence and, within a full stop, I can be going into something that's quite deep and dark, so--[440]

438 Transcript of the Inquiry, 30 April 2019 T756.16-758.11.
439 Transcript of Inquiry, 30 April 2019 T714.1-6.
440 Transcript of Inquiry, 30 April 2019 T714.10-16.

* * *

Q. We got to the stage, I'll just refresh your memory, the sentence before that was, "Plus, I'm ready to continue my family now. Obviously, I'm my father's daughter but I think losing my temper stage and being frustrated with everything has passed." You were asked about that in your original interview, about what you meant by, "Obviously, I'm my father's daughter." Do you remember that?

A. Yes.

Q. At 644 and 656 you said it meant to you that, "My father is just like a big total loser." 656, "Thinking I was a loser of some kind, just a passing thought." Do you accept that they're the answers that you gave?

A. Yes.

Q. As to what you were referring to when you said obviously you're your father's daughter?

A. Yes, I believed and thought at the time that my father's actions ruined my life and my life never seemed to go right from there. And it was a thought of, along the lines of sins of the father being on the daughter. Was I paying the price?

Q. What was the sin of your father?

A. My father killed my mother.

Q. You thought the sin of the father might come through to the daughter?

A. I believed at the time, yes that the, as I said by the, writing these diaries in preparation for Laura, everything was very dark and every thought was very dark and I blamed my father a lot for most of my life just going wrong.

Q. But you see, what you're talking about there, there are three concepts there. You're talking about being ready to continue your family. Then you say, "Obviously, I'm my father's daughter" and then you say, "But, I think losing my temper stage and being frustrated with everything has passed." So, you're saying there aren't you, that you think you're ready to have another family because the losing your temper stage and being frustrated has passed. Is that right?

A. No, number one is I thought I might have been ready to have another family. The second thought is about my father and how my life never went right after that. And the temper and frustration bit was me lamenting and desperately trying to seek answers and trying to control and make sure that everything went right and this attempt at having another child went the way it was supposed to.

Q. But you see, your father and you knew this at the time, had killed your mother?

A. Yes.

Q. Your understanding was that he'd killed her as a result of losing his temper?

A. Yes.

Q. What you're saying there is that you're ready because you won't lose your temper anymore like your father lost his temper and killed your mother. That's what you're saying aren't you?

A. No.

Q. You see, I put it to you that this reference to you just meant your father was a loser, doesn't make any sense at all in the context in which you say that, "Obviously I'm my father's daughter." What do you say?

A. That there are two separate thoughts there. I'm ready for my family now and then I've randomly thought because of thinking of family I've randomly thought because of my father, how life has gone all wrong. And then I've jumped back to going back to did the sins of my father visit on me as in me being frustrated in any way whatsoever and then my inability to control absolutely everything in my life at the time. I have to make sure. I was preparing and had to make sure that all went well. [441]

* * *

Q. You've said now if you feel jealousy or anger you'd just leave Craig and the baby, right?

A. Jealousy is referring to - they're two separate thoughts there. They're broken up by the word "or", okay. Jealousy is referring to, it was a recollection of how Patrick was, even though I cared for him all the time, he was more Craig's boy, Sarah was my girl, and Laura ended up being my girl. The jealousy refers to that. It's not a jealousy as in me being jealous of my children. I was never ever jealous as such of my children.

Q. Is the word "jealousy" in relation to you, feeling jealous?

A. No, the word "jealousy" is a, is a reflection as the difference in, between the children, and how, you know, you can have a daddy's girl, you can have a mummy's girl. It's a, it's a different reference. Then it's broken by the word "or" and "anger too much", well as I said I didn't back then differentiate between frustration, anger, annoyance. It was all the same to me.

Q. But where does jealousy come into that explanation? I just don't understand that.

A. As I say, if I had any feelings of the jealousy as in just with, you know, was this child going to be more Craig's or more mine, you know, and to – if it fell into any sort of frustration or an anger situation, then yes, I felt that I would leave Craig and leave the baby with Craig. Again it's another fleeting thought. These are not set in stone. They were never set in stone. All my thoughts were just random thoughts.

Q. But do you accept that it was a feeling of jealousy that you had?

A. Not jealousy as in a jealousy that I would do anything about. It's a fleeting, it's a fleeting thought.

Q. You would do something about it. You'd leave Craig and the baby.

A. But I never did.

Q. You see, I want to get - to try to understand what you've said in your diary. You say that if you had any feelings of jealousy, too much that you'd leave Craig and the baby.

A. Jealousy is also--

Q. Is that right?

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[441] Transcript of the Inquiry, 29 April 2019 T672.21-673.35.

A. Jealousy is also as I said, it's, it's, it was a reflective thought. It, it, it wasn't connected to the next phrase.[442]

264. In respect of the entry dated 16 May 1997 which Ms Folbigg had accepted during her interview with police concerned the night of Sarah's death and had been left to the jury as a potential lie and thus consciousness of guilt evidence, Ms Folbigg gave the following explanation:

Q. "I really needed him to wake up that morning and take over from me", so are you referring to that morning when Sarah died?

A. I'm referring to the early morning, as in the day and just the thoughts that I felt like Craig did sleep too easily through a lot and that I just felt like he could've helped out more.

Q. But you see it's a cry of desperation almost isn't it?

A. (No verbal reply)

Q. Understand what I'm getting at?

A. I understand what you're getting at, yes.

Q. But you're feeling that you desperately needed him to wake up that morning and take over from you, and you're referring to the morning, the time that Sarah died aren't you?

A. I don't believe I am, I believe I'm being more general.

Q. Well what else can it mean?

A. That because of the sleep deprivation and being exhausted and tired, that I just felt I needed more help and I wasn't getting it.

Q. "Wake up that morning", what is "that", isn't that the morning that Sarah died?

A. It may have been.

Q. Well it is isn't it, that's what you're referring to?

A. I'm not, I'm not a hundred per cent clear on that.

Q. "I really needed him to wake up", let's just go back a bit, "Says he will stress and worry, but he still seems to sleep okay every night and did with Sarah, I really needed him to wake up that morning and take over from me", well clearly you're referring to the time that Sarah died aren't you?

A. I may have been, I'm not a hundred per cent clear.

Q. And you are saying that you were awake, "I really needed him to wake up that morning and take over from me", well that means you were awake, doesn't it?

A. I'm still not a hundred per cent clear that I'm referring to that morning that she died there in that reference. I'm still--

Q. You see--

[442] Transcript of the Inquiry, 29 April 2019 T651.37-652.29.

A. I believed and felt then that I wasn't getting the support that I thought I wanted and yes I was having a frustration with how easily I think Craig could sleep, compared to me and that I needed care and help with Sarah.

Q. You were awake with Sarah that morning, weren't you?

A. If I was caring for her then yes I most likely was.

Q. And that was the morning she died wasn't it?

A. I can't say with any clarity on that.

Q. And you were saying there that you were awake when Sarah died, aren't you?

A. No.

Q. That's what it means doesn't it?

A. No it doesn't.

Q. And you're trying to give it a different meaning I suggest to you, what do you say?

A. No, it doesn't mean that at all.

Q. Because that's not what – you haven't told the police or – you say that you went to Sarah and she was dead and that you weren't awake at the time, the actual time she died, that's what your position is isn't it?

A. Yes.

Q. But I suggest to you that this diary entry demonstrates something different to that?

A. I'm not agreeing with that.[443]

…

JUDICIAL OFFICER

Q. Can I just ask you this. In that passage that's been read out to you, it says you wish that somebody would wake up that morning and "take over from me" which tends, seems to indicate you want them to take over from you?

A. The care, yes, the general care of my child, yes.

Q. But is your explanation for this passage that you wanted somebody to be there when the baby was found to have died?

A. Yes, because by this stage your Honour I had an absolute fear of that. I never wanted to go through that again.

Q. But what I'm pointing out, that you're saying you wanted somebody to take over from you, which seems inconsistent with just having somebody there to be there when you found the dead baby.

A. I'm, I'm not sure I understand the difference, your Honour. I'm still saying that I didn't wish to be alone in either situation.[444]

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[443] Transcript of the Inquiry, 29 April 2019 T657.5-658.24.

[444] Transcript of the Inquiry, 30 April 2019 T732.17-32.

* * *

Q. "Craig says he will stress and worry but he still seems to sleep okay every night and did with Sarah. I really needed him to wake that morning and take over from me."

A. Yes, and as I explained, taking over from me as in the care of my child

Q. Well, what—

A. Be it that it might be—

Q. --what was "that morning"?

A. Even if it was that morning.

Q. What morning?

A. I've been, you know I'm getting told that's the morning that she died.

Q. You wrote the entry in the journal madam.

A. Yes.[445]

Further evidence about Ms Folbigg's possession and disposal of the diaries

265. The evidence before the jury at the trial concerning Ms Folbigg's possession and disposal of the diaries has been canvassed in detail above. By way of very brief chronological summary:

    a. Mr Folbigg gave evidence at the trial that Ms Folbigg left the matrimonial home and moved into a flat in mid April 1999, about six weeks or so after Laura's death on 1 March 1999. He discovered a diary in her bedside table when he was packing up Ms Folbigg's things a few weeks later.

    b. Mr Folbigg initially approached police on 14 May 1999, and on 19 May 1999, went into the police station and provided two diaries: one consisting of entries between 1 February – 1 March 1989 (tendered in the trial as Exhibit L and one single entry from 20 February 1989 as Exhibit AK) and the other from 4 June 1996 – 5 June 1997 (tendered in the trial as part of Exhibit J).

    c. Mr Folbigg gave evidence that shortly after 19 May 1999, he met with Ms Folbigg and informed her he had given a diary to police. He gave evidence that her attitude towards him changed and she became friendlier. One month later she returned to the matrimonial home and their relationship resumed.

    d. On 23 July 1999, Detective Senior Constable Ryan invited Ms Folbigg to attend an electronically recorded interview. During that interview she said she had disposed of three diaries on Mother's Day that year (9 May 1999) and had not written in one since. She denied she threw them away after Mr Folbigg told her he had given a diary to the police.

    e. At the conclusion of the interview police informed Ms Folbigg they were going to execute search warrants on the matrimonial home and her flat. When asked whether she had any more diaries she said "I've just started a new diary and it's up at the house." At the matrimonial house she handed police a 1999 diary (Exhibit AZ in the Inquiry, not tendered at trial) and said it was "one that I bought yesterday".

[445] Transcript of the Inquiry, 30 April 2019, T733.6-21.

f. During the search of the matrimonial home police found another diary containing entries between 6 June 1997 and 10 April 1998 (tendered at trial as part of Exhibit J). When asked whether she wished to make any comment, Ms Folbigg said "I didn't know it was there, I thought it was gone."

g. During the search of Ms Folbigg's flat, police found the 1992 "May Gibbs" calendar diary with entries from 31 December 1991 to 3 May 1993.

266. The following further evidence was received in the Inquiry about Ms Folbigg's disposal of the diaries.

267. On 16 July 1999, a listening device at the matrimonial home had been activated pursuant to an authorising warrant.[446] Detective Senior Constable Ryan examined recordings of conversations between 16 July – 4 August 1999 between Ms Folbigg and Mr Folbigg, whose voices he recognised from his dealings with them, and prepared transcriptions of certain conversations.[447]

268. The listening device recorded the following conversations which were not part of the evidence before the jury but were tendered into evidence in the Inquiry. At the time of the Inquiry only the transcripts of the conversations were available.

269. On 22 July 1999 at 10:15pm, the evening before Ms Folbigg's interview with police, the listening device recorded a conversation between Mr and Ms Folbigg in which Ms Folbigg was recorded as saying:

> *I just thought I'd tell you that even though I'm writin' in this diary I've decided that if you want to read it you can… being getting in the habit of writing in the diary and then not tell you about it.*[448]

270. At 9:40pm after the search warrant was completed, the listening device recorded a conversation between Mr Folbigg and Ms Folbigg which was transcribed by police as follows:

> *Craig: I came home to get the diary and, take it back to work and I couldn't find it.*
>
> *Kathy: I couldn't find it and they found it… and I've gone, I didn't have it so…*[449]

271. On 24 July 1999, at 7:53am police recorded the following in a transcript of the listening device recording:

> *MRS FOLBIGG ENTERS BEDROOM AND APPEARS TO OPEN A CUPBOARD OR DRAWER CAUSING A LOUD NOISE.*
>
> *Kathy: "I should have fucking done what I was gunna do, stuck it underneath that."*
>
> *Police believe Mrs Folbigg was talking about the diary police found during the search warrant.*[450]

272. Also received in the Inquiry but not before the jury was a statement of the police officer who located the 1997 – 1998 diary at the matrimonial home. He confirmed he had located the diary inside the built-in wardrobe, specifically inside a crocheted carry bag wrapped in clothing, which was contained inside a blue plastic container which was on the floor inside the wardrobe.[451]

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446 Exhibit BS, Statement of Detective Senior Constable Bernard Ryan (19 November1999) p 1.
447 Exhibit BS, Statement of Detective Senior Constable Bernard Ryan (19 November1999) pp 3-4.
448 Exhibit AZ, Diaries tender bundle, p 295.
449 Exhibit AZ, Diaries tender bundle, p 552.
450 Exhibit AZ, Diaries tender bundle, p 559.
451 Exhibit AZ Diaries Tender Bundle, Statement of Sergeant John Gralton (24 August 1999) p 6.

273. During the course of her oral evidence in the Inquiry, Ms Folbigg gave a more comprehensive account of her possession and dispossession of the diaries than in her police interview.

274. It was Ms Folbigg's account that once she finished a diary it became "irrelevant", she "moved on to another one" and "didn't think of that particular diary any further".[452] She later said she had no pattern in terms of getting rid of or retaining diaries.[453]

275. When asked about specific diaries that were available, she said of the 1989 diary regarding Caleb that it was one of the few things about him she had so she kept it.[454] In relation to the 1992 diary which included events in Sarah's lifetime, and was located by police at the flat she had been living in, she said she did not recall any specific reason for keeping it.[455]

276. In relation to the 1996 – 1997 diary found by Mr Folbigg, and the 1997 – 1998 diary found by police, she said she had no recollection of what she had done with those once she finished writing with them, whether she had misplaced them or moved them about the house.[456]

277. In cross-examination by counsel for the DPP, Ms Folbigg said it did not concern her that Mr Folbigg had found the 1996 – 1997 diary, because she did not consider the statements in that diary about the death of her children to be "anything to be highly concerned about".[457]

278. It was Ms Folbigg's account that her diaries were not a concern for her such that she had to hide them.[458] She said they were never hidden: "people always knew I was writing in them, they were always in places where people could see them".[459]

279. In subsequent cross-examination by counsel for Mr Folbigg, she acknowledged that she was concerned that Mr Folbigg had read the 1996 – 1997 diary because "diaries aren't meant for other people to be reading".[460] She considered it was an invasion of her privacy when he read her diary.[461] She agreed this was why she was disturbed to the point of quite strong language when she realised that police had found one of her diaries during a search warrant.[462]

280. Ms Folbigg agreed that the diary discussed by her and Mr Folbigg on the listening device recording was the 1997 – 1998 diary seized by police, and that her recorded words indicated she knew of the existence of the diary beforehand. She said "Yes because Craig had already told me that he'd handed a diary in, I am assuming there may have been others around the house".[463]

281. Ms Folbigg denied however that in the listening device recording on the morning of 24 July 1999, after the police search warrant had been executed and the 1997 – 1998 diary seized, she was expressing regret at not having put the 1997 – 1998 diary in a more difficult place to find, stating her diaries were not a concern to her to have to hide them.[464]

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452 Transcript of the Inquiry, 30 April 2019 T699.40-43; 1 May 2019 T806.40-45.
453 Transcript of the Inquiry, 1 May 2019 T806.36-39.
454 Transcript of the Inquiry, 1 May 2019 T807.3-5.
455 Transcript of the Inquiry, 1 May 2019 T807.21-25.
456 Transcript of the Inquiry, 1 May 2019 T807.7-13.
457 Transcript of the Inquiry, 29 April 2019 T631.24-632.21.
458 Transcript of the Inquiry, 29 April 2019 T634.30-32.
459 Transcript of the Inquiry, 29 April 2019 T633.40-41.
460 Transcript of the Inquiry, 30 April 2019 T697.10-15.
461 Transcript of the Inquiry, 30 April 2019 T699.20-25.
462 Transcript of the Inquiry, 30 April 2019 T697.15-18.
463 Transcript of the Inquiry, 1 May 2019 T803.40-804.20.
464 Transcript of the Inquiry, 30 April 2019 T697.20-30.

282. Her evidence on the first day of her evidence before the Inquiry was that she did not know of that diary's location in the wardrobe where it was found,[465] and "[didn't] have a clue" what she had been talking about on the listening device recording.[466] When asked about the recording again on the third day of her evidence she said she still had no recollection at all.[467]

283. As to her account to police during the interview in July 1999 that she had disposed of three diaries on Mother's Day in 1999, before Mr Folbigg told her he had been to police and provided them with a diary, Ms Folbigg repeatedly stated she only recollected throwing out one diary.[468] She recollected the diary she disposed of was one she had been writing at the time in May 1999, which may have extended back to 1998.[469]

284. In cross-examination by counsel for the DPP she said that when she told police there were three diaries she meant that she was remembering possessing three.[470] When examined by counsel assisting she accepted that the transcript of the interview was to be read as her saying that she had thrown away three diaries on Mother's Day 1999, but maintained that giving evidence in 2019 she only remembered throwing out one.[471] She rejected the suggestion that it was more likely that she threw that diary away after she heard that Mr Folbigg had given a diary to police.[472]

285. Ms Folbigg said she never had an intention to "get rid of anything" and explained:

> *To me the diaries were not that important as in information wise, and they didn't concern me in any way, that okay he's handed this diary over, you know. There, there was no conscious thought from me to go, "Right, that's it, I've got to get rid of everything".*[473]

286. Ms Folbigg's attention was drawn to the inconsistency between her statement during the interview with police on 23 July 1999 that she had not written in a diary since Mother's Day in May 1999, and the existence of a diary written in by her in July 1999. She denied that she lied to the police and said:

> *When I got rid of or lost those diaries, I'd made a decision that I wasn't going to write in them but, as it turned out, I'd changed my mind and I decided to and I did.*[474]

Further evidence about Ms Folbigg's mental state

287. At the conclusion of Ms Folbigg's evidence I formed the view that aspects of her evidence had entered into areas which would be addressed appropriately by psychiatric reports.[475]

288. Accordingly, a psychiatric report recently prepared by Dr Michael Diamond at the instruction of Ms Folbigg's representatives, together with reports prepared by psychiatrists at the time of Ms Folbigg's sentence by Drs Michael Giuffrida, Bruce Westmore and Yvonne Skinner were received into evidence.[476] None of these reports was tendered in the trial, however the latter three were evidence on sentence.

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465 Transcript of the Inquiry, 29 April 2019 T632.44-633.27.
466 Transcript of the Inquiry, 29 April 2019 T634.18-24.
467 Transcript of the Inquiry, 1 May 2019 T802.30-45.
468 Transcript of the Inquiry, 29 April 2019 T623.8-18; 1 May 2019 T797.42-798.7.
469 Transcript of the Inquiry, 1 May 2019 T798.49-799.8.
470 Transcript of the Inquiry, 29 April 2019 T623.7-18.
471 Transcript of the Inquiry, 1 May 2019 T799.33-800.4.
472 Transcript of the Inquiry, 1 May 2019 T800.23-30.
473 Transcript of the Inquiry, 1 May 2019 T800.45-50.
474 Transcript of the Inquiry, 30 April 2019 T700.20-701.1.
475 Transcript of the Inquiry, 1 May 2019 T810.13-18.
476 Transcript of the Inquiry, 1 May 2019 T811.12-22.

289. Following the conclusion of the hearings, a further report of Dr Giuffrida was sought by the Inquiry, on the particular issue of Dr Diamond's diagnosis of Ms Folbigg with "Complex Post-traumatic Stress Disorder" ("Complex PTSD").[477]

290. This part of the report considers the psychiatrists' reports insofar as they are relevant to the interpretation of Ms Folbigg's diary entries and consequently the assessment of any doubt about her guilt. The reports are considered separately and in more detail in Chapter 10 insofar as they are relevant to the assessment of any doubt as to a matter that may have affected the nature or the severity of the sentence.

Reports of Dr Michael Giuffrida

291. In his 2003 report prepared for sentence at the request of Ms Folbigg's representatives Dr Giuffrida noted that Ms Folbigg disclosed she began writing her thoughts and feelings in diaries and journals from 1996 to 1999 but that these had been "extracted and taken out of context in a manner she hadn't intended".[478] She said the diaries "represented a way of coping with her distress as she had been unable to talk about it with her husband or in counselling".[479]

292. From his review of the material, including the diary entries provided, Dr Giuffrida concluded that at the time of writing in the diaries Ms Folbigg "was a greatly tormented and indeed exceedingly disturbed woman".[480]

293. He observed that throughout the diaries there is a:

> *Prevailing theme of intensely depressed mood, expressions of worthlessness and low self-esteem and repeated references to feelings of rejection and abandonment by her husband Craig and her family and friends.*[481]

294. She "had intensely ambivalent feelings and attitudes to the notion of pregnancy and motherhood" and was "always coping at the very margins of her capacity to bond, relate, provide and care for her children".[482]

295. At the end of his sessions with Ms Folbigg, Dr Giuffrida came to a conclusion that Ms Folbigg's case was a "very significant phenomenon" following the trauma she experienced as a young girl.[483] He thought this resulted in a profound and probably irreversible impairment of her capacity to develop any meaningful emotional bonding and attachment, which "contributed in some part at least to her total inability to relate, care for and protect her own children".[484]

296. In his additional report dated 10 May 2019 (discussed further below in relation to the assessment of doubt as to the nature or severity of Ms Folbigg's sentence) Dr Giuffrida did not give any relevantly different opinion.

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477 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019).
478 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 10.
479 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 10.
480 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 18.
481 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 18.
482 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 18.
483 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 20.
484 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 22.

Report of Dr Bruce Westmore

297. Dr Bruce Westmore also prepared a report at the request of Ms Folbigg's representatives at sentence.[485] As part of his examination of Ms Folbigg, he discussed some of the diary entries with her. She admitted to him that at times she did not cope with her children but denied that there were any "angry feelings" directed towards the children, explaining that these were more directed at Mr Folbigg.[486]

298. Dr Westmore considered the diary entries support the proposition that she was "a very over-controlled woman".[487] He noted that over-controlled people can be prone to episodes of extreme angry outbursts and that it is possible Ms Folbigg has personality characteristics of this type.[488]

299. Dr Westmore concluded:

> *Based on the assumption that she was indeed responsible for the death of her children, it is probable in my view that she displaced onto the children her own anger and frustration with the difficulties she was having with her partner. It is unclear to me to what extent childhood difficulties played any immediate role in her behaviours although her childhood history is likely to have influenced her personality development…*
>
> *Her own concerns about not being a good or adequate mother, combined with her personality difficulties and vulnerability and her problems dealing with emotions such as anger and depression and frustration are all likely in combination to have led her to feel she could not cope with the children and subsequently her acting towards them in a way in which caused their deaths.*[489]

Report of Dr Yvonne Skinner

300. Dr Yvonne Skinner prepared a report for the Crown in advance of trial regarding the availability of any psychiatric defence to Ms Folbigg.[490] She did not have the opportunity to examine Ms Folbigg.

301. On her review of Ms Folbigg's diaries, Dr Skinner found no evidence of formal thought disorder, nor any evidence of cognitive dysfunction or to suggest that Ms Folbigg might have been suffering a significant depressive disorder.[491]

302. Dr Skinner acknowledged that Ms Folbigg had an "emotionally disturbed childhood" characterised by an "unsatisfactory foster placement, institutional placement and later a foster placement that proved more satisfactory".[492] In considering the significance of Ms Folbigg's chaotic early childhood, Dr Skinner stated:

> *Most psychiatrists would agree the background history of such disturbance would lead to personality problems or possibly psychiatric disorder, but studies show that there is no recognisable link between such childhood emotional disturbance and a particular psychiatric disorder or psychological condition.*[493]

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485 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003).
486 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 2.
487 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 6.
488 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 6.
489 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 6.
490 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003).
491 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 9.
492 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 13.
493 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 13.

303. Dr Skinner was not able to find any evidence that Ms Folbigg suffered from a postpartum psychiatric disorder, nor any other psychiatric condition that might have affected her judgment or ability to cope.[494]

2019 report of Dr Michael Diamond

304. Dr Diamond was briefed by Ms Folbigg's representatives in the Inquiry to provide a psychiatric assessment report.[495]

305. Dr Diamond opined as to a significant and pervasive psychiatric diagnosis of Complex PTSD in Ms Folbigg. He did not define this disorder but noted Ms Folbigg has:

> *lifelong symptoms of emotional detachment, emotional numbing, difficulty trusting, engaging with others and experiencing periods of severe detachment to the point of dissociation,*[496]

following the severe disruption of the fundamental early life necessity for attachment, nurture and security, and a history strongly indicative of early childhood abuse, sexual and physical violence.[497]

306. Dr Diamond noted that he did not have the opportunity to go through the diary entries in any detail with Ms Folbigg and was therefore unable to produce any meaningful view as to their relevance.[498]

307. However, he suggested that the diary entries need to be assessed in the context of his diagnosis of Complex PTSD and the deep-seated psychological subjective experiences of Ms Folbigg.[499]

Submissions regarding the non-medical evidence

Submissions of counsel assisting

308. Counsel assisting submitted the following in relation to how the non-medical evidence at trial ought to be considered in light of the evidence in the Inquiry.

Ms Folbigg's account of the meaning of the diary entries

309. Counsel assisting submitted that Ms Folbigg's account in the Inquiry in 2019 about the meaning to be given to the diary entries was to, a significant extent, consistent with the account she gave to police in July 1999 insofar as:

    a. she held herself responsible for the deaths of the children because she thought she must not have tried hard enough or done enough as a mother, because they had all died;

    b. her reference to "terrible things" meant something benign like "an angry thought here or there";

    c. any frustrations she felt were never detrimental to the children;

    d. she had wondered whether it was her battling of the wills with Sarah which caused her death, by tiring her out to the point of her not wanting to wake up;

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494 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 13.
495 Exhibit BA, Report of Dr Michael Diamond (16 April 2019)
496 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 39.
497 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 38.
498 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 38.
499 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 41.

e. another child would not die because she would learn to accept offers of help and handle the responsibilities of motherhood better, including by being more mellow and less frustrated;

f. she wondered whether forces like fate and destiny played any role in the children's deaths;

g. she was afraid of being alone with a baby because "terrible things kept happening when I was by myself"[500]; and

h. that the reference to being her "father's daughter" was a general reference to being a loser, not a person who killed another.[501]

310. It was submitted that Ms Folbigg's evidence in the Inquiry did differ from her account to police in some respects. In the Inquiry, she suggested for the first time that she believed that Sarah and the other children may have decided to leave her and die, and that she was afraid of being alone when finding a dead child.[502]

311. More significantly, Ms Folbigg appeared for the first time to concede in the Inquiry, at one point under cross-examination, that she may have been awake with Sarah during the night/early morning before her death when Mr Folbigg was asleep.[503] This was in the context of suggesting that the entry "I really needed him to wake up and take over from me that night. This time I have already decided that if I ever feel that way again I'm going to wake him up",[504] was referring to her desire generally that Mr Folbigg would assist her more with the care of Sarah.[505]

312. In her interview with police, which did not assume the form of cross-examination, she had said she could not explain the words "take over from me that night",[506] that the two sentences referred to different thoughts, and she thought she was talking about general mothering care, and why could not Mr Folbigg have woken up and found Sarah instead of her.

313. Counsel assisting submitted this apparent concession in her 2019 evidence was entirely consistent with a plain reading of the entry, namely that Ms Folbigg was awake with Sarah at the time of her death and needed Mr Folbigg to wake up and take over from her because she was having feelings of frustration and loss of control, which led to her smothering the child.[507]

314. Counsel assisting noted that at the trial the Crown submitted that Ms Folbigg had deliberately lied about the meaning of this entry, and that the true meaning of this entry was "if only Craig had got up and taken over from me Sarah would not have died".[508] The trial judge directed the jury that they could consider a consciousness of guilt finding in respect of Ms Folbigg's answers about this entry.[509] It was submitted that direction was properly given.[510]

315. To the extent that Ms Folbigg's account of the meaning to be attributed to diary entries relies on an interpretation of the words written other than their plain and ordinary meaning, counsel assisting submitted her account should be rejected.[511]

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500 Exhibit E, ERISP of Kathleen Folbigg Q726.
501 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [93].
502 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [92].
503 Transcript of the Inquiry, 30 April 2019 T733.29-35.
504 Exhibit AZ, Diaries tender bundle, p 162.
505 Transcript of the Inquiry, 29 April 2019 T733.9.
506 Exhibit E, ERISP of Kathleen Folbigg Q766.
507 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [97].
508 20 May 2003 T126.
509 20 May 2003 T124-128.
510 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [98].
511 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [99].

316. As set out above, Ms Folbigg said repeatedly that words like "frustrated", "angry", "terrible" and "cruel" all meant more or less the same thing, that is, she meant the same things by different words.

317. Having maintained this account about similar meaning throughout her evidence, she accepted in concluding examination by counsel assisting that the ordinary plain meaning of the words that she sought to be interpreted as meaning the same thing, are in fact different to one another.[512] Her explanation for this was that at the time of writing the diaries, "that's not how my mind worked".[513]

318. It was submitted that Ms Folbigg was plainly acutely aware of the significance of the interpretation of the content of the diaries at the trial, and in the Inquiry. She said, "it was a major part of how my guilt was decided"[514] and:

> *I may be in prison for the fact that these diaries were used… I'm now trying to answer what everyone has been wanting to know and trying to help your Honour in this Inquiry.*[515]

319. Ms Folbigg agreed she had had 15 years to think about the diaries.[516] She also agreed she had been reading the diaries very carefully over the month or two since she had received copies in the course of the Inquiry.[517]

320. Ms Folbigg also agreed that it was almost impossible for her to have read and given evidence about the diaries without being affected by the significant events which she accepted had occurred since she wrote the 1997 – 1998 diary.[518]

321. She nevertheless denied that the evidence she had given to the Inquiry had been affected by those significant events.[519] When asked if she agreed that it is human nature to sit and give evidence taking into account all the events that had happened since she wrote the diaries (more than 20 years ago) she said:

> *My mind doesn't work that way… What I am saying is, these diaries that – and the extracts that have been constantly – and I'm – this is also – the extracts that we* [sic] *used in my trial are the extracts and the diary entries that everybody is always constantly talking about. I have been reminded of those for all the time that I've been in prison. But, as I said, I didn't actually get to read them myself again until it was presented to me to do so. And when presented with something that can trigger a memory, as such, then, yes, I'm sitting there in the moment thinking to myself, "Okay, why have I written this? Maybe I can help explain why I've written this". 'Cause the problem that I felt that landed me in the position that I'm in is assumptions being made and things being taken out of context and nobody understanding what it was I was trying to say when I was writing these diaries.*[520]

322. Counsel assisting submitted that while Ms Folbigg presented as confident, consistent and unwavering in her account over the two and a half days of giving evidence and being cross-examined in the Inquiry, it does not automatically follow that her account is to be accepted. It was submitted that her obvious motivation for me to accept an exculpatory account of the diary entries calls for her evidence to be assessed closely against the other evidence.[521]

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512 Transcript of the Inquiry, 1 May 2019 T795.39-796.5.
513 Transcript of the Inquiry, 1 May 2019 T795.45-46.
514 Transcript of the Inquiry, 1 May 2019 T793.1-10.
515 Transcript of the Inquiry, 1 May 2019 T792.45-49.
516 Transcript of the Inquiry, 1 May 2019 T791.40.
517 Transcript of the Inquiry, 1 May 2019 T791.43-792.22.
518 Transcript of the Inquiry, 1 May 2019 T792.29-32.
519 Transcript of the Inquiry, 1 May 2019 T792.41-49.
520 Transcript of the Inquiry, 1 May 2019 T793.32-50.
521 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [106].

323. Counsel assisting submitted that Ms Folbigg's account of the diary entries as illustrating a benign interpretation is implausible and should be rejected, given:[522]

    a. the inconsistency between the benign meaning contended for by Ms Folbigg in her evidence, and the outward behaviours of frustration and anger which Mr Folbigg (in respect of Sarah and Laura)[523] and Ms Bown (in respect of Laura)[524] gave evidence of at the trial and which Ms Folbigg accepted in her July 1999 police interview that she had shown (to an extent, such as yelling);[525]

    b. Ms Folbigg's account to police in July 1999 that "to me losing your temper is well, totally losin' it. I'd regard losing your temper as sort of smashing things or throwing things or doin' something";[526]

    c. Ms Folbigg's acceptance to police in July 1999 of having experienced feelings of frustration with the children, in particular Sarah and Patrick;[527]

    d. the lack of ambiguity in the meaning of the entries when considered in their context and with regard to evidence of the surrounding circumstances;

    e. the private expressive and venting purposes which Ms Folbigg ascribed to her diaries;

    f. the absence of evidence of Ms Folbigg having expressed her beliefs to anyone during the course of the children's lives and deaths;

    g. the fanciful nature of the additional explanations which featured in Ms Folbigg's 2019 evidence; and

    h. the apparent concession under cross-examination that she was awake with Sarah before her death while Mr Folbigg was asleep.

The psychiatric expert opinion evidence in relation to the diaries

324. Counsel assisting submitted that the opinion evidence in the Inquiry of those psychiatrists who met Ms Folbigg as to her mental state does not provide a basis on which to interpret the diary entries in an exculpatory manner. It was submitted that insofar as the opinion evidence identifies in Ms Folbigg difficulties in her ability to relate to, cope with, and form attachments with her children, the opinion evidence supports an inculpatory interpretation of the diaries, consistent with a plain reading of the entries.[528]

325. Dr Giuffrida relevantly opined that the trauma Ms Folbigg experienced as a young girl resulted in a profound and probably irreversible impairment of her capacity to develop any meaningful emotional bonding and attachment, which "contributed in some part at least to her total inability to relate, care for and protect her own children".[529]

326. Dr Westmore relevantly opined that "her childhood history is likely to have influenced her personality development"[530] and referred to her concerns about not being a good or adequate mother, personality difficulties and vulnerability, and problems dealing with emotions such as anger, depression and frustration.[531]

327. Counsel assisting recognised that the reports of Dr Westmore and Dr Giuffrida were prepared on the assumption Ms Folbigg was guilty and took them into account only insofar as necessary to consider an inconsistency in diagnosis.[532]

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522 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [107].
523 2 April 2003 T123.21-28, T126.10-30, T127.50-57 (Sarah); T162.4-22, T171.17-38, T172.27-173.9 (Laura).
524 16 April 2003 T769.25-53, T770.25-771.12.
525 Exhibit E, ERISP of Kathleen Folbigg Q527, 657, 685, 802, 807, 813, 818.
526 Exhibit E, ERISP of Kathleen Folbigg Q657.
527 Exhibit E, ERISP of Kathleen Folbigg Q481-484, 527, 657, 685.
528 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [108].
529 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 22.
530 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 6.
531 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 6.
532 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [111].

328. Counsel assisting submitted these opinions were in effect consistent with the opinion of Dr Diamond that Ms Folbigg has lifelong symptoms of emotional detachment, emotional numbing, difficulty trusting, engaging with others and experiencing periods of severe detachment to the point of dissociation, following severe disruption and violence in childhood.[533]

329. It was submitted that additionally, each of the expert's opinions was reflective of Ms Folbigg's own evidence in the Inquiry, that she had not bonded or developed a maternal instinct with Caleb, Patrick or Sarah.[534]

Possession and disposal of the diaries

330. In her interview with police regarding her disposal of her diaries Ms Folbigg said that she had "just got rid of them all" on Mother's Day, 9 May 1999.[535] When asked by police how many she was referring to, she said three.[536]

331. In her evidence before the Inquiry she said that she could only recall throwing out one diary.[537] She initially rejected the suggestion that her memory surrounding getting rid of the diaries would be fresher on 23 July 1999 than it is now.[538] But later she conceded that it was more likely that what she told police in 1999 about events around that time was more likely to be accurate than her evidence now.[539]

332. In her interview with police Ms Folbigg had also said that she had not written in a diary since 9 May 1999.[540] At the Inquiry she was taken to an entry made on 19 June 1999. Ms Folbigg explained that at the time of the interview on 23 July she did not recall writing in the diary on 19 June.[541] She accepted that what she told police was false but suggested that at the time of her interview she did not remember writing in it.[542]

333. Counsel assisting submitted this latter explanation is not plausible given only a few weeks had passed since the entry. Directly following the interview, she was told the police had a search warrant. She then disclosed the existence of the 1999 diary and provided it to them at the start of the search.[543]

334. Mr Folbigg gave police Ms Folbigg's 1989 and 1996 – 1997 diaries on 19 May 1999. Before the Inquiry Ms Folbigg accepted that he told her he had done so sometime between 19 May and 23 July 1999.[544] However, she did not accept that it was more likely that she had thrown the diaries out after finding out Mr Folbigg had handed in some of the diaries to police.[545] She instead suggested that she "hadn't placed too much importance on the fact that he did, because I wasn't thinking there was anything in them that I had to be highly concerned about."[546]

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533 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [112]; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 39.

534 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [113] Exhibit BA, Report of Dr Michael Diamond (16 April 2019) pp 13, 15, 19; Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 6; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) pp 21-22; Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) pp 9-10, 13; Transcript of the Inquiry, 30 April 2019 T747.12-27, T767.3-43, T770.39-50.

535 Exhibit E, ERISP of Kathleen Folbigg Q459-462.

536 Exhibit E, ERISP of Kathleen Folbigg Q463.

537 Transcript of the Inquiry, 29 April 2019 T621.44-45.

538 Transcript of the Inquiry, 29 April 2019 T624.35-37.

539 Transcript of the Inquiry, 1 May 2019 T798.25.

540 Exhibit E, ERISP of Kathleen Folbigg Q459.

541 Transcript of the Inquiry, 29 April 2019 T625.41-627.1.

542 Transcript of the Inquiry, 29 April 2019 T629.1-10.

543 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [117]; 28 April 2003 T965.11-966.10.

544 Transcript of the Inquiry, 1 May 2019 T796.32-37.

545 Transcript of the Inquiry, 1 May 2019 T800.28-30.

546 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [118]; Transcript of the Inquiry, 1 May 2019 T800.33-36.

335. Counsel assisting submitted this explanation is inconsistent with Ms Folbigg's diary entry of July 1999 where she says "Decided to start writing in a diary again. Have missed being able to vent regularly. I just pray it doesn't come back to bite me like my 97 one has."[547] This entry was made prior to Ms Folbigg's interview with police as this diary was seized by police following the execution of a search warrant directly after the conclusion of the interview. It demonstrates her concern about her diary entries even before her interview with police. Counsel assisting therefore submitted Ms Folbigg's explanation that she was not concerned about the diary entries is untruthful.[548]

336. The execution of the search warrants was followed by listening devices which recorded:

    a. a conversation that evening between Mr and Ms Folbigg:

        Craig: I came home to get the diary and, take it back to work and I couldn't find it.

        Kathy: I couldn't find it and they found it… and I've gone, I didn't have it so…[549]

    b. Ms Folbigg saying later that evening:

        prime example now as to why I shouldn't drop the fuckin' walls because I dropped them enough just to write stuff and now it's coming back to bite me in the arse;[550] and

    c. the following morning Ms Folbigg saying after opening a cupboard "I should have fucking done what I was gunna do, stuck it underneath that".[551]

337. Counsel assisting submitted that the inference plainly open to be drawn is that references in these statements were to the 1997 – 1998 diary found by police in which many of the inculpatory diary entries were found. Ms Folbigg accepted as much. Ms Folbigg's denial that she now has "no clue" what she was referring to in the listening device transcript of the morning after the interview counsel assisting said is implausible.[552]

338. It was further submitted that in light of the above inconsistencies (and bearing in mind the points in the chronology of events at which they occurred) it was open to me to accept that the evidence about Ms Folbigg's possession and disposal of the diaries indicates that she was lying because of her consciousness of guilt.[553]

Conclusion

339. Counsel assisting submitted that, noting the significance of the diaries to the Crown case against Ms Folbigg at trial, there had been a substantial change in the evidence about and surrounding the diaries since the trial.[554]

340. Ms Folbigg has now given evidence about various entries. Counsel assisting submitted that I would find that Ms Folbigg was untruthful in much of that evidence.[555]

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547 Exhibit AZ, Diaries tender bundle, p 290.
548 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [119].
549 Exhibit AZ, Diaries tender bundle, p 552.
550 Exhibit AZ, Diaries tender bundle, p 554.
551 Exhibit AZ, Diaries tender bundle, p 559.
552 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [121]; Transcript of the Inquiry, 29 April 2019 T634.1-38.
553 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [122].
554 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [123].
555 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [124].

341. It was submitted that I should be satisfied, having heard sworn oral evidence from Ms Folbigg as to the interpretation of the diaries, and received considered expert opinion evidence about her mental state, that it was well open to the jury at trial, to draw inculpatory inferences in the jury's interpretation of the diaries.[556]

342. Counsel assisting submitted that the inculpatory interpretation in relation to the diaries dated between 1996 and 1998, taking into account evidence of the surrounding circumstances, is that in the diaries Ms Folbigg was, among other matters, expressing firstly her regret at having smothered Caleb, Patrick and Sarah in circumstances where she succumbed to a momentary loss of control while experiencing significant frustration with them, and secondly her commitment to avoiding that situation recurring with Laura.[557]

343. Counsel assisting submitted that the effect of Ms Folbigg's sworn evidence to the Inquiry was to strengthen a hypothesis as to her guilt rather than the reverse. It was submitted that it followed that Ms Folbigg's evidence to the Inquiry did not give rise to any reasonable doubt about her guilt.[558]

Submissions of the DPP

344. The submissions of the DPP focussed on the evidence given by Ms Folbigg in the Inquiry and how her evidence in relation to the diary entries should be assessed against the backdrop of the circumstantial evidence led in the 2003 trial.[559]

345. It was the position of the DPP that certain diary entries made by Ms Folbigg should be viewed as confessions or admissions of guilt to causing the death of her four children by smothering them.[560]

346. The ultimate submission of the DPP was that upon consideration of all the available evidence, I would not have any reasonable doubt as to the guilt of Ms Folbigg and indeed that the circumstantial case against her is even stronger than it was at the 2003 trial, as a result of the evidence given by her before the Inquiry.[561]

Possession and disposal of the diaries

347. The DPP submitted I would find that Ms Folbigg had disposed of more than one diary and that she was not truthful in her evidence before the Inquiry when she asserted she could only remember getting rid of one.[562]

348. It was submitted that Ms Folbigg was "characteristically and deliberately vague" about her disposal of the diaries. Her answers were said to be unsatisfactory, and she was clearly trying to downplay the significance of the disposal of the diaries and reduce the number of diaries that she had thrown away or perhaps hidden.[563]

349. The DPP submitted I would find that Ms Folbigg lied to police in her 2003 interview about not writing in a diary since she disposed of her diaries on Mother's Day in 1999, and that she would not admit this lie before the Inquiry and was "typically evasive" in trying to avoid honestly answering the questions.[564]

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556 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [126].
557 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [127].
558 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [125].
559 Submissions of the ODPP to the Inquiry (24 May 2019) p 4.
560 Submissions of the ODPP to the Inquiry (24 May 2019) p 68.
561 Submissions of the ODPP to the Inquiry (24 May 2019) p 4.
562 Submissions of the ODPP to the Inquiry (24 May 2019) p 16.
563 Submissions of the ODPP to the Inquiry (24 May 2019) p 16.
564 Submissions of the ODPP to the Inquiry (24 May 2019) p 18.

350. The DPP further submitted that I would not accept Ms Folbigg's reasons for getting rid of the diaries as being either because she had decided to stop venting and writing in them as doing so reminded her of her problems, or because she had simply finished with them. It was submitted she got rid of the diaries for a different reason, namely that she did not want Mr Folbigg, or the authorities, to find and read her diaries, given the incriminating evidence they contained.[565] Her disposal of the diaries was said to be evidence of a consciousness of guilt because of the contents of the diaries.[566]

351. The DPP submitted further that the probative force of the damaging evidence about the disposal of the diaries was increased by Ms Folbigg's unsatisfactory evidence about the number of diaries she disposed of; that she got rid of them on Mother's Day; the unacceptability of the reasons she gave for the disposal; and the listening device conversation where she was clearly talking about not having hidden a diary better and was angry about this.[567]

Ms Folbigg's account of the interpretation of the diary entries

352. The DPP submitted the diary entries provide very powerful evidence implicating Ms Folbigg in the killing of her children.[568] It was emphasised that the entries are part of the "strands in a cable circumstantial case", such that the entries are not to be viewed separately and in isolation, but in combination with all other circumstances, in accordance with the established principle.[569]

353. It was submitted there was an inconsistency between the benign meaning of Ms Folbigg's words and actions contended by her in her evidence before the Inquiry, compared with the outward behaviours of frustration and anger that Mr Folbigg described seeing in his evidence at the 2003 trial. It was said that Mr Folbigg's observations were consistent with the feelings and emotions that Ms Folbigg expressed in her diaries.[570]

354. The DPP submitted that I should reject the position adopted by Ms Folbigg in her evidence before the Inquiry for a number of reasons. The most important reason, it was suggested, was that when read and understood according to the meaning of words in ordinary English language, the entries are clearly admissions of guilt.[571]

355. The DPP submitted that the many attempts by Ms Folbigg to explain away her entries in an exculpatory fashion wherever an ordinary meaning interpretation would give rise to an admission were "far-fetched" and flew in the face of the meaning of the words used and written.[572]

356. The DPP further submitted that Ms Folbigg's evidence should not be accepted because the explanations she gave about particular entries and their meaning both stretched credulity and were very often inconsistent.[573]

357. The DPP also submitted an important observation now available was that Ms Folbigg is an intelligent woman who understands word use and language very well, as demonstrated throughout the course of her evidence. The DPP suggested it could not be argued that Ms Folbigg may have used language loosely in her diary without fully understanding what she was saying.[574]

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565 Submissions of the ODPP to the Inquiry (24 May 2019) p 18.
566 Submissions of the ODPP to the Inquiry (24 May 2019) p 22.
567 Submissions of the ODPP to the Inquiry (24 May 2019) p 22.
568 Submissions of the ODPP to the Inquiry (24 May 2019) p 22.
569 Submissions of the ODPP to the Inquiry (24 May 2019) p 24.
570 Submissions of the ODPP to the Inquiry (24 May 2019) p 23.
571 Submissions of the ODPP to the Inquiry (24 May 2019) p 23.
572 Submissions of the ODPP to the Inquiry (24 May 2019) p 23.
573 Submissions of the ODPP to the Inquiry (24 May 2019) p 23.
574 Submissions of the ODPP to the Inquiry (24 May 2019) p 23.

358. By reference to individual entries, the DPP submitted that Ms Folbigg's explanations and reasoning were attempts to minimise the actual meaning of phrases she used. It was submitted that the only rational explanation for entries suggesting Ms Folbigg had lost control with her children on the "last times"[575], which led to them being in danger from herself, was Ms Folbigg telling herself she would hand over the next baby to prevent herself from harming it as she did to the previous three children when she lost control.[576]

359. It was further submitted that a proper analysis of the entries supported the inference that Ms Folbigg was desperate not to lose control and harm her fourth child as she had with the previous three children,[577] and had a heightened sense of understanding that she could pose a great deal of harm to her newborn child if her feelings of anger and jealousy returned as they had on previous occasions.[578]

360. In respect of entries which Ms Folbigg told the Inquiry contained "separate" or "random" thoughts, the DPP submitted these were extracts which revealed Ms Folbigg's innermost thoughts about the deaths of her children. It was said that Ms Folbigg's explanation was no more than a manifestation of her effort to have the Inquiry view the diary entries in a distorted way, removing the clear and unambiguous reading that she was responsible for the deaths of her children and that she was, at times, struggling with her guilt about this.[579]

The psychiatric expert opinion evidence in relation to the diaries

361. The DPP submitted that the evidence as to Ms Folbigg's functioning, intelligence and any symptoms or disorders operating on her is relevant when the Inquiry considers the evidence of Ms Folbigg about "separate" and "random" thoughts, and the meaning of the words used in the entries.[580]

362. Specifically, the DPP submitted it was remarkable and striking that the psychiatric reports lacked any diagnosis or symptom or other reason that could explain the way in which Ms Folbigg constantly strove to explain and characterise the diary entries in her evidence before the Inquiry.[581]

363. It was said by reference to the psychiatric reports that even though it appeared open that Ms Folbigg became increasingly depressed following the death of each child, and that she was going through at times significant degrees of inner turmoil, there was nothing to explain the evidence she gave in the Inquiry in relation to the meanings of the entries and how certain entries contained separate thoughts on different themes that were unrelated to each other as outlined above.[582] The DPP pointed to the following features of the reports:

    a. although Ms Folbigg had a very troubled and traumatic childhood, particularly in her very early years, this had not affected her intelligence, and Dr Diamond and Dr Giuffrida in particular remarked on her average to above average intelligence;[583]

    b. Ms Folbigg has no known cognitive or developmental disability;[584] and

    c. the reports contain repeated references to Ms Folbigg's lack of any symptoms of thought disorder.[585]

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575 Exhibit AZ, Diaries tender bundle, p 70.
576 Submissions of the ODPP to the Inquiry (24 May 2019) p 25.
577 Submissions of the ODPP to the Inquiry (24 May 2019) p 25.
578 Submissions of the ODPP to the Inquiry (24 May 2019) p 31.
579 Submissions of the ODPP to the Inquiry (24 May 2019) pp 48-52.
580 Submissions of the ODPP to the Inquiry (24 May 2019) p 57.
581 Submissions of the ODPP to the Inquiry (24 May 2019) p 57.
582 Submissions of the ODPP to the Inquiry (24 May 2019) p 67.
583 Submissions of the ODPP to the Inquiry (24 May 2019) p 68; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 34; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) pp 20, 21.
584 Submissions of the ODPP to the Inquiry (24 May 2019) p 57; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 24; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 21.
585 Submissions of the ODPP to the Inquiry (24 May 2019) p 57; Report of Dr Michael Diamond (16 April 2019) p 24; Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) pp 3, 14, 20.

Mr Folbigg's submissions

364. Mr Folbigg's submissions agreed with counsel assisting's submissions. Particular support was given to the submissions that Ms Folbigg's explanation that she was not concerned about the diary entries being read was untruthful, and that the effect of Ms Folbigg's sworn evidence in the Inquiry was to strengthen a hypothesis as to her guilt rather than the reverse, such that Ms Folbigg's evidence does not give rise to any reasonable doubt about her guilt.[586]

Ms Folbigg's submissions

365. Ms Folbigg's submissions were made having read the submissions of counsel assisting, the DPP and Mr Folbigg.

366. The main propositions on behalf of Ms Folbigg were as follows:

    a. the diary entries contained no unambiguous, clear or unequivocal admissions that Ms Folbigg murdered her children;[587]

    b. the diary entries are capable of more than one meaning when such context as there is available is taken into account;[588]

    c. in particular, Ms Folbigg's expressions of guilt could and should be understood as omission-based, rather than commission-based expressions of responsibility, by reference to her Christian upbringing, her belief system (with respect to fate, reward, punishment, and a higher or supernatural power or being), and a recognition that SIDS parents blame themselves for their children's deaths;[589]

    d. it matters not that Ms Folbigg's belief system was or is irrational. The point is that she did hold that belief system at the time of writing the entries, and maintains that belief system now;[590] and

    e. if there is an available natural cause of death for one or more of the children, any inculpatory meaning to the diary entries necessarily falls away.[591]

SIDS and grief

367. The submissions on this topic commenced with a submission that there should be recognition that, given Ms Folbigg's Christian upbringing and the recognition that parents blame themselves for the death of a child through SIDS or sudden death in infancy despite no actual wrongdoing on their part, that a parent may express responsibility for that death through a belief that any omission by the parent for the care for the child has contributed to the death.

368. It was submitted that the acceptance of responsibility for an omission is a natural aspect of the human frailty. It was further submitted that the examination of Ms Folbigg by counsel assisting, and counsel for Mr Folbigg and the DPP:

> *proceeded on the assumption that any expression by Ms Folbigg of responsibility for the children reflected an admission by her of a positive act that caused the death of the child.*[592]

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586 Submissions of Craig Folbigg to the Inquiry (24 May 2019) p 2.
587 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [26].
588 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [298].
589 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [2]-[10].
590 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [259], [307].
591 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [183], [203], [228].
592 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [12].

369. The submissions went on to repeatedly refer to an "omission/commission distinction" which was said to have been ignored in the cross-examination of Ms Folbigg and the submissions of counsel assisting, the DPP and Mr Folbigg.

370. Excerpts of Ms Folbigg's evidence in which she expressed guilt for the deaths of her children on the basis of "not being good enough"[593] and "the fact that I did not do something in any way whatsoever that could've helped prevent that in some way",[594] and on the basis of her "negative emotions" or "frustration", or "stress"[595] which "impacted"[596] on the children, were said to be statements of her perception of responsibility through her omissions.[597]

371. Additionally referred to as evidence of omission-based guilt were Dr Diamond's account of Ms Folbigg's persecution complex that she was being punished for not being perfect, and Ms Folbigg's account in the 2003 interview with police that her guilt referred to "as in did I try hard enough, did I do enough, was I where I was supposed to be, was I not trying hard enough".[598]

372. It was submitted that Ms Folbigg was not challenged on her statement of acceptance of responsibility on the basis of omissions, such that the only procedurally fair finding open is that many of the statements contained in the diaries are consistent with her explanation of responsibility based on her omissions. It was submitted that the further context available through the exhibits and trial transcript is consistent with Ms Folbigg's explanation.[599]

Context

373. Ms Folbigg submitted that the context in which the diaries were written, including her belief system and emotional state, and the chronology of events that preceded the diaries, was essential to understanding the meaning of the diary entries. The context was also submitted as directly relevant to any suggestion of reconstruction by Ms Folbigg in her evidence.[600]

374. It was suggested that Ms Folbigg had been denied forensic opportunities to explain such context because I refused an application at the commencement of Ms Folbigg's evidence that her counsel be permitted to lead evidence from her of "context as to how the diaries were generated and what the issues were in this woman's life at the time"[601], including:

> *the nature of her relationship with her adoptive family, the nature of her relationship with her husband, so on and so forth and some of the issues that were going on at the time.*[602]

375. It was submitted that, given the nature of the ensuing cross-examination, in the circumstances Ms Folbigg was exposed to procedural unfairness. Nevertheless, the submissions acknowledged there was "material tendered before the Inquiry to establish some (but not all) of the context in which the diaries were written".[603] That context was detailed in the submissions under the headings "Ms Folbigg" and "Background Evidence".

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593 Transcript of the Inquiry, 30 April 2019 T744.25-26.
594 Transcript of the Inquiry, 30 April 2019 T724.22-26.
595 Transcript of the Inquiry, 30 April 2019 T715.23.
596 Transcript of the Inquiry, 30 April 2019 T715.24.
597 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [22].
598 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [15]-[17]; Exhibit E, ERISP of Kathleen Folbigg Q719; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 18.
599 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [18]-[19].
600 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [35].
601 Transcript of the Inquiry, 29 April 2019 T617.50-618.1.
602 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [34]-[35]; Transcript of the Inquiry, 29 April 2019 T618.33-36.
603 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [36].

Ms Folbigg

376. Firstly, by reference to the psychiatric opinion reports, it was submitted that despite the terrible trauma suffered by Ms Folbigg in her life she had no psychiatric illness that would explain her killing of four children.[604] Also pointed to in this regard was Dr Giuffrida's experience that almost all of the women he had assessed who killed or attempted to kill their children had a "serious psychopathology",[605] which Ms Folbigg did not, and his opinion that she suffered pervasive depression and a sense of "failure, shame and guilt".[606] It was submitted that Ms Folbigg does not fit the class of persons who are likely to murder their children, and that she was notably remarkably conventional in her lifestyle, interests and aspirations.[607]

377. Secondly, the submissions conversely disputed Dr Giuffrida's assessment, based on Ms Folbigg's reports to him and the diaries, that Ms Folbigg had failed to experience any true sense of bonding or attachment to her children, and that she was definitely overwhelmed by what she perceived as the intense and insatiable demands of her infant children such that she was coping at the very limits of her capacity.[608] It was said this opinion did not match the "clinical history" of Caleb and Patrick, pointing to her diligence in taking them to appointments. In respect of Sarah it was said she had a "tussle" which was not unusual, and she cared for her and developed her own relationship with her. In respect of Laura it was said she strongly bonded with her. The submissions in this regard did not refer to Ms Folbigg's evidence in the Inquiry that she did not bond with or have a maternal instinct with her first three children.[609]

378. Thirdly the submissions also disputed Dr Giuffrida's assessment that Ms Folbigg's response to the children's deaths was characterised by an almost total absence of normal grief and bereavement. That assessment was based by Dr Giuffrida on "a very detailed history [from Ms Folbigg] of her relationship with her children and her response to each of their deaths". It was submitted that there was not a lack of bonding but there was "likely an impairment of bonding for which Ms Folbigg felt a failure", and that "in any event, [Dr Giuffrida] provides a rational explanation for such impairment of bonding in her childhood".[610]

379. Fourthly Ms Folbig's submissions were critical that Dr Giuffrida did not assess the possibility of an expressed view of responsibility in Ms Folbigg's diaries arising from her perception that her omissions may have contributed to their deaths.[611]

Background evidence

380. The submissions on behalf of Ms Folbigg referred at length to non-medical evidence, both from the trial and in the Inquiry, under the heading "Background Evidence". The evidence was referred to in a chronological fashion. The following aspects of the non-medical evidence were highlighted as "context" to the diary entries.

381. It was submitted that Ms Folbigg had a "traumatic and unusual childhood"[612] which involved the following:

    a. *the murder of her mother by her father;*

    b. *probable sexual assault as an infant;*

    c. *disturbed living arrangements with relatives and later foster parents;*

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604 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [37].
605 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 19.
606 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [28]-[39].
607 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [49].
608 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 19.
609 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [41]-[45]; Transcript of the Inquiry, 30 April 2019 T747.12-27, T770.39-50.
610 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [49]-[51].
611 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [52].
612 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [57].

d. inter-personal conflict with her foster parents; and

e. sexual proclivities that were not age appropriate.[613]

382. Mr and Ms Folbigg came to a fairly traditional domestic arrangement, with Mr Folbigg the primary income earner and Ms Folbigg the primary carer of the children. Each police officer who attended their home described it as well-maintained, neat and tidy with nothing to indicate neglect, which was consistent with Mr Folbigg's evidence.[614]

383. There was no evidence Ms Folbigg suffered from alcoholism or substance abuse issues at any time.[615] Ms Folbigg had sought assistance for trauma in 1999, including one consultation together with Mr Folbigg.[616]

384. In respect of Caleb's life and death Ms Folbigg was happy with the birth of Caleb, and happy to be a mother. Mr Folbigg did not notice any emotional problem with Ms Folbigg at the time of Caleb's birth or at any time proximate to his death. He observed she was calm, comfortable and diligent in her care for Caleb.[617]

385. The 1989 diary extended well beyond Caleb's death indicating she was planning a future for him. She took him to doctor's appointments as recommended. There was no sign of physical abuse, injury or overly zealous medical treatment of him. Mr Folbigg described Ms Folbigg as devastated when Caleb died. There was no evidence of a post-partum psychiatric disorder, or psychotic disorder or delusional beliefs, and no history of drug use or alcoholism at the time of Caleb's death.[618]

386. There were no direct diary entries relating to the death of Caleb let alone any admissions to his death, and no suggestion in the evidence of Mr Folbigg that Ms Folbigg ever had an issue of control or rage during the 19 days of Caleb's life. When Caleb died, Ms Folbigg grieved for him. She and Mr Folbigg received counselling from a SIDS organisation.[619]

387. When Patrick was conceived, both Ms Folbigg and Mr Folbigg were happy again. She appeared to be excited about the birth of a child and was euphoric when he was born.[620]

388. Mr Folbigg stayed at home with Patrick and Ms Folbigg and his evidence contained no statement about any loss of temper or control temporally associated with Patrick's ALTE, even though he had "every opportunity" to observe it while he was awake and in the presence of Ms Folbigg. Ms Folbigg seemed happy to be a mother and seemed to be enjoying motherhood. Mr Folbigg had no concerns or misgivings, there was nothing to suggest she was not coping with being a mother.[621]

389. The time after Patrick's ALTE was difficult. Ms Folbigg suffered the strain of bearing the brunt of care for Patrick but she persisted with it. Mr Folbigg observed her to lose her temper a bit with both Mr Folbigg and Patrick, and get frustrated. However she bathed and fed him and taught him things, and diligently attended to his needs.[622]

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613 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [57].
614 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [55].
615 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [56].
616 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [58].
617 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [61]-[62].
618 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [62]-[63].
619 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [65].
620 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [75]-[76].
621 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [77]-[78].
622 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [78], [81].

390. Mr Folbigg said Ms Folbigg kept things to herself and when he found her diary, concluded she was going through a lot of emotional turmoil. She talked of leaving Patrick with Mr Folbigg because of the considerable strain. Ms Folbigg admitted she was finding it difficult to cope at times. Despite her ruminations, she resolved to stay and accept her responsibilities as a mother.[623]

391. There was no evidence of violence or ill temper towards Patrick, and no evidence of abuse. When Mr Folbigg arrived home after Ms Folbigg telephoned him upon finding Patrick at the time of his death, she was crying. Police observed she was sobbing and screaming hysterically. Mr Folbigg's sister observed her crying. Patrick's death devastated both Mr and Ms Folbigg. There were constant arguments between the two of them about their differences in grieving.[624]

392. In respect of Sarah, the sleep monitor they used went off almost every night, which caused anxiety but was largely false alarms. Ms Folbigg became frustrated with the monitor and she and Mr Folbigg argued about it. He found it irritating but comforting.[625]

393. Mr Folbigg observed that Ms Folbigg enjoyed motherhood at times but at other times did not. She was a very rigid and regimented type of person, and became "harder" about things like Sarah's bedtime. The relationship between Mr and Ms Folbigg became highly strained at times as a result of her frustration with him for getting Sarah over-excited prior to bedtime, and the issue of sleep discipline. Ms Folbigg became frustrated with Sarah.[626]

394. Ms Folbigg also became very concerned about her appearance and her weight gain. Her diary entries about these issues were an example of her concern about control, and also demonstrated an underlying emotional vulnerability of Ms Folbigg.[627]

395. On the night of Sarah's death Mr Folbigg was awoken by Ms Folbigg screaming, and he observed her sitting in the hallway outside the door screaming and crying. Ms Folbigg hid herself away from the world at first after Sarah's death. From the point of Sarah's death the relationship was troubled. After Sarah's death Ms Folbigg wanted another child and after some time she conceived.[628]

396. In respect of Laura, Ms Folbigg was enthusiastic about her arrival, and seemed to be happy when she was born.[629] An alarm system was also used with Laura, which became a nuisance. The system caused tension between Mr and Ms Folbigg. Mr Folbigg wrote to the nurse Margaret Tanner and said he thought Ms Folbigg was "merely in trusting [sic] in Laura's survival to fate".[630]

397. This letter by Mr Folbigg was said to demonstrate that Ms Folbigg had told Mr Folbigg about her belief system insofar as "fate" was concerned. It was submitted there was no evidence from Mr Folbigg at trial that she had not discussed her personal beliefs with him, and he was not called as a witness in the Inquiry.[631]

398. Ms Folbigg was happy being a mother although she did get cranky, and this started to get worse after Laura started walking and "carrying on". At this stage Mr and Ms Folbigg's relationship was in trouble and most of the time they were sleeping in separate rooms.[632] Friends and neighbours observed that Laura was a happy child who was well cared for and well fed.[633]

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623 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [81]-[83].
624 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [83]-[88].
625 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [89].
626 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [89]-[92].
627 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [93].
628 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [95]-[99].
629 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [100]-[101].
630 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [102].
631 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [103].
632 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [105].
633 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [106].

399. There was an exchange of correspondence between Mr and Ms Folbigg in February 1999, after which Ms Folbigg agreed to give the marriage another go. By the time of Laura's death they had resumed sleeping in the same bedroom. There was an argument between them on the morning of Laura's death, yet Ms Folbigg took Laura into Mr Folbigg's work for morning tea.[634] After Laura's death Ms Folbigg was clearly devastated.[635]

400. Still under the heading "background evidence" further submissions were made about the relevance of Ms Folbigg's personality to the interpretation of her diary entries.

401. It was submitted that Ms Folbigg equated order and routine with "control" and when order and routine were lost, she perceived this as a loss of control. A focus on such issues was said to be unsurprising for a woman who suffered sexual abuse, trauma and emotional deprivation in her infant life.[636] It was submitted that if Ms Folbigg was convinced routine and order were an essential part of mothering, then "it is not unnatural for her to have equated those matters with a loss of control and in the absence of any clear medical cause for their deaths".[637]

402. It was further submitted that the fact of impaired emotional bonding does not equate with Ms Folbigg murdering her children, and that this approach requires a certain degree of impermissible speculation and imagining tainted with a presumption of guilt.[638]

403. Instead, it was submitted, the diaries must be read in the context of the apparent agreement between Dr Diamond and Dr Giuffrida as to Ms Folbigg having had and continued to have a "persistent post-traumatic stress disorder type syndrome".[639] In this context it was said to be in error to read the diaries through a prism of objectivity, rationality or clinical and impartial analysis based on the words used. It was submitted that "any statement in which she accepts responsibility for any of the deaths can be assessed through a prism of a reference to an omission or commission by Ms Folbigg".[640]

Reconstruction

404. The submissions on behalf of Ms Folbigg took issue with any suggestion of reconstruction in her evidence before the Inquiry about her "view that there was some question of fate, or blame or supernatural force that has governed her life and those of her children".[641]

405. It was submitted, by reference to the letter from Mr Folbigg to the nurse Margaret Tanner, that Ms Folbigg conveyed this belief system to Mr Folbigg before she was charged, and that the belief system was also evidenced in her writings in her diaries, and in her interview with police in 2003.[642]

406. The submissions cited the following parts of Ms Folbigg's evidence in the Inquiry to demonstrate her belief system:

> *Q. Are you saying to me that you believe that there was some supernatural power that took the other three children away from you and you were concerned that that same supernatural power would take Laura away from you, and that she saved her life by being different?*
>
> *A. Yes.*

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634 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [108].
635 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [106].
636 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [116].
637 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [119].
638 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [120].
639 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [124].
640 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [126].
641 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [131]-[132].
642 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [132].

Q. On that basis?

A. Yes, along those lines, yes your Honour.[643]

* * *

Q. You don't remember. Can I come to some evidence that you gave yesterday, now when answering a question put by his Honour, and this is at transcript 752 you were asked whether or not you believed there was some supernatural power, now this is 752 of the evidence that was given yesterday, if we can just have that on the screen, and you say, you were asked the question there at about line 28, "Now you're saying that you believe there was some supernatural power that took the other three children away from you, and you were concerned that the same supernatural power would take Laura away" and you answered "yes." Now is that a belief that you had when you wrote that entry in the diary?

A. Yes. I took your Honour questioning me about that as trying to understand what it is I'm trying to-the message I'm trying to get out and across, which was my belief of a higher power, be that God, Mother Nature, fate, destiny, karma, all of those things, metaphysical combined, and when your Honour said the word "supernatural", I believed it's along the basis of the thinking that I had at the time yes.

Q. Is that the view that you hold today, that some supernatural power took your first three children away?

A. Yes.

Q. You hold that view today?

A. Yes, I had no answers as to why my-I have survived my children and outlived my children, I was constantly trying to search for that answer.

Q. But you can understand that there is a difference between not having any answers and coming upon as an answer, a supernatural power?

A. As, as your Honour was trying to, I thought your Honour was trying to understand the meaning of what I was trying to get across, the word supernatural I'm certainly not saying some ghost or entity or whatever came down and took my children, I'm saying that it follows along as a basis of trying to put together all the mystical and spiritual beliefs I had at the time and I still have now.

Q. So you still hold the view that some supernatural power took all of your children, or just the first three?

A. No, all of them.

Q. So took Laura as well?

A. Yes.

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[643] Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [133]; Transcript of the Inquiry, 30 April 2019 T752.28-32.

Q. And you also gave evidence of your beliefs that the children who had died, communicated with Laura, who was then alive and warned her as to how she should behave, do you remember giving evidence to that effect yesterday?

A. Yes.

Q. And is it the case that today, you believe that that occurred?

A. Yes, still believe that yes.

Q. Do you believe that they warned their sister to be good or else you might "crack it" or some other expression that you used?

A. I believed and believe that-how do I explain this, being brought up on the faith and the Christian based belief that I had as I was growing up as a child, I've always believed in once you've died, your spirit rests or goes to another place, or is at peace. I've always believed that when it came to my children, it turned out to be a necessary belief for me, that they could speak to each other or that they were peaceful, they were happy. And when I was pregnant with Laura, I believed that it was possible that the spirits of my children could have spoken to my child and they had discussions about things.

Q. So in relation to those two matters, the belief that you had then, as reflected in your diaries, is the same as the belief you have now, that's right?

A. Yes, it hasn't changed.

Q. Is there any other aspect of the diaries that you have a view now that is different from the view that you expressed back in the late 90s?

A. I don't think so, I'm not foreseeing, my core beliefs are my core beliefs.

Q. I'm not talking about your core beliefs, I'm talking about whether or not you have a view now that's different from what was expressed in your diaries back in the 1990s?

A. No.

Q. You don't?

A. No.[644]

(Emphasis as added in Ms Folbigg's submissions)

[644] Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [134] (emphasis added in submissions); Transcript of the Inquiry, 1 May 2019 T804.42-806.16.

407. This evidence was said to have a number of powerful features as follows:

    a. there was no challenge to the effect that Ms Folbigg did not hold those beliefs;[645]

    b. there was no doubt that Ms Folbigg has retained those beliefs, and to that extent there is no reconstruction on the issue;[646]

    c. Ms Folbigg's evidence on her diaries must be considered in the context of her belief system, and in the broader context as set out above;[647]

    d. Ms Folbigg was absolutely committed and unwavering in her answers despite the potential for people to lambast and ridicule her;[648] and

    e. such beliefs are also "widespread and part of human nature". Ms Folbigg's evidence was that her foster family were practising Christians and she was educated in that faith with an understanding of afterlife and concepts of sin.[649]

408. Having regard to these matters, it was submitted that any rejection of Ms Folbigg's evidence about her beliefs would require me to reject evidence that people could believe in God, guardian angels, fate, nature, destiny or karma. It was otherwise said it would be an error to interpret the diaries without due regard to Ms Folbigg's belief system, whether that belief system was judged by this Inquiry to be rational or not.[650]

The diary entries

409. A great deal of Ms Folbigg's submissions regarding the diary entries pointed to her expressions of her belief in matters such as fate, punishment, reward, a higher power, and her expressions of her self-blame for her failure as a mother. The submissions also pointed to aspects of the diary entries which demonstrated Ms Folbigg had no intention or desire to harm Laura.

410. Ms Folbigg's submissions about the entry of 14 June 1996 demonstrate the explanation offered to reconcile entries where Ms Folbigg apparently referred to acts by her, such as losing her temper and becoming frustrated and doing "terrible things",[651] with the omissions based guilt mindset and her belief system.[652]

411. It was submitted that the reference to "fate" and "the man upstairs" deciding her fate is linked to her behaviour and thought process. It was submitted that drawing a conclusion, by reference to losing her temper and being frustrated coupled with a higher being deciding she did not deserve to have a child, that she killed her children and was likely to kill her next child Laura would require me to accept that a loss of temper and frustration by a mother would most likely cause that person to harm their child. It was said this conclusion would have to be regarded as "absurd", because it would "simply be extraordinary for a parent not to have moments of anger and frustration from time to time and later feel guilty about it".[653]

412. It was also submitted that if the entry was assessed through Ms Folbigg's sense of responsibility for the death of her children through omission, the inculpatory meaning ascribed by the DPP, Mr Folbigg and counsel assisting falls away. It was submitted that entry was highly exculpatory, when considered in its proper context.[654]

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645 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [135(a)].
646 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [135(b)].
647 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [135(c)].
648 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [135(d)].
649 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [136].
650 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [136]-[140].
651 Exhibit AZ, Diaries tender bundle, p 114.
652 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [188]-[191].
653 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [175].
654 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [176]-[177].

413. In dealing with the 1 January 1997 entry in which Ms Folbigg said "stress made me do terrible things",[655] Ms Folbigg submitted this described an inculpatory interpretation as "selective over-interpretation".[656] The submissions then went on to point to the absence of evidence of inappropriate behaviour towards Caleb and Patrick, and suggested that if it is the case that a natural cause of death in one or all three of Caleb's, Patrick's and Sarah's cases cannot be excluded by the Crown, then the inference that in this entry Ms Folbigg was admitting to murder cannot be sustained.[657]

414. In dealing with the 16 May 1997 entry where Ms Folbigg wrote "night time and early mornings such as these will be the worst for me, that's when wishing someone else was awake with me will happen purely because of what happened before" and:

> *I really needed him [Craig] to wake that morning and take over from me. This time I've already decided if I ever feel that way again I'm going to wake him up,*[658]

it was submitted there is no clear inference available on this entry.[659]

415. It was submitted the entry likely relates to Ms Folbigg's fear of discovering her child dead, and does not permit an inference that it is any admission of murder or an inference she is referring to a blinding rage that may have led to murder.[660] By reference to her answer "that is a spontaneous decision I've made right there and then whilst writing that, that I would wake him up",[661] it was submitted the entry could clearly reflect a rumination of self-blame and doubt for an omission, said to be a common experience with SIDS parents.[662]

416. In respect of the entry dated 25 October 1997 in which Ms Folbigg wrote "I take the time to figure out what is wrong now, instead of just snapping my cog" and "she's [Laura's] saved her life by being different",[663] it was submitted the exculpatory interpretation "drifts back into Ms Folbigg's belief system that there is an omnipresent being that metes out reward and punishment for behaviour".[664]

417. It was submitted that Ms Folbigg's evidence that "snapping my cog" to her "could have been simply as even showing a slight frustration" was not unexpected in a person who has suffered the trauma of a SIDS death and has engaged in damaging self-blame and guilt at her acts and omissions which she feels has contributed to the death of her children. It was further submitted that "snapping my cog" is an "unknown metaphor therefore whatever interpretation Kathleen Folbigg places on it, is its meaning".[665] It was suggested that for a tribunal of fact or lawyers to purport to place a meaning on this metaphor, when it has no accepted meaning, is objectively inapproprfiate.[666]

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655 Exhibit AZ, Diaries tender bundle, p 114.
656 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [186]-[190].
657 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [197].
658 Exhibit AZ, Diaries tender bundle, p 162.
659 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [210]-[213].
660 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [213].
661 Transcript of the Inquiry, 30 April 2009 T732.37-38.
662 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [214]-[215].
663 Exhibit AZ, Diaries tender bundle, p 266.
664 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [239]-[242].
665 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [246].
666 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [245]-[246].

418. In respect of the 3 November 1997 entry in which Ms Folbigg wrote:

> *lost it with her [Laura] earlier. Left her crying in our bedroom and had to walk out. That feeling was happening again & I think it was because I had to clear my head & priotise [sic]… I love her, I really do, I don't want anything to happen,*[667]

it was submitted that the entry demonstrated Ms Folbigg's perception of "losing it" as including leaving her children crying in her bedroom.[668]

419. It was otherwise submitted that the most the entry showed was that Ms Folbigg loved Laura and did not want her to die, and demonstrated her own inadequacies and her "insistence that self-control will better her position".[669]

420. In respect of the 8 December 1997 entry in which Ms Folbigg wrote:

> *Much [sic] try to release my stress somehow. I'm starting to take it out on her. Bad move. Bad things & thoughts happen when that happens. It will never happen again",*[670]

it was submitted that this entry was nothing more than a mother expressing normal frustration at how life changes when having children.[671]

421. Dealing with the entry of 15 December 1997 in which Ms Folbigg wrote "Me well I'm sure she met everyone & they've told her, don't be a sickly kid, mum may you know crack it, they've warned her – good",[672] it was submitted the terms "crack it" did not suggest killing or murder. No submission was made as to the significance of the word "warned". The reference in the entry to Laura's "soul" was emphasised as demonstrating Ms Folbigg's belief system.[673]

422. Dealing with the 31 December 1997 entry in which Ms Folbigg wrote:

> *Wonder if the battle of the wills will start with her & I then… She's a fairly good natured baby – thank god, it… saved her from the fate of her siblings, I think she was warned,*[674]

it was submitted that "if nothing else, this entry showed that Ms Folbigg was looking forward to having fun with Laura in the coming year" and that it demonstrated her belief in fate. Again, no submission was made as to the significance of the word "warned".[675]

423. In respect of the 23 January 1998 entry in which Ms Folbigg wrote in respect of Laura "I've done it. I lost it with her" and "I knew I was short tempered & cruel sometimes to her [Sarah] & she left with a bit of help. I don't want that to ever happen again" and "only seems to happen if I'm too tired. Her moaning, bored, whingy sound drives me up the wall", it was submitted this was not evidence of someone who was likely to lose her temper and kill her child. It was suggested this was another example of Ms Folbigg's self-blame and guilt over "minor matters" and her "compounding emotional disturbance over time".[676]

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667 Exhibit AZ, Diaries tender bundle, p 230.
668 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [248].
669 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [247]-[250].
670 Exhibit AZ, Diaries tender bundle, p 238.
671 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [253]-[255].
672 Exhibit AZ, Diaries tender bundle, p 242.
673 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [255]-[257].
674 Exhibit AZ, Diaries tender bundle, p 246.
675 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [262]-[264].
676 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [268]-[273].

424. It was further submitted the entry was important because it demonstrated:

    a. while Ms Folbigg may get angry, she was capable of restraining that anger ("Got so bad I nearly purposely dropped her on the floor… I restrained enough to put her on the floor and walk away"), which was consistent with the Crown case;[677] and

    b. Ms Folbigg expressed her fear that Laura would leave her, importing her view that any departure was passive, said to tie in with the belief that some being was responsible for taking her children, for punishing her, which would validly explain the "with a bit of help" reference.[678]

425. In respect of the entry dated 6 March 1998 in which Ms Folbigg wrote "Laura not well, really got on my nerves today snapped & got really angry, but not nearly as bad as I used to get", the submissions set out the full text of the entry but made no other submissions.[679]

Further submissions

426. Under the heading "Further Submissions" it was noted that the Crown prosecutor submitted to the jury after reading the diary entries that Ms Folbigg had managed to restrain herself from killing Laura until March 1999.[680] It was submitted that what the Crown equally could have said was that the diaries did not reveal any evidence of guilt in respect of Laura nor any evidence of smothering in respect of any of the children.[681]

427. It was submitted that a number of very relevant words were missing from the diary entries, such as "kill or killed", "murder or murdered", "smother or smothered" and "asphyxiate or asphyxiated", such that the diaries did not contain any confessions.[682]

Counsel assisting's submissions

428. Ms Folbigg's submissions dealt directly with the matters relied on by counsel assisting to ground a submission that Ms Folbigg's account of the diaries entries as illustrating a benign interpretation is implausible and should be rejected.[683]

429. Firstly, the submissions on behalf of Ms Folbigg submitted that counsel assisting had relied on "selected evidence at trial to tease out extremely limited context",[684] and also had ignored the context of the entries within the diaries and the context of events.[685] It was suggested that if the evidence of Mr Folbigg or Ms Bown was considered relevant to the interpretation of the diary entries, Ms Folbigg should have been given the opportunity to respond as a matter of procedural fairness and she was not asked about their evidence.[686]

430. In respect of Mr Folbigg's evidence at trial of his observations of Ms Folbigg expressing her frustrations with Sarah and Laura through her words, tone and physical actions shortly prior to each of their deaths, it was submitted that even "assuming the evidence is accurate and truthful", none of that evidence pointed to the guilt of Ms Folbigg for their murders, or the deaths of Caleb and Patrick.[687]

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677 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [270(a)].
678 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [270(c)].
679 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [273]-[274].
680 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [275].
681 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [276].
682 Submissions of Ms KFolbigg's representatives to the Inquiry (7 June 2019) Part D, [277]-[278].
683 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [279]; Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 9, [107].
684 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [279].
685 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [280].
686 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [281].
687 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [275(a)], [282], [287].

431. It was noted, without particularity, that Ms Folbigg did not accept all of Mr Folbigg's evidence on these issues, and suggested that his evidence did nothing to assist with the interpretation of the diaries.[688] It was further submitted that if the approach of taking into account Mr Folbigg's evidence is accepted as being legitimate, then additional evidence should have been brought before the Inquiry detailing Mr Folbigg's own behaviour at relevant times and why at least some of his evidence could be regarded as tainted. Again, there was no particularity provided to this suggestion.[689]

432. Similarly in respect of Ms Bown's evidence observing Ms Folbigg's conduct towards Laura, it was submitted that evidence did not assist in understanding the words Ms Folbigg wrote in her diary and recourse to the evidence demonstrated that a presumption of guilt approach had been adopted.[690]

433. Ms Folbigg's submissions took issue with counsel assisting's submission that there was an absence of evidence of Ms Folbigg having "expressed her beliefs to anyone during the course of the children's lives and deaths".[691] It was suggested that this was never put to her during her evidence.[692] It was further submitted that had it been put to her, it could have been answered by reference to the letter from Mr Folbigg to the nurse Margaret Tanner regarding Ms Folbigg's belief in fate, her diary entry of 28 June 1997, her visit to the clairvoyant and other entries.[693] It was also said she could have given evidence of the loss of her personal papers that may have demonstrated that belief.[694] Ms Folbigg's reference to the same belief system to police during her interview with police was also referred to.[695]

434. In respect of counsel assisting's characterisation of Ms Folbigg's explanations in her evidence in the Inquiry as fanciful,[696] it was submitted that the rationality of her belief system was not to the point.[697] It was submitted it was never put to her that she did not hold that belief system and as such it is an "accepted fact she has it, whether it is irrational or not".[698] That being that case, it was submitted, it is probable that her belief system would pervade her personal writings.[699]

435. In respect of counsel assisting's reliance on an apparent concession under cross-examination that Ms Folbigg was awake with Sarah before her death while Mr Folbigg was asleep[700] (by reference to the diary entry "I really needed him to wake up that night"),[701] it was submitted there was no entry in the diaries giving rise to such a concession.[702]

436. It was further submitted that the first recorded account of the events that night came from Detective Senior Constable Ward who "recorded that Sarah was in bed with her parents at the time of her death".[703] The Detective's statement was dated 14 December 1999 and in fact recorded that part of the information Constable Saunders provided him with when he arrived at the Folbigg home at about 3:30am on the morning of Sarah's death included:

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688 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [288].
689 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [288].
690 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [289]-[291].
691 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [279(f)].
692 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [300].
693 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [301].
694 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [301].
695 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [304].
696 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [279(g)].
697 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [307].
698 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [307].
699 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [308].
700 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [279(g)].
701 Exhibit AZ, Diaries tender bundle, p 162.
702 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [309].
703 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [310].

> *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 bed with them.*[704]

437. It was submitted that Ms Folbigg may have been up with Sarah on the night before her death because Sarah was unsettled and that because she slept well whilst in bed with the parents, Ms Folbigg put her into bed with them such there "may be no inconsistency at all".[705] It was suggested Ms Folbigg's account in the interview with police in July 2003 on this issue suffered from a risk of pollution by reason of the elapsed time, and that a listening device recorded conversation between her and Mr Folbigg which may have been influential on her recollection at the time of the interview with police was excluded in the Inquiry.[706]

438. No reference was made to Ms Folbigg's evidence in the Inquiry which counsel assisting referred to as amounting to a concession that she was awake with Sarah before her death while Mr Folbigg was asleep in the early morning, not earlier in the evening.

Disposal of the diaries

439. Ms Folbigg's submissions on this issue did not engage with the discrepancy between her description to police about having disposed of multiple diaries, and her evidence in the Inquiry about only having disposed of one.[707]

440. It was suggested in the submissions that it was never put to her that she was aware the diaries she disposed of contained incriminating material and that she disposed of them in order to cover her tracks.[708] The submissions on this topic pointed to the diaries not having been hidden from Mr Folbigg or anyone else, evidenced by Mr Folbigg having found a couple of diaries and read them.[709]

441. It was submitted that Ms Folbigg's account of having disposed of "her diaries" on Mother's Day was "not surprising" given her feelings of depression, self-doubt and guilt for failure as a mother, and the heightened sense of these feelings on Mother's Day.[710]

442. It was submitted that if she had been disposing of her diaries so as to "cover her tracks" then she would have been diligent on that day and in the weeks and months subsequently, to root out every diary and destroy it.[711]

443. In respect of the 1997 – 1998 diary located by police inside a container, inside a bag underneath clothing inside the bedroom wardrobe, it was submitted that the manner of its location was "consistent with a casual disregard" for it. It was suggested that if she thought it was incriminating and sought to cover her tracks, then she would have destroyed it rather than place it inside a bag.[712]

444. It was submitted on behalf of Ms Folbigg that any suggestion she deliberately hid diaries that contained admissions was nothing more than speculation.[713]

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704 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part C – Sarah, [38]-[40]; Exhibit BQ, Statement of Detective Senior Constable Glen Ward (14 December 1999) p 2.
705 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [310]-[311].
706 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [309].
707 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [313]-[321]; Transcript of the Inquiry, 29 April 2019 T621.45.
708 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [318].
709 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [319]-[320].
710 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [313]-[316].
711 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [317].
712 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [319].
713 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [321].

445. It was submitted that Ms Folbigg's incorrect response to police during her interview that she did not keep a diary also did not give rise to a credit issue. Firstly because the term "diary" could include an organised document in the nature of a calendar, or it could relate to a journal and it was unclear to which the police officer was referring. Secondly because there was "some confusion" over times which made the evidence uncertain.[714]

Findings regarding procedural fairness

446. There is no doubt that a duty arises in this Inquiry to afford procedural fairness to Ms Folbigg so as to avoid practical injustice to her.

447. However a submission that Ms Folbigg was denied procedural fairness by reason of my ruling that she would not be permitted to give "context" evidence is without merit. The effect of my ruling at that stage was simply to restate my earlier ruling as to the scope of evidence Ms Folbigg would be permitted to give. That ruling was made in December 2018. It was not the subject of challenge, or application to expand it in any way, at any time.

448. The "context" evidence foreshadowed in the application on behalf of Ms Folbigg before the commencement of her evidence comprised how the diaries were generated, the nature of her relationship with her adoptive family and her husband, and "some of the issues that were going on at the time" of the diary entries.

449. In her submissions Ms Folbigg acknowledged that "some (but not all) of the context in which the diaries were written" was in material before the Inquiry. She went on to refer at length to the context in which the entries had been written, her belief system, and her emotional or likely emotional state, as summarised above. The submissions were silent as to what other context-related material or evidence Ms Folbigg said should and could have been before the Inquiry.

450. It is plain that the Inquiry received extensive evidence about each area of context raised on her behalf before her evidence and in the written submissions.

451. First, Ms Folbigg gave extensive evidence of context in response to examination by counsel assisting the Inquiry, and cross-examination by counsel for the DPP and counsel for Mr Folbigg. She also gave evidence of context in response to questions by her counsel. Her own evidence covered her belief system, her emotional state at the time of writing the diary entries, and her relationships with her biological and adoptive families, and her husband.

452. Secondly, the Inquiry received into evidence context evidence from the trial in the form of non-medical evidence such as Mr Folbigg's testimony about Ms Folbigg's behaviour and attitudes towards the children and her response to their deaths, and letters between Ms Folbigg and Mr Folbigg which she was asked questions about by her counsel in the Inquiry. The transcript of trial evidence before the jury was tendered in the Inquiry on 12 January 2018. Parts of the trial transcript, and trial exhibits, were relied upon as relevant context evidence in the submissions on behalf of Ms Folbigg.

453. Thirdly, the Inquiry also received into evidence context evidence in the form of the psychiatric assessment report prepared by Dr Diamond at the request of Ms Folbigg's representatives in 2019, as well as reports prepared by other psychiatrists at the time of Ms Folbigg's sentence in 2003.

[714] Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [322].

454. These reports dealt at length with Ms Folbigg's upbringing, including her relationships with her family, her education, her belief system, her emotional state over the period of her life covered by the diaries, her relationships with Mr Folbigg and with the children, and the context in which her diary entries were written, for example as a way of keeping her feelings contained without the expectation they would be read, and as a way of coping with her distress which she had been unable to talk about. Aspects of the psychiatric assessment reports tendered in the Inquiry were also relied upon as relevant context evidence in Ms Folbigg's submissions.

455. The suggestion that evidence of context was excluded as a result of the ruling is wrong. Ms Folbigg was provided with reasonable notice of the evidence to be received in the Inquiry and a reasonable opportunity to present her case, including context evidence. She did so both orally in her evidence, and in writing in her submissions.

456. Similarly the submission that certain findings cannot be made because certain matters and evidence were not put to Ms Folbigg during the course of her cross-examination in the Inquiry is without foundation.

457. First, the rigorous cross-examination of Ms Folbigg to the effect that in her diary entries she was expressing guilt for her acts in killing the children adequately put to her that she should not be believed on any aspect of the benign interpretation of the entries which she contended for.

458. Secondly, the rigorous cross-examination of Ms Folbigg to the effect that she was concerned about the police reading her diaries and was being untruthful in her answers that she did not recall the 1997 – 1998 diary being in the wardrobe where it was found by police, adequately put to her that she should not be believed on her account about her reasons for disposing of her diaries and telling police that she did not have any more diaries.

459. The submission that if Mr Folbigg's evidence was to be accepted as legitimate then additional evidence should have been brought before the Inquiry detailing his own behaviour at relevant times and why at least some of his evidence could be regarded as tainted is unparticularised and baseless. There was no application by Ms Folbigg's representatives, or indeed anyone else, to have Mr Folbigg called as a witness in the Inquiry. In any event, this was beyond the scope of the Inquiry and there was no application to extend the scope in this way.

Findings regarding the non-medical evidence in the Inquiry

Interpretation of the diary entries

460. It is true that the diary entries contained no express admissions by Ms Folbigg to having killed her children. However, it is entirely proper in fact finding in circumstantial evidence cases for inferences to be drawn, and for individual pieces of evidence to be considered in combination, like strands in a cable.

Expressions of guilt and responsibility

461. I accept Ms Folbigg's submission, and Dr Diamond's opinion, that Ms Folbigg's diary entries have to be read on the basis of her "deep-seated psychological (but very private) subjective experiences" in light of her early childhood trauma.[715]

462. Even without the assistance of that advice from Dr Diamond, it is obvious that the diary entries must be read against a background of the fact also that Ms Folbigg's four children died. Whatever the causes of the deaths and the serious injury, they did happen, and it must be expected she was emotionally affected by those events, even if she caused the events herself.

463. The entries must be read with an understanding, to the extent such an understanding is permitted by contents of the diary entries, that the entries could merely reflect self-blame for deaths which occurred from natural unexplained causes.

464. Ms Folbigg did express such sentiment to police during the course of her interview in 2003 when she said "did I try enough, did I do enough, was I where I was supposed to be, was I not trying hard enough".[716] She repeated the sentiment during her evidence in the Inquiry when she said "the fact that I did not do something in anyway whatsoever that could've helped prevent that in some way then yes I will always say I'm responsible".[717]

465. The difficulty with this interpretation – an expression of guilt or responsibility based on omission for acts not done rather than the commission of acts done, as contended by Ms Folbigg – is that such an interpretation cannot be applied sensibly to many of the most inculpatory entries in Ms Folbigg's diaries.

466. In many places, Ms Folbigg expressed feelings of guilt or responsibility by references to acts by her, with such matters being within her control and thus the subject of concerted efforts by her to avoid their repetition. For example, Ms Folbigg wrote:

> 4 December 1996: *I'm ready this time. But have already decided if I get any feelings of jealousy or anger to [sic] much I will leave Craig & baby, rather than answer being as before… That will be when I will ask help & sleep whenever I can. To keep myself in a decent mood. I know now that battling wills and sleep depravaision [sic] were the causes last time.*[718]

715 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 41.
716 Exhibit E, ERISP of Kathleen Folbigg Q719.
717 Transcript of the Inquiry, 30 April 2019 T724.24-26.
718 Exhibit AZ, Diaries tender bundle, p 111.

> 18 June 1996: *I'm ready this time and I'll have help & support this time. When I think I'm going to loose [sic] control like last times, I'll just hand baby over to someone else.*[719]
>
> 1 January 1997: *But I feel confident about it all going well this time. I am going to call for help this time & not attempt to do everything myself anymore. I know that that was the main reason for all my stress before & stress made me do terrible things.*[720]
>
> 25 October 1997: *I think I am more patient with Laura. I take the time to figure what is rong [sic] now instead of just snapping my cog.*[721]
>
> 3 November 1997: *Lost it with her earlier. Left her crying in our bedroom & had to walk out - that feeling was happening. And I think it was because I had to clear my head & priotise [sic]. As I've done in here now. I love her I really do I don't want anything to happen.*[722]
>
> 28 January 1998: *I knew I was short tempered & cruel sometimes to her & she left. With a bit of help.*[723]

467. Additionally, while Ms Folbigg sought to suggest to police in 2003 and in the Inquiry in 2019 that she felt responsible for her children's deaths by reasons of *not* doing things, she never expressed that thought or suggestion in the available diary entries when referring to Caleb's, Patrick's and Sarah's deaths.

468. Ms Folbigg's evidence about the entries which identified her negative emotions and feelings around the stress of the children, was that at the time of writing those entries she believed these emotions and feelings impacted on the children, though not because her moods led her to bring about their deaths. This interpretation not only sits uncomfortably with a plain reading of the entries themselves, but also with the notion of expressions of guilt based on a failure to do things.

469. Similarly, while it is clear Ms Folbigg did, at the time of writing the diary entries, believe in the involvement in her life of a higher or supernatural power or being who meted out rewards and punishments, and in the existence of souls, spirits and the afterlife, this "belief system" did not fit with or explain the content of the most inculpatory entries which implied Ms Folbigg's own actions arising from her moods were involved in the deaths of the children.

470. Indeed Ms Folbigg expressed in the diary entries a high degree of certainty about the role of her own moods in the deaths of the children "the last times",[724] and a corresponding certainty about what she herself needed to and would do upon Laura's arrival in order to avoid risk of her death. The entries clearly convey Ms Folbigg's belief that it was within her control, by modifying her behaviour to both be more relaxed in her approach to parenting and to ask for assistance when she needed it, to avoid the death of another child.

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719 Exhibit AZ, Diaries tender bundle, p 70.
720 Exhibit AZ, Diaries tender bundle, p 114.
721 Exhibit AZ, Diaries tender bundle, p 226.
722 Exhibit AZ, Diaries tender bundle, p 230.
723 Exhibit AZ, Diaries tender bundle, p 258.
724 Exhibit AZ, Diaries tender bundle, p 70.

471. The psychiatric assessment reports identify that Ms Folbigg was exposed to traumatic experiences in her early life, experienced significant depression over the course of her children's lives and since their deaths, and in the opinion of Dr Diamond, was and remains diagnosed with Complex PTSD. Those observations were only supportive of Ms Folbigg's account of the interpretation of the diary entries to the extent the entries were sensibly able to be read as expressions of guilt or responsibility based on omissions.

472. There was no observation or opinion in those reports, such as identification of disordered thinking, which supported or corroborated Ms Folbigg's interpretation of the words in her diaries which referred to actions on her part, as having meaning other than their ordinary English meaning and as involving separate and unrelated thoughts in immediate proximity to each other.

473. In considering the diary entries it has to be remembered also that Ms Folbigg is a reasonably intelligent woman. The only evidence of IQ testing of her before the Inquiry was done when she was seven and she had an IQ of 110 which put her in the "above average to superior range".[725] Dr Diamond said "her cognitive functioning suggested at least average intellect."[726]

474. The other evidence was that she went through school to Year 12 and sat for the trial Higher School Certificate. She did not stay on at school to sit for the Higher School Certificate because an argument developed with her foster mother about her relationship with a young man and she left home and did not continue on at school. At school she regarded her best subjects as English and History.[727]

Plain meaning

475. Even making every allowance for her deep-seated psychological subjective experiences and childhood trauma, and any emotional state she may have been in at the time of writing the various entries, it is impossible to give the diary entries any meaning other than their ordinary English meaning.

476. Ms Folbigg's account that terms such as becoming frustrated, angry, losing her temper, losing control and snapping her cog all meant the same thing, and referred to a slight frustration but not to any physical conduct by her towards the children, was also inconsistent with other evidence.

477. Most significantly this was inconsistent with Mr Folbigg's evidence at the trial of Ms Folbigg's severe frustration and anger towards him and Sarah and Laura, on occasions very proximate to their deaths, when the children would not do as she wished them to. His evidence was that this frustration and anger manifested both in Ms Folbigg yelling or screaming at him and at the children, and in her using excessive physical force to throw Sarah at him, and to restrain Laura in her highchair.

478. Mr Folbigg's description of Ms Folbigg's behaviour in this regard was entirely consistent with the sentiments and feelings of severe frustration, losing it and snapping her cog as Ms Folbigg wrote in her diaries, if one ascribes a plain language meaning to her words.

479. Ms Folbigg's attempts at explanation simply come from her best attempts to twist the ordinary English meaning of her own words, to arrive at some innocent explanation.

480. So much was apparent from Ms Folbigg's explanations given to police during her interview in July 1999, which were before the jury at the trial, and the explanations she gave in the Inquiry in 2019.

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[725] Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 17.
[726] Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 24.
[727] Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 13.

481. By way of example, in her interview with police Ms Folbigg said that she got up to go to the toilet in the middle of the night and on the way back she checked on Sarah and found that she was not breathing.[728] She confirmed in her evidence in the Inquiry that she told police that when she checked on Sarah she was already dead and that she (Ms Folbigg) was not awake at the time she had died, and that this remains her position.[729]

482. However, Ms Folbigg made the following diary entry in respect of Sarah, prior to Laura's birth:

> *I really needed him to wake up that morning and take over from me. This time I have already decided that if I ever feel that way again I'm going to wake him up.*[730]

483. This entry is to be considered in light of the August 1993 calendar sheet on which Ms Folbigg recorded "Sarah left us 1am",[731] and Mr Folbigg's account of having awoken at 1.10am and observed that Ms Folbigg and Sarah were not in the room, before going back to sleep and subsequently being woken by Ms Folbigg screaming.[732]

484. During her interview with police Ms Folbigg had accepted this entry referred to Sarah. She said "The, the bit where I said I wanted him to take over from me, that's Sarah."[733]

485. In the Inquiry, under cross-examination, Ms Folbigg:

a. first denied she was referring in that entry to the morning of Sarah's death,

b. then accepted "it may have been" but she said repeatedly that she was not "a hundred per cent clear on that."[734]

c. when it was put to her that she was awake with Sarah that morning she said "[i]f I was caring for her then yes I most likely was".[735]

d. when it was suggested to her that that was in fact the case on the morning that Sarah died, Ms Folbigg responded "I can't say with any clarity on that."[736]

e. she then denied she was awake when Sarah died.[737]

f. she refused to agree that although she had told police she was not awake at the time Sarah had died, her diary entry demonstrated something different had occurred.[738]

486. Separetly she also said "take over" meant "[t]he care, yes, the general care of my child, yes" then said it meant both that and having someone there when the baby was found dead.[739]

487. In my view, Ms Folbigg's prevarication in her evidence was a clear attempt by her to avoid the only rational possible meaning of the words written in her diary and to avoid admitting she had been untruthful when questioned by the police.

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728 Exhibit E, ERISP of Kathleen Folbigg Q269, Q298 and Q304-317.
729 Transcript of the Inquiry, 29 April 2019 T658.16-20.
730 Exhibit AZ, Diaries tender bundle, p 162.
731 Exhibit AZ, Diaries tender bundle, p 62.
732 2 April 2003 T128.22-131.45.
733 Exhibit E, ERISP of Kathleen Folbigg Q761.
734 Transcript of the Inquiry, 29 April 2019 T657.5-43.
735 Transcript of the Inquiry, 29 April 2019 T657.50-658.1.
736 Transcript of the Inquiry, 29 April 2019 T658.3-4.
737 Transcript of the Inquiry, 29 April 2019 T658.6-11.
738 Transcript of the Inquiry, 29 April 2019 T658.13-24.
739 Transcript of the Inquiry, 30 April 2019 T732.17-31.

488. At trial the credibility of Mr Folbigg was called into question when he said that Ms Folbigg was awake and out of their bedroom with Sarah shortly before Sarah died.[740] That was because he had gone to the police to retract an earlier statement to that effect. He said he had done that at the encouragement of Ms Folbigg.[741]

489. The fresh evidence before the Inquiry from the listening device at Ms Folbigg's home and the prevarication in Ms Folbigg's answers in cross-examination on this subject provide significant support to the truthfulness of Mr Folbigg's testimony and that demonstrates again that she lied to the police when she said she simply found Sarah dead. It also demonstrates her interest in trying to avoid suspicion about what she had done by prevailing on Mr Fobigg to go to the police and change his evidence.

490. Similarly, the attempts by Ms Folbigg to explain away the diary entries as saying one sentence did not follow from another sentence and that they were just random thoughts, cannot be accepted.

Conclusion

491. I am satisfied that Ms Folbigg was untruthful to the police during her interview, and in the evidence she gave before the Inquiry, in a clearly deliberately designed attempt to obscure the fact that she had committed the offences.

492. I am satisfied the diary entries were written by a reasonably intelligent woman in plain language, carrying their plain meaning.

493. Accordingly, neither Ms Folbigg's evidence before the Inquiry, nor the psychiatric assessment reports tendered in the Inquiry, causes me to interpret Ms Folbigg's diary entries other than in accordance with the ordinary English meaning of the words which she wrote.

494. I am satisfied that the plain meaning interpretation of the diary entries carries the character contended by the Crown at the trial, of virtual admissions of guilt for the deaths of the Sarah, Patrick and Caleb, and admissions that she appreciated she was at risk of causing similarly the death of Laura.

Disposal of the diaries

495. When Ms Folbigg was questioned by police, she said she had thrown three diaries away on Mother's Day, 9 May 1999 and not written in one since. At the end of her record of interview, police informed her they had a warrant to search her premises and asked her whether she had any other diaries. She immediately responded that she had a new diary that she had just started.

496. As it transpired there was a new diary, one she had "bought yesterday" and in that diary was a "July 99" entry saying she hoped this diary did not like the others "come back to bite me like my 97 one has".[742] There was also a diary entry dated 19 June 1999.[743]

497. When asked about those matters when she gave evidence in the Inquiry, she said she had forgotten about the entry in June but she did not say and indeed could hardly say she had forgotten about the diary she then said she had bought the day before and written in.

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740 2 April 2003 T94.51-97.31, T128.25-129.4; 13 May 2003 T1331.24-36.

741 2 April 2003 T94.51-97.31.

742 Exhibit AZ, Diaries tender bundle, p 290.

743 Exhibit AZ, Diaries tender bundle, pp 281-289.

498. It is barely conceivable that Ms Folbigg overlooked an entry she had made in a diary in June. It is inconceivable that she forgot an entry made in a diary she had bought the day before the police interview.

499. It is quite clear that she lied to the police when she said she had thrown all her diaries out on Mother's Day and had not written in one since. She did not know at that stage that police had a search warrant ready to execute and as soon as she was told she immediately volunteerd information about the new diary. That demonstrates how quickly she was able to appreciate the possible implication of telling a lie and how quickly she tried to cover it up.

500. When asked about these matters in cross-examination, she said she did not believe there was anything in the diaries to cause her any problem and therefore she had no reason to be concerned about them. However, one of the entries in her new diary clearly indicated that before the police interview she did understand that the diaries could cause her a problem, namely that her diary could "come back to bite me". Again it appears she was being deliberately untruthful when giving evidence to the Inquiry.

501. That is also demonstrated by the conversations recorded on the listening device in the house. The conversation recorded at 9:40pm on the night of the police interview had Mr Folbigg say "I came home to get the diary and take it back to work and I couldn't find it." She replied "I couldn't find it and they found it… and I've gone, I didn't have it so".[744] That clearly relates to the diary found by the police at the bottom of the wardrobe. It also indicates an intention to get rid of the diary because of an awareness it could cause her problems.

502. The comment she is recorded making the next morning "I should have fucking done what I was gunna do, stuck it underneath that,"[745] lends more support to this interpretation of her actions.

503. The listening device in her lounge room the next day on 23 July 1999 at 22:12 recorded her saying the "stuff" she wrote was "comin' back to bite me in the arse."[746] That was after her interview with the police but it is only restating the sentiment she expressed in her diary before she had been questioned by the police.

504. The only reasonable conclusion is that she was well aware of the danger to herself of the diary entries and she was concerned to ensure the police did not see any more diary entries. That raises questions about her account that she threw diaries away on Mother's Day 9 June 1999, only 10 days before Mr Folbigg took diaries to the police. These were diaries she kept for years. When asked by police she denied throwing them out after Mr Folbigg went to the police.

505. Evidence available in the Inquiry from the listening devices, which was not available at the trial, demonstrates Ms Folbigg's interest in preventing the content of her diaries being seen.

506. I am satisfied that Ms Folbigg lied to the police about not writing in a diary after she threw out her diaries, and that she was also untruthful when she gave evidence that she did not have any concerns about her diary entries.

744 Exhibit AZ, Diaries tender bundle, p 552.
745 Exhibit AZ, Diaries tender bundle, p 559.
746 Exhibit AZ, Diaries tender bundle, p 554.

Conclusion

507. Having regard to the non-medical evidence, including the diaries, I am satisified that:

    a. Ms Folbigg was untruthful in several respects to the police in her interview and in the evidence she gave before the Inquiry, including about her possession and dispossession of her diaries and whether she appreciated the significance of their contents;

    b. the diary entries were written by a reasonably intelligent woman in plain language and that I should interpret them in accordance with the ordinary English meaning of the words she wrote; and

    c. the plain meaning of the diay entries is one of virtrual admissions of guilt for the deaths of Caleb, Patrick and Sarah, and admissions that she appreciated she was at risk of causing similarly the death of Laura.

508. Accordingly, on the basis of the non-medical evidence, including the diaries I have no reasonable doubt about Ms Folbigg's guilt for the offences for which she was convicted.

Report of the Inquiry into the convictions of Kathleen Megan Folbigg

July 2019

Schedule

Schedule

Diary Entry	Reference
1989 diary	
1 February 1989	
8 00[am] BABY DUE. 8 15[am] Lea's still here. 9 00[am] Pull out hair if not here. 9 30[am] Took Kath to hospital. BABY CALEB BORN. 11.15pm. 7Lb 3¾oz. 19 ½ inches long. CALEB GIBSON FOLBIGG 1/2/89.[1]	Transcript of the Inquiry, 29 April 2019 T675.30 676.11
5 February 1989	
700 – 800 – 10 00 – 50ml 11 00 – 12 00 – 5ml water ENF 60ml Restless – 1am – change nappy 2am – 3am – 4am – ENF – 60ml 5am – 6am – 7am – ENF – 55ml 11 30 fed Caleb – enf 60ml. slight spew. 2 00 fed Caleb enf – 40ml 3 00 Brought Caleb home!!!!! ********* 6.00pm – fed Caleb – enf 60ml. no spew.[2]	Transcript of the Inquiry, 1 May 2019 T777.5-34
14 February 1989	
*8** – gave [un-transcribable] 8.30 – Still restless (seemed to help) 9.00 – always crying – put back down 9.30 – still asleep luckily [sic] 10 10.30 11 11.30 12	Transcript of the Inquiry, 29 April 2019 T676.13-27

12.30AM wind pretty good
1 – 75-80mLs? (doing NO^2)
1.30
2
2.30
3
3.0
4 – starting to stir
Big Sleep
4.30 – wind pretty good 80mLs
5 – 90mL (wind pretty good)
5.30 – Asleep?
6 – wind okay. (slight vomit) 20mL
6.30
7 – awake doing NO^2
7.30 – slight vomit
– Still awake. 85mLs
8.00pm – including top up 20mLs

Has been pretty good all day! [3]

15 February 1989

Dad looking after Caleb.
Have everything ready.
830[am] Appointment Hospital (EEG)[4]

Transcript of the Inquiry, 1 May 2019 T778.3-32

1 March 1989

4 weeks (1 month)[5]

Transcript of the Inquiry, 1 May 2019 SC T778.34-36

24 May 1989

16 weeks (4 months)
Make appointment Dr Leeder
Booster for Caleb.[6]

Transcript of the Inquiry, 1 May 2019T778.43-50

19 July 1989

24 weeks (6 months)
Booster for Caleb[7]

Transcript of the Inquiry, 1 May 2019 T779.1-780.36

1990 diary

10 July 1990

Well another day. Looks like Pat might be starting to sleep all night – hope so. He just plays up a little during the night though.
I think a full large feed during the night is in order. He seems to be more hungry then. He is getting a bad habit of going to bed with Craig. I'll have to really try to stop that. He's getting to an age where he realises that its [sic] lovely to do. Well, better get some work done.[8]

Transcript of the Inquiry, 29 April 2019 T680.30 681.26

11 July 1990

Transcript of the Inquiry, 29 April 2019 T681.28-31

Well, what a dismal day, raining, windy and cold. At least we will have the gas on tonight, our house should be nice and warm. Patrick is asleep. He always settles after his morning feed and bath. Think I'll give up trying to get him to settle down of a nighttime [sic] it obviously doesn't work. The life of a mother.

Been really lucky lately he has been missing the early morning feeds. Its [sic] been great. 5-6 hrs sleep.[9]

12 July 1990

Transcript of the Inquiry, 1 May 2019 T780.40-46

Well, Craig has been sacked today. But doesn't seem to be any skin off his off our nose. He's lucky he has got another job already!

I've been seriously thinking a lot going back to work. We could do with the extra money. But only draw back is that I'de [sic] have to try to organise someone to look after him during the day. If I get a nightime [sic] job, I'de [sic] have to be picky about the hours. Only good thing is that Craig would be here to look after Patrick. I'll look in Sat morn paper to see what sort of jobs are around at the moment. Well – Pats been asleep for about an ½ hour don't know wether [sic] it will last.[10]

23 July 1990
[note written on 23 July 1990 date but signed 21st Sat 1990]

Transcript of the Inquiry, 1 May 2019 T780.39 781.24

The idea of being independent and free from medling [sic] family members would be wonderful. My mother couldn't complain at least I'm not miles & miles away from her. Ide [sic] come down home, occasionally but not over night [sic]. Bringing Patrick up in that sort of a life is a little concerning but the kid wouldn't want for anything that's for sure. And to make the move now wouldn't affect him at all. He'd grow up to be a Hawkes nest boy instead of a Mayfield body. He'd have ideal conditions during summer and well. We'de [sic] manage. Well better sign off, considering I've taken over 3 days to write just this (21st Sat 1990) Kathleen.[11]

August 1993 calendar

30 August 1993 – Sarah died (10 months, 16 days)

Transcript of the Inquiry, 30 April 2019 T733.45-50 15 May 2003 T1483-1484

Sarah left us. 1am[12]

1996/97 diary (4 June 1996 – 5 June 1997)

4 June 1996

Transcript of the Inquiry, 30 April 2019 T701.7-16

Well, here I am, Dubbo. Bought this today. My last one got destroyed.[13]

7 June 1996 – Friday

I need some time of myself. I don't really get to have any of that. Craig has to be with me all the time….Well, that's all for now. I know Craig's pood because of my mood. But fairs [sic] fair he's never had to suffer one ever with me so PMT away we go. (HA)[14]

Transcript of the Inquiry, 29 April 2019 T681.35 682.20

7 June 1996 – 12.45pm

Mel was pissed as a nit when I went over earlier. Poor sod. She has to have the maj [sic] attention or she feels like a failure. But you'd of [sic] thought that she would of [sic] thought of Steve first in this instance, but it was probably her way of handling a crisis. We're all different that way. Me I become a nasty person. I felt good tonight women seemed to envy me. Men seemed to be perving on me even with my overweight behind.[15]

Transcript of the Inquiry, 29 April 2019 T682.26-45, T686.9-35

24 June 1996 – Sunday 9.20pm

… Haven't lost that maternal instinct. Emma seems at peace with my presence. Maybe I shouldn't be as worried as I was feeling. I had a thought that my own baby wouldn't bond with me. Craig will have to do all the work??? Still. Craig's reaction was a typical hand it to the woman – she knows what to do, truly hope that changes with [un-transcribable]. I will need all the support I can get if possible.[16]

Trial Defence sentencing exhibit 3, article 3

16 July 1996 – Tues nite 10.30pm

… Sometimes I feel life is a film scene, just practiced & rehearsed, each actor, perfect & surreal, times I don't fit in the play, have never fit, but keep attempting to anyway for fear of being isolated & alone. Times – I feel alone no matter who I'm with.[17]

Trial Defence sentencing exhibit 3, article 4

21 July 1996 – Sunday 10.43pm

Moved furniture & put cot back up today. Mixed emotions, sadness, nervousness, exciting. Looked at books I've got – never opened. I do hope & pray that the next child we have will get to have them read & read them also.

…

Depressed a little now…[18]

Trial Defence sentencing exhibit 3, article 5

25 July 1996 – Thursday 9.30am

Did miss him in bed though. Just the comfort that someone else was going to be there. Like, I know that it would be me who would hear a break-in first, not him, but at least if I screamed loud enough he'd hear me.[19]

Submissions of Ms Folbigg to the Inquiry (7 June 2019), Part D, [149]
1 April 2003 T49.53-50.5

25 July 1996 – Thursday 9.30am

Having bad thoughts about him [Craig] leaving me in some way though. Strange, he's either died or left me for someone else.

…

Thought of a baby and being left alone is a little frightening. Hope it never happens.[20]

Trial Defence sentencing exhibit 3, article 6

6 August 1996 – Tuesday nite 9.30

Is it a sign don't bother with having a child. Would be just desserts for me if it is – exactly what I deserve for my under-reacting of life. We'll see.

…

My ego's a little busted with my problems I seem to be having. With summer coming & starting to doubt wether [sic] ruining my body with a kid is a good idea. Worry that I'll be undesirable by any mans [sic] standard after I've had it. But that's life, and you adjust accordingly.[21]

Transcript of the Inquiry, 29 April 2019 T692.21 693.4

9 August 1996 – Friday morn 3am

Been feeling weird lately – Depressed, indisive [sic] etc not my usual self. Can't seem to put a finger on whats rong. [sic]

…

Feeling lonely!! I know that's silly because I have friends I can see, but I suppose it's because I want friends that will come to see me & want to be with me, I usually feel that I'm intruding or pushing my way onto people. Okay enough self-analysing. It's my ego & weight-problem that's giving me a bashing. Rang to go back to J/C, they haven't bothered to return my call. Feeling left out, taken for granted, unattractive and self-centred. There, I've purged myself. Now to change this, its [sic] up to me – as usual.[22]

Trial Defence sentencing exhibit 3, article 8

26 August 1996 – Monday nite 9.30pm

Didn't end up going to work today. Was deeply depressed & thoughtful.[23]

Trial Defence sentencing exhibit 3, article 9

8 September 1996 – Sunday arvo

Feel now is the time for us to have another baby. Have finally realised it's the right time for me. I have Craig & he wants a child. That I can give him. And I have enough friends now, not to loose [sic] it like before.[24]

Transcript of the Inquiry, 30 April 2019 T701.18 703.22

Submissions of the ODPP to the Inquiry (24 May 2019) p 27

Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D [162]-[164].

Exhibit E, ERISP of Kathleen Folbigg Q629-636

Trial Defence sentencing exhibit 3, article 10

15 September 1996 – Sunday nite

Friday and Sat night were great fun. As usual Craig and I argued about my love of dancing & being around people. I think I've figured out what to do. Only go out once a fortnight. That way he'll see that I'm making an effort. And always ask if he would like to go as well.[25]

Transcript of the Inquiry, 30 April 2019 T707.36 708.30

3 October 1996 – Thursday nite 9.50pm

Enough is enough.
If we have trouble it will be on his head not mine. I've compromised & given up enough for him.
No more.[26]

Transcript of the Inquiry, 30 April 2019 T708.31-48

30 October 1996 – Wednesday 5am

So many things troubling me lately. Not sure where to start. Craig and I are fine as in our relationship, becoming pregnant or rather not, in my case is starting to weigh me down. Think I must be suffering a stress reaction. I know as each month goes by depressions are getting worse.

…

Work is truly depressing me most days.

…

I think that the business with my mother is finally wearing me down. I just can't understand a hate so strong.

…

Things I remember are not good about upbringing, but one fact remains I have a safe home, food & clothing. I. a person who had a choice of that or stay at an orphanage is all her life can't expect much more.[27]

Trial Defence sentencing exhibit 3, article 13

13 November 1996 – Wednesday 4.30am

Not sure why I'm so depressed lately. Seem to be suffering mood swings. I also have no energy lately either.

…

Why is family so important to me? I now have the start of my very own, but it doesn't seem good enough. I know Craig doesn't understand. He has the knowledge of stability and love from siblings & parents even if he chooses to ignore them. Me – I have no one but him. It seems to affect me so. Why should it matter. It shouldn't.[28]

Trial Defence sentencing exhibit 3, article 14

14 January 1997 – Tues morn 3am

Not happy with myself lately. Finally starting to physically show that I'm pregnant. Doesn't do much for the self-esteem. Don't get me wrong, I couldn't be happier its [sic] just Craig's roving eye will always be a concern to me. I suppose that is a concept known by all women. We are vulnerable, emotionally at this stage. So everything is exaggerated 10 fold.

Trial Defence sentencing exhibit 3, article 17.

…

I think it's stress related. I must learn to calm down & be rational & worry about things as they happen, not if they do.[29]

4 February 1997 – Tues morn 2:30am

Transcript of the Inquiry, 30 April 2019 T723.18 724.2

Yes AM, I know, should be in bed sleeping. Think maybe I'm in practice for my future wake up calls for jonier [sic – junior]. That's okay. I've learnt finally that I can catch up sleep later on each day.

…

Could it be, because I personally feel, that no one was for me. My parents never came to school functions, sports carnivals or anything like that, even my tennis matches when I won my trophy. She had more important things to do all the time, things for herself.[30]

24 February 1997 – Monday 2.00pm

Trial Defence sentencing exhibit 3, article 22

Very emotional now, upset feeling useless, not myself, no confidence at all with any decision.

…

What do I do, I want to keep earning money for Craig but they've decided its [sic] not with them. I've let everyone down.

…

Too upset to keep writing, crying all the time.[31]

13 March 1997 – Tuesday 10.30pm

Trial Defence sentencing exhibit 3, article 23

Told Craig about my concerns of being alone in Sydney. But he wasn't impressed. It's something I will just have to get over & deal with myself. Today I got the impression he just didn't want to be or have me around.[32]

5 April 1997 – Saturday morn 1am

Trial Defence sentencing exhibit 3, article 14

Don't hear from any of my family now. Sometimes I feel as abandoned again with no real family roots.

…

I don't have that security & now now [sic] that I never really did. I'm a true loner. Without the roots & family I provide myself I'de [sic] be totally alone.[33]

1 May 1997 – Thursday nite 9.48pm

Transcript of the Inquiry, 30 April 2019 T730.23-37

Worst thing last time was the feeling that I was being deprived. I don't think that feeling will be to [sic] great this time, because I have everything I could ever want already. Baby will be icing on our cake.[34]

18 May 1997 – Sunday 6.00pm

Not feeling good about anything. Tired, achey [sic], exhausted, can't breathe properly, sick of everyone, everything, life in general[35]

Trial Defence sentencing exhibit 3, article 27

29 May 1997 – Thurs 9:30pm

Need new diary soon I've actually nearly filled up this one. Think it has helped, writing my thoughts & feelings down regularly fell [sic] as though its [sic] become a friend that I can off load on. And it doesn't back answer me – thats [sic] the best thing. Laugh & stupid things I've written in Past, but they were important to me back then as this is now.[36]

Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [216]-[217]

1 April 2003 T52.46-53.1

30 May 1997 – Fri 9.15am

Got myself into quite an emotional state last night.

…

Felt, feeling very alone, unattractive & now uncomfortable with all the many thoughts that are running through my mind and about the stability of our relationship. This is not the time to be upset & stressing over everything. He pulls away from me if I touch him in any other way than comforting. Feel as though I've lost him, that his feelings aren't the same anymore. Never felt so alone in all my life.[37]

Trial Defence sentencing exhibit 3, article 28

2 July 1997 – 3am Wednesday

Was very upset yesterday evening, crying & being totally emotional. Couldn't think of anything else to do but cry.

…

Was just so sad and still am, Scared is the word. I know that it won't be long now. 4 weeks? Sounds a fair amount of time but, he/she could decide to come earlier than that. If its [sic] got any sense, it will, my poor bod [sic]

isn't handling it well at all any more[38]

Trial Defence sentencing exhibit 3, article 19

1997/98 diary (6 June 1997-10 April 1998)

8 June 1997 – Sunday 10pm

Heard from Megan Foger today. Wasn't interested in talking to her. Blast from the past that has proven to mean pretty much nothing to us. She's fat, lazy, not much good at anything but thinking of herself, not a good mother either.[39]

Transcript of the Inquiry, 30 April 2019 T735.40 736.4

14 June 1997 – Sat 12.30am (my 30th birthday)

I have no family of my own to acknowledge me except Lea & more and more she's proving that I don't really really matter to her.

…

Depresses me that everyone else has a fair idea, where and what time they were born. I don't, have never been told.[40]

Trial Defence sentencing exhibit 3, article 31

2 July 1997 – 3am Wednesday

Transcript of the Inquiry, 30 April 2019 T746.19 747.7

Trial Defence sentencing exhibit 3, article 33

So there is no real excuse except my laziness. I have 4 weeks left. To provide healthy food, energetic food, for both baby & me. Would only benefit me surely & possibly help me to have a quiet, healthy baby. It will be my fault when I have a baby that required sweet sugary things to keep it quiet, because I have addicted it to it already.

…

I already know that he [Craig] won't take any time off. My not working has hit him hard all he sees is 15 grand less in his hand/bank a year now. He's already starting to worry about it. Like I stressed that he would. I'll have to accept, he won't be as much support to me as I thought he might. Change is a coming. A big one. Well [sic] just have to take day by day, hour by hour & cope. Hopefully everything will prove to be different this time. It has to be. I have to be![41]

18 July 1997

Trial Defence sentencing exhibit 3, article 34

Curious as to what's happened or who is responsible for her [Mel] having such a low opinion of herself. I think Steve, partly, he calls her stupid etc. Joking or not all comments like that hurt. Its [sic] what made me believe I was nothing or a nobody. Craig even was partly responsible for making me feel that. He doesn't do it as often anymore. I've learnt to pull him up on it.[42]

20 July 1997 – Sunday nite 9:30pm

Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [231]-[232]

1 April 2003 T53.49-54.4

I accept that my identity as a person starts with me. I've decided thats [sic] the way that it has to be. I have no past, no relatives to [un-transcribable] remind me & I am it. So therefore the choice of this baby was to "extend" me. Natural & one Ive [sic] made happily & whole heartedly. And would make again I'm sure. Problem was with the other 3 kids- felt I didn't deserve to be extended & that I was condemning them to life with me- that feeling has changed- so this time all is well & well it will go.[43]

12 August 1997 – Wednesday 6:30pm

Transcript of the Inquiry, 29 April 2019 T659.17-660.9

Trial Defence sentencing exhibit 3, article 35

Relieved she here, apprehension that she is. Worry & concern if my decision was right. But then I look at Craig & know I did the right thing. Am just letting myself cry if I want to this time. It helps – I'm not organised & strong like everyone expects me to be this time & I tell people. Craig is home with me, will be so different when the time comes for him to be gone all day. That will be my test, but I hope by then I'll be able to walk okay & get back to my exercise.[44]

17 October 1997 – Friday night 9pm

Transcript of the Inquiry, 30 April 2019 T749.14-33

Laura is growing up so fast now, 3 months old next week. Hard to believe. She is now really starting to come alive personality wise. I'm pretty lucky, she's a fairly good baby. At least she doesn't fight sleep as bad as Sarah did. Sometimes my days come across as a bit boring & tedious for me. I sometimes miss the freedom of being single. But on the other hand, this new life is great. I know that is what Craig has always wanted.[45]

10 November 1997 - Wednesday night 9.30pm
(Query – is this actually December?)

Craigs [sic] finally taking some of the heat for now. He puts her to sleep now. She doesn't seem to like me very much except to feed & play with lately.[46]

Transcript of the Inquiry, 30 April 2019 T762.10-15

28 November 1997

Could get back into the gym, but I have to take her with me & it's too hard & I don't enjoy the classes anymore because she's there and Craig doesn't like Mel or anyone else looking after Laura, except me, so gym's out - Of course that shouldn't be stopping me from walking and eating properly… but I just don't seem to have the heart anymore and I know that's all the cyclological [sic] and connected to feelings of neglect, rejection, loneliness which brings on a depression which I disguise by eating chocolate and junk food and feeling sorry for myself most of the time. I need to get back to the basics the reason for losing the weight.[47]

Trial Defence sentencing exhibit 3, article 41

Submissions of Ms Folbigg to the Inquiry, Part D, [252]-[253]

11 December 1997 – Thursday might 9.43pm

Me I still wake up from about 12.30 onwards, broken sleep is also making me very irratiable [sic] & snappy. I think Craig & I are having trouble. This stress of Laura is finally showing. In the one place we're trying to keep it from 'our marriage'. In myself Im [sic] sure I am over eating because of boredom, any time on my hands, my urge is to eat. Would be okay if I ate healthy stuff. Which I now start intending too [sic]. Depression seems to get me more now too. Must control it not it me.[48]

Transcript of the Inquiry, 30 April 2019 T762.17 762.42

Trial Defence sentencing exhibit 3, article 42

28 December 1997 – Sunday 10.30pm

Feeling depressed, unhappy with myself. Know why, need willpower and I'll succeed. Ward getting engaged.

…

There's something wrong with Craig and I. Still haven't figured it out yet. Laura keeping us together as a couple I think. Think if I hadn't had her, not sure we'de of [sic] survived as a couple.[49]

Trial Defence sentencing exhibit 3, article 43

31 December 1997 – New Year's Eve 11pm

Funny but if it wasn't for Laura, I'de [sic] feel as though I've wasted another year of my life. Everyone seems to be enjoying themselves. Pool getting a real workout.[50]

Trial Defence sentencing exhibit 3, article 44

12 January 1998 – Monday nite 10pm

Not doing well, need to get some will power! Eating rong [sic] - not exercising. Too inactive. But how do I over come inherent laziness. Would be happy to be a sloth. Tired 90% of the time too makes life a little tougher. Must try to stop lounging around all the time. Get machines should use them.[51]

Trial Defence sentencing exhibit 3, article 45

16 January 1997– Fri nite 9.05pm
(Error by KF should be 16 January 1998)

Been daydreaming again about life on my own. Wild, highly exaggerated as if I could or really want to. Always seem to when not really happy. Sorry to say. I don't get excited any more. Craig just doesn't do it for me anymore. Has to be because of this last pregnancy, plus I'm tired all the time. Want to do nothing but sleep. Its' [sic] not Craig; it's me. Plus we don't get to go out to dinner or dancing together any more. There isn't much – well, there's no romance between us anymore. It's all let's make money and raise Laura. We've forgotten ourselves in the process. Sad how that happens. One of the problems is I've lost me again. I'm just Mrs Craig Folbigg. Now I'm just Laura's mother as well. Where's Kath gone, a person in her own right who needs to have writing lessons, but probably better if I don't then no-one not even me, will be able to read this when I'm gone.[52]

Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part D, [265]-[266]

2 April 2003 T64.58-65.29

20 January 1997 (Error by KF should be 20 January 1998)

The gym was a pivotal part of me and now, because I can't go without taking Laura, it's put a damper on everything. I've had my one and only escape taken away from me.[53]

Submissions of Ms Folbigg to the Inquiry, Part D [266] [268]

2 April 2003 T65.31-37

7 February 1998 – Sat nite 10.25pm

Long days. Tiring & have been extremely short tempered. Cryed [sic] today. Told Craig lack of sleep & constant worry about Laura has got too me felt better after. Craig has tried to be helpful today. Doing chores that I have always wanted to do but never found time. What I wanted though was for him to just take her off my hands for a while. Or me go for a drive away. And be by myself. But she's not well, had her shots & feeling crappy. She's just a baby and doesn't understand. Hopefully she'll be back to normal soon.[54]

Trial Defence sentencing exhibit 3, article 47

13 March 1998 – Fri 10pm

Feeling very dissatisfied tonight – with myself, my life, Craig – what can I do.

…

I need him to take some of the stress of looking after her off me, he seems to be failing lately.[55]

Trial Defence sentencing exhibit 3, article 48

1 April 1998 – Wednesday 10:50pm

Thought to myself today difference with Sarah, Pat, Caleb to Laura, with Laura I am now ready to share my life, I definitely wasn't before. Why selfish maybe that's why my mother didn't have kids to [sic] successfully until she was older. Only thing left undone in my life is My Real Father? But closure will never come with that unless someone decides to be compastionate [sic] one day & tell me all about him.[56]

Transcript of the Inquiry, 30 April 2019 T772.27 773.33

1999

Jan 1st 1999 12.01pm Friday

Transcript of the Inquiry, 30 April 2019 T773.12-34

Hope I feel more satisfied with myself & life this year
Resolutions (1) More tolerance where Laura is concerned (2) More acceptance of my life and lack of youth (3) Try to keep friends Ive [sic] developed (4) More effort with my marriage (5) Get on with it.[57]

3 January 1999 – Sunday 9pm

Transcript of the Inquiry, 30 April 2019 T773.36 774.21

Well, another day. Was okay. Have had unsatisfied feelings about my marriage. Can't do anything though, not until Laura is much older. For her I'll stay. It's gotten to the stage that a word to describe how I feel about things would be "comfortable". Craig I can take or leave. I suppose it's how we are supposed to feel after 12 yrs? Is it. Isn't there more? Or is that just in the movies & all that. Maybe I'm just fantasising. Don't know anymore. Sometimes I feel I could just drain the bank accounts and leave. Leave Singleton, Craig & Laura behind. Start new, somewhere. But I'm not that brave a person. Could go to Queensland, but what would they say. Would they attempt to send me back or get Craig to come. Will never know. Haven't even had a happy new year from them. Although did get an xmas card. Wow. I'm always wanting to run away. Maybe I have more mum in me that I give her credit for. Scary thought. I suppose I'm happy enough. I think this is all that there is supposed to be now. Watching Laura grow up and hopefully turn into a decent human being. We'll see?

…

1/2 my problems are a bad self-image. Only I can change that.[58]

19 June 1999 – 4am

Transcript of the Inquiry, 29 April 2019 T625.41 627.1; 30 April 2019 T705.32 707.34, T725.30-727.44

Submissions of Ms Folbigg to the Inquiry, Part D, [14(c)]

Can't sleep. Tossing, turning. So much going through my mind. And none of it pleasant.
Another year has passed for me, each one is getting tougher. Trying to understand why the hell I'm even on this planet.
So many things point to the fact that I'm not meant to be.
Unwanted at birth. A father who was so selfish, unthoughtful that he took my mother from me & ruined my life from that one action.

…

I know I love him [Craig]. He has shown me what love is. I just have so much trouble justifying to myself that I deserve it all. I just want to hide or watch the world go by it would be so much easier. But I know that Craig's life, wellbeing, happiness, security and mental state seem to depend on me. Is that a good thing? I think so, its preventing me from just dying inside. Which once my Laura left is whats [sic] happened to a great extent anyway. I just want to cry all day and night. No one see's this but I think its [sic] all just getting a bit too much. I vowed Ide [sic] never write any of my feelings down on paper again. But it's the only way I know how to release.

…

It depresses & saddens me to realise that none of them will ever reach this stage in life. Only comfort I have is that where they are now they definitely have no stress or decisions to make and eternity can be spent carefree and loved always. At least that's what I have to believe or sanity I cling on to wouldn't last very long.

…

I think what has stirred all these emotions up is what I found out on Monday. Information is finally coming out & more & more I discover that they all, everyone of them are responsible for my predicament that I'm in now 30 years later.

…

I can't help but feel my life would have been so different & how it was meant to be if only Tom hadn't made a stupid mistake one night & "the family" hadn't interfered in the way that they did.
I believe that each person is here for a reason. Paths of life are chosen. So me having to adjust & alter mine so drastically has upset things. Because I can't believe that if there is a higher power that selects these paths for people. How could he choose this one for me.[59]

July 1999 – 10pm Thursday night

Decided to start writing in a diary again. Have missed being able to vent regularly. I just pray it doesn't come back to bite me like my 97 one has.
…
But this time I'm not going to use it as a means to avoid communicating with him [Craig]. I did before my journal became my friend and confident.
…
Also looking forward to becoming a jewel princess hopefully for the Mayoral Ball on the 7th. The day will comemorate [sic] a loss for us, but the thought of pampering ourselves and formally dressing up and parading, stroking our egos for the evening will help me cope. Can't talk for Craig. To remember something beautiful, well, it was beautiful, but you know what I mean. Will never forget her, as I haven't all the others. But, new memories will eventually help heal the pain and shape our lives to come. [60]

Transcript of the Inquiry, 29 April 2019 T627.3 628.35; 30 April 2019 T698.48-701.16

Submissions of the ODPP to the Inquiry (24 May 2019) p 20

1 Exhibit AZ, Diaries tender bundle, p 1.

2 Exhibit AZ, Diaries tender bundle, p 5.

3 Exhibit AZ, Diaries tender bundle, p 17.

4 Exhibit AZ, Diaries tender bundle, p 15.

5 Exhibit AZ, Diaries tender bundle, p 29.

6 Exhibit BN, Diary entries relating to Caleb Folbigg (24 May 1989, 18 July 1989).

7 Exhibit BN, Diary entries relating to Caleb Folbigg (24 May 1989, 18 July 1989).

8 Exhibit AZ, Diaries tender bundle, p 44.

9 Exhibit AZ, Diaries tender bundle, p 44.

10 Exhibit AZ, Diaries tender bundle, p 45.

11 Exhibit AZ, Diaries tender bundle, p 46.

12 Exhibit AZ, Diaries tender bundle, p 62.

13 Exhibit AZ, Diaries tender bundle, p 63.

14 Exhibit AZ, Diaries tender bundle, pp 63-64.

15 Exhibit AZ, Diaries tender bundle, pp 65-66.

16 Exhibit AZ, Diaries tender bundle, p 73.

17 Exhibit AZ, Diaries tender bundle, p 77.

18 Exhibit AZ, Diaries tender bundle, p 82.

19 Exhibit AZ, Diaries tender bundle, p 83.

20 Exhibit AZ, Diaries tender bundle, p 84.

21 Exhibit AZ, Diaries tender bundle, pp 85-86.

22 Exhibit AZ, Diaries tender bundle, pp 86-87.

23 Exhibit AZ, Diaries tender bundle, p 94.

24 Exhibit AZ, Diaries tender bundle, p 97.

25 Exhibit AZ, Diaries tender bundle, p 98.

26 Exhibit AZ, Diaries tender bundle, p 102.

27 Exhibit AZ, Diaries tender bundle, pp 104-107.

28 Exhibit AZ, Diaries tender bundle, p 108.

29 Exhibit AZ, Diaries tender bundle, pp 117-118.

30 Exhibit AZ, Diaries tender bundle, pp 123-124.

31 Exhibit AZ, Diaries tender bundle, pp 128-129.

32 Exhibit AZ, Diaries tender bundle, p 138.

33 Exhibit AZ, Diaries tender bundle, p 149.

34 Exhibit AZ, Diaries tender bundle, pp 157-158.

35 Exhibit AZ, Diaries tender bundle, p 164.

36 Exhibit AZ, Diaries tender bundle, p 169.

37 Exhibit AZ, Diaries tender bundle, p 170.

38 Exhibit AZ, Diaries tender bundle, p 197.

39 Exhibit AZ, Diaries tender bundle, p 184.

40 Exhibit AZ, Diaries tender bundle, p 190.

41 Exhibit AZ, Diaries tender bundle, pp 199-200.

42 Exhibit AZ, Diaries tender bundle, p 206.

43 Exhibit AZ, Diaries tender bundle, pp 208-209.

44 Exhibit AZ, Diaries tender bundle, p 219.

45 Exhibit AZ, Diaries tender bundle, pp 223-224.

46 Exhibit AZ, Diaries tender bundle, p 239.

47 Exhibit AZ, Diaries tender bundle, pp 235-236.

48 Exhibit AZ, Diaries tender bundle, p 240.

49 Exhibit AZ, Diaries tender bundle, p 245.

50 Exhibit AZ, Diaries tender bundle, p 246.

51 Exhibit AZ, Diaries tender bundle, p 252-253.

52 Exhibit AZ, Diaries tender bundle, p 254.

53 Exhibit AZ, Diaries tender bundle, p 255.

54 Exhibit AZ, Diaries tender bundle, pp 260-261.

55 Exhibit AZ, Diaries tender bundle, p 265.

56 Exhibit AZ, Diaries tender bundle, p 270.

57 Exhibit AZ, Diaries tender bundle, p 274.

58 Exhibit AZ, Diaries tender bundle, pp 275-276.

59 Exhibit AZ, Diaries tender bundle, pp 281-288.

60 Exhibit AZ, Diaries tender bundle, pp 290, 292.

Report of the Inquiry into the convictions of Kathleen Megan Folbigg

July 2019

Volume 1: Chapter 9

Chapter 9: Conclusions on review of convictions

Introduction

1. The Inquiry was established at the direction of the Governor of New South Wales to examine a doubt or question as to part of the evidence in the proceedings leading to the conviction of Kathleen Megan Folbigg in May 2003 for the manslaughter of Caleb Folbigg, the malicious infliction of grievous bodily harm and murder of Patrick Folbigg, and the murder of Sarah and Laura Folbigg.

2. The evidence before the Inquiry included evidence available at the time of the trial, both that which was before the jury and that which was not, and the new evidence which has come to light after Ms Folbigg's convictions as a result of the Inquiry's investigations.

3. In this chapter I set out a summary of my findings in respect of each of Ms Folbigg's convictions.

Findings relevant to the conduct of the trial

4. Chapter 3 of this Report sets out in detail the conduct of the trial including the pretrial proceedings, evidentiary and procedural rulings made during trial and Ms Folbigg's sentence and the subsequent appeals.

5. As set out in Chapter 1, a reasonable doubt as to guilt may arise from a procedural irregularity or some error in the trial process.

6. The fact that some points have previously been the subject of scrutiny and decision as part of an appellate process is not conclusive of my task. I have therefore formed my own view on these issues, and the conduct of the trial more generally.

7. In doing so, in light of the submissions made by Ms Folbigg in the Inquiry, I have considered the separate trials application, the use made of the coincidence evidence at trial and the conduct of the Crown prosecutor.

8. I agree with the findings and decisions made pre-trial, during the trial and on appeal. Having considered the evidence carefully, there is no reasonable basis for me to take a contrary view. On the basis of the evidence available at the time, I find no error or procedural irregularity in the trial process that causes me to have a reasonable doubt as to Ms Folbigg's guilt.

Use of coincidence evidence in the Inquiry

9. In addition to taking issue with the use of coincidence evidence at trial on the basis of the evidence available at the time, Ms Folbigg submitted that the evidence adduced in the Inquiry substantially undermined the coincidence evidence.[1] She submitted that this means that on the evidence as it stands now, the application for separate trials would be granted and that I should proceed in my assessment of her guilt without using coincidence reasoning.[2]

10. I disagree. To the contrary, it is clear that the 10 coincidence points relied on by the prosecution at the close of Ms Folbigg's trial remain as striking points of similarity. Each of the children died suddenly and unexpectedly at home, during a sleep period, in circumstances where the only person at home or awake was Ms Folbigg, and were found by her around or shortly after their deaths. None of the evidence in the Inquiry has materially changed these points so as to affect the similarity between them. I do not consider that the evidence in the Inquiry undermines the validity of the coincidence evidence.

11. I entirely agree with Wood CJ at CL, who considered these 10 points of coincidence in the context of the application for separate trials, and also with the judgment of the Court of Criminal Appeal, which reviewed Wood CJ at CL's judgment. In refusing leave to appeal, Hodgson JA considered that he would find a deficiency of proof of guilt in relation to each count without the evidence concerning the other children, but that additional evidence concerning the others would leave no rational view consistent with innocence.[3] Sully J made the point in the appeal against conviction, that even if the trials had been separated, the Crown would be entitled to call evidence of all the other deaths in any single trial. I agree with that observation and find that this remains the case following the evidence heard in the Inquiry. It is also still the case that this evidence has significant probative value and is not outweighed by any unfair prejudice.

SIDS

12. The understanding of SIDS at the time of trial is set out in Chapter 4 of the Report. On the basis of what were considered at that time to be the main risk factors, each of the children's risk of SIDS was low.

13. In the years since Ms Folbigg's trial, SIDS has remained a diagnosis of exclusion based on an absence of any other cause and is understood as being multifactorial.

14. However, since 2003 there have been advances by way of further research on SIDS, including categorising of sudden unexplained deaths in infants and children, and greater knowledge of risk factors associated with SIDS.

15. Changes in the definition of SIDS since 2003 do not materially add to an understanding of the cause of the Folbigg children's deaths. Sub-categories were introduced in 2004, however these do not alter the circumstances of their deaths. Caleb's and Sarah's deaths were both outside the age at which SIDS usually occurs, between two and four months. Each had a mild infection, but on the basis of expert medical evidence it is not realistic that either infection caused death. It has never been contended that Patrick's or Laura's death was associated with SIDS.

16. Recent research emphasises the significance of sleep position and maternal smoking to increasing an infant's risk of SIDS. Each Folbigg child was found in a safe sleeping position and their mother did not smoke. In light of current understanding of risk factors and SIDS, the children's risk at the time of their deaths was in fact even lower than as understood at the time of Ms Folbigg's trial.

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1 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [15].

2 Submissions of Ms Folbigg to the Inquiry (7 June 2019) Part E, [6].

3 *R v Folbigg* [2003] NSWCCA 17, [32].

17. I find that the evidence about SIDS, including fresh expert evidence received in the Inquiry about advances in understanding of SIDS since the time of trial, does not give rise to a reasonable doubt as to Ms Folbigg's guilt.

Recurrence

18. It is clear from investigations conducted by the Inquiry, as also set out in Chapter 4, that before 2003 there had been reported cases involving the deaths of three or more infants in the same family attributed to unidentified natural causes, or at least not established as attributable to unnatural causes. To the extent that the Crown case as left to the jury at Ms Folbigg's trial asserted or invited otherwise, that was incorrect.

19. However, the current descriptions in literature and in evidence by experts emphasise the low nature or rarity of recurrence risk, something that was accurately reflected in the directions of the trial judge.

20. In light of the above, I am satisfied that the treatment of the issue of recurrence at trial has not resulted in a miscarriage of justice or irregularity that gives rise to a reasonable doubt as to Ms Folbigg's guilt.

21. In terms of my approach to the issue of recurrence in the Inquiry, I note that the weight of the evidence is that any increased risk of recurrence in a family of a death attributed to unidentified natural causes is affected by genetic and environmental factors. In the Folbigg family, no genetic factor has been identified and, as I have found in relation to SIDS, environmental factors gave rise to a low risk of sudden unexplained infant death.

22. It was not the Crown case at the trial, and nor would I entertain, that the fact of the deaths of four children in unexplained and sudden circumstances is sufficient to prove guilt beyond reasonable doubt. The rarity of SIDS, and the greater rarity of a repeat instance of SIDS in the same family, are circumstances which are relevant for me to take into account, but are in no way determinative.

Medical evidence

23. Chapter 5 of this Report sets out the significant amount of medical evidence before the Inquiry in respect of each of the children. That evidence comprises all of the medical evidence from the time of the trial, including the evidence before the jury, and the expert reports and evidence that were not before the jury. It also includes the medical evidence received in the Inquiry.

24. I summarise my findings in relation to the medical evidence, when considered in isolation, below.

Caleb

25. Taking into account all the medical evidence available to me, there remains no identified natural cause of Caleb's death.

26. Evidence available to the Inquiry does not completely exclude laryngomalacia, or a floppy larynx, as a possible contributor, but I find the possibility is so low as to be remote. No forensic pathologist gave an opinion at trial or in the Inquiry that laryngomalacia was the cause of Caleb's death. I find that there is no reasonable possibility that Caleb's death was caused by laryngomalacia.

27. Caleb's risk factors for SIDS were low given his mother did not smoke and he was found supine, but his laryngomalacia may have contributed to a vulnerability in that regard.

28. I find that on the basis of the medical evidence in isolation, both "undetermined" and SIDS can apply to Caleb's death. Both descriptors leave open the possibility of an unidentified natural cause, or unidentified unnatural cause, of death. Expert evidence was consistent in both the trial and the Inquiry that it can be very difficult, indeed virtually impossible, to distinguish between SIDS and suffocation at autopsy.

Patrick's ALTE

29. On the medical evidence before me, I am satisfied encephalitis has been excluded as a possible cause of Patrick's ALTE.

30. The following conditions cannot be excluded as having caused the ALTE, but I find that in light of the medical evidence it is not reasonably possible that any of them caused the ALTE:

    a. epilepsy, or an initial seizure;

    b. an unidentified genetic or metabolic condition;

    c. an unidentified degenerative brain condition or neurological disease or condition;

    d. infection or virus (including particularly pneumonia, meningitis, septicaemia, meningococcal and bronchiolitis) other than encephalitis; and

    e. a SIDS-type event.

31. I find that on the basis of expert opinion evidence, it is reasonably possible that Patrick's ALTE was caused by a single asphyxial event on 18 October 1990, with a cause other than one attributable to a respiratory or a recognised neurological condition. For clarity, by "asphyxial", I mean an event leading to obstruction of his airways.

Patrick's death

32. When considering the medical evidence in isolation, in respect of Patrick's death I find that it is possible, on the basis of forensic pathology opinions in the Inquiry, that this was attributable to encephalopathy in his brain. While this identifies a possible cause, it does not explain how the encephalopathy caused death. On the basis of the opinions of Professors Duflou, Hilton and Cordner, and that no relevant medical expert either at trial or in the Inquiry ruled out the possibility of a seizure, it is possible that the encephalopathy caused a seizure, which caused death. I note, however, evidence in the Inquiry of the rarity of SUDEP.

33. I also find that the medical evidence establishes that it is reasonably possible that Patrick's death was caused by an asphyxial event, and which in context was some obstruction from a cause other than a seizure. This was the view of Dr SinghKhaira, Dr Cala, Dr Beal, Professor Herdson, Professor Berry, Professor Ouvrier and Dr Wilkinson at trial. Dr Cala maintained this view in the Inquiry and it could not be ruled out by Professors Duflou and Cordner.

Sarah

34. Taking into account all the medical evidence available to me, there remains no identified natural cause of Sarah's death.

35. I find on the available medical evidence that it is only conjecture that Sarah's death was caused by obstruction of her airways associated with her uvula. On the forensic pathology evidence there is no reasonable possibility this caused her death.

36. I place no significance on the abrasions on Sarah's chin noted by Professor Hilton at autopsy. They could have been due to resuscitation or some other cause completely unrelated to her death.

37. I do not accept that Sarah was sharing her parents' bed on the night she died. That proposition is contrary to more reliable factual evidence, including that of both Ms and Mr Folbigg.

38. As with Caleb, Sarah's risk of SIDS was low given her mother did not smoke and she was found supine, but I find that SIDS could apply to Sarah's death. That leaves open the possibility, on the medical evidence in isolation, of

an unidentified natural cause, or unidentified unnatural cause. It is also reasonably possible that her death was caused by an asphyxial event.

Laura

39. The evidence given in the Inquiry is not significantly different from the evidence given at trial where Professor Berry said most forensic pathologists would say myocarditis was the cause of death of Laura.[4] That is clearly because from the point of view of a forensic pathologist, myocarditis is the only possible observable cause of Laura's death. Having regard to the medical evidence in isolation, it is a reasonable possibility that the myocarditis found in Laura's heart at autopsy was either incidental to her death, or that it was fatal.

40. I accept that deaths of children from myocarditis are rare, and that deaths of children from myocarditis which are sudden and unexpected are even fewer. However, this is simply one circumstance to take into account. It is a separate consideration from whether Laura's myocarditis was sufficient to kill; I have accepted that most forensic pathologists would say that it was.

41. It is also reasonably possible on the medical evidence that Laura's death was caused by an asphyxial event.

42. I find that the presence of the agonal rhythm when an ECG was conducted on Laura by ambulance officers does not assist in determining the cause of her death.

Smothering

43. I accept the evidence of the forensic pathologists, both those who gave evidence at the Inquiry and those who prepared a report for or gave evidence at trial, that it is very difficult to distinguish smothering from SIDS at autopsy.

44. In short, the medical evidence received in the Inquiry, when considered in isolation and not in light of any other evidence, neither proves nor disproves that any of the children were smothered.

45. However, no forensic pathologist has excluded the possibility that each instance of death or ALTE could have been caused by smothering. That is a circumstance which, when forming my conclusions, I take into account.

46. In a circumstantial case, a finding of guilt should be the only rational conclusion that can be drawn from the circumstances. The question of smothering is the ultimate issue, with an opinion on this to be formed upon all of the circumstantial evidence, to the criminal standard of proof.

Immunology

47. In Chapter 6 I considered the evidence relevant to immunology, microbiology and infection.

48. Organisms were found on autopsy in each of Patrick, Sarah and Laura. At trial, the evidence was that there were signs consistent with mild infection in Sarah and Laura, and that the organisms found were largely thought to be postmortem contaminants. None of the findings at autopsy were considered significant or causative of death.

49. In the Inquiry, the forensic pathologists were all of the view that the microbiological findings were likely post-mortem contaminants, with Professor Cordner preferring to "keep open the possibility that there is something there" in respect of Sarah.

[4] Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000) p 26.

50. The microbiology and immunology experts in the Inquiry, by comparison, gave varying views on the significance of the organisms. I have assessed their evidence carefully, but find that it goes no further than to speculate that infection may have played a part in some of the Folbigg children's deaths.

51. In view of the clear opinions of the forensic pathologists and their collective depth of experience, I prefer the view that the microbiological findings at autopsy were likely contamination.

52. I accept that current research and scientific material on infection was not available at trial which refers to a scientific theory that might explain the continuing mortality rate from sudden infant death. However, it cannot be elevated beyond that.

53. The evidence relevant to microbiology, immunology and infection is not such so as to give rise to a reasonable doubt as to Ms Folbigg's guilt.

Genetics

54. As set out in Chapter 2 of the Report, I accept the evidence of Dr Colley that before their sudden deaths, and in Patrick's case before his ALTE, the Folbigg children were healthy, well-grown and normally developing children who were normal in appearance. There was no evidence of congenital malformations, or dysmorphic features, none of the children had a surgical operation or were admitted to hospital with a significant medical problem or were on continuous medication, or had more than eight respiratory infections per year (which was normal for young children), and the tests conducted on the children were all normal. Each child was thriving at the time of their unexpected event. Such a finding is consistent with the medical evidence available, and the evidence of both Mr and Ms Folbigg.

55. Ms Folbigg's medical records were also reviewed and she underwent extensive cardiac testing during the Inquiry. I accept the evidence of Professor Skinner and Associate Professor Raju that Ms Folbigg has no identified cardiac-related condition and that no further investigations would be of material assistance.

56. As discussed in Chapter 7, while limited genetic testing of samples from the children had been conducted by the time of Ms Folbigg's trial, since 2003 there have been significant advances in genetics that now permit a much broader scope of investigation. In particular, since 2013 assessment of genetic information via WGS and WES has become mainstream and this has rapidly increased the rate of discovery of genes responsible for genetic disorders.

57. Accordingly, the Inquiry arranged for WGS to be conducted on samples from Caleb, Patrick, Sarah, and Ms Folbigg and for WES (due to contamination) to be conducted on a sample from Laura. The Sydney and Canberra Teams who interpreted the raw sequencing data arrived at almost identical findings. Neither found variants in genes which were assessed as pathogenic or likely pathogenic in all four children so as to cause their sudden death.[5]

58. The views of the Sydney and Canberra Teams only differed in respect of three genetic variants: one which was only found in Caleb, Patrick, Laura and Ms Folbigg, one which was found in only Patrick, and one which was only found in Sarah, Laura and Ms Folbigg. The Sydney Team considered each of these variants to be variants of uncertain significance; the Canberra Team considered each to be likely pathogenic. The reason for the difference was primarily in relation to the clinical information and its application in the classification process.

[5] Exhibit Z, Joint report of Sydney genetics team (29 March 2019) p 8; Exhibit AF, Joint report of Canberra genetics team (29 March 2019) p 13.

59. In this regard, I prefer the expertise and evidence of the Sydney Team and Professor Skinner. While of course genetics is an area in which there will continue to be rapid advance, on the evidence before the Inquiry I find that there is no reasonable possibility that any of the Folbigg children had a known or recognised pathogenic or likely pathogenic genetic variant which caused their deaths or Patrick's ALTE. The results of the genetic testing do not cause me to have a reasonable doubt as to the guilt of Ms Folbigg.

Non-medical evidence including Ms Folbigg's diaries

60. In Chapter 8 I considered the non-medical evidence before the jury, which included sworn oral evidence from lay witnesses including Mr Folbigg and various friends and neighbours of Mr and Ms Folbigg, as well as the video and transcript of the electronically recorded interview between police and Ms Folbigg on 23 July 1999 before she was charged with any offence.

61. But it was Ms Folbigg's diary entries that constituted the most significant non-medical evidence at trial.

62. It was the Crown case that the entries contained virtual admissions by Ms Folbigg of her guilt for the deaths of Caleb, Patrick and Sarah, as well as admissions by her that she appreciated she was at risk of causing, similarly, the death of Laura. The defence suggested that the diary entries reflected normal reactions, not only of grief, but of shame, guilt and responsibility, though not in the sense contended for by the Crown.

63. On appeal Sully J found an inculpatory interpretation of the entries to be persuasive, concluding that the entries made "chilling reading" and that the probative value of the evidence was "damning".[6]

64. I accept Ms Folbigg's submission, and psychiatrist Dr Diamond's opinion, that Ms Folbigg's diary entries have to be read on the basis of her "deep-seated psychological (but very private) subjective experiences" in light of her early childhood trauma.[7]

65. I find the answers given by Ms Folbigg in examination and crossexamination before me, in which she gave explanations as to the meaning of various diary entries, to be simply unbelievable. I am satisfied the diary entries were written by a reasonably intelligent woman in plain language, carrying their plain meaning. The attempts by Ms Folbigg to explain away the diary entries as saying one sentence did not follow from another sentence, and that they were just "random thoughts", cannot be accepted.

66. Further, I find that Ms Folbigg was untruthful to the police during her interview and in the evidence she gave before me:

    a. I do not accept her evidence that she did not have any concerns about her diary entries. Evidence in the Inquiry from the listening devices, which was not tendered at trial, demonstrates her interest in preventing the content of her diaries being seen. Moreover, the entry in which she hopes a diary "doesn't come back to bite me like my 97 one has" shows she understood very well the danger her diaries presented to her.[8]

    b. In her interview with police in July 1999, Ms Folbigg said she had not written in a diary since May and had thrown all of her diaries out. When Ms Folbigg was told after her interview that the police intended to execute a search warrant on her premises, she immediately said that she did have a new diary that she had bought the day before (which was found to have an entry from July 1999). This completely contradicts what she had just said in the interview and demonstrates her ability to think quickly in an attempt to avoid suspicion.

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6 *R v Folbigg* [2005] NSWCCA 23, [132].

7 Submissions of Ms Folbigg to the Inquiry, (7 June 2019) Part D – Diaries, [57]; Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 41.

8 Exhibit AZ, Diaries tender bundle, p 290.

c. Her statements in her interview and evidence in the Inquiry about events on the night Sarah died are also contradictory and cannot all be true.

67. I find that Ms Folbigg's untruthfulness to the police and in the evidence she gave before the Inquiry was a deliberate attempt to obscure the fact that she committed the offences of which she was convicted.

68. None of the extensive non-medical evidence before me, including Ms Folbigg's evidence, causes me to interpret the diary entries other than in accordance with the ordinary English meaning of the words which she wrote. Rather than supporting any existence of a reasonable doubt of her guilt, I am satisfied that the plain meaning interpretation of the diary entries carries the character contended by the Crown at the trial of virtual admissions of guilt for the deaths of Caleb, Patrick and Sarah and admissions that she appreciated she was at risk of causing similarly the death of Laura.

Findings in respect of each of Ms Folbigg's convictions

Manslaughter of Caleb Gibson Folbigg

69. On the medical evidence in isolation, I find that there was no reasonable possibility that an infection or a genetic disorder was responsible for Caleb's death.

70. Taking into account the non-medical evidence, including the diary entries made by Ms Folbigg and her lies and obfuscation, and the tendency and coincidence evidence, I find that the only conclusion reasonably open is that Ms Folbigg smothered Caleb.

71. I have no reasonable doubt as to the guilt of Ms Folbigg for the manslaughter of Caleb Gibson Folbigg on 20 February 1989.

Malicious infliction of grievous bodily harm upon Patrick Allen Folbigg

72. On the medical evidence in isolation, I find that it was reasonably possible that Patrick's ALTE was caused by a single asphyxial event on 18 October 1990, with a cause other than one attributable to a respiratory or a recognised neurological condition. For clarity, by "asphyxial", I mean an event leading to obstruction of his airways. There was no reasonable possibility that an infection or a genetic disorder was responsible for Patrick's ALTE.

73. Taking into account the non-medical evidence, including the diary entries made by Ms Folbigg and her lies and obfuscation, and the tendency and coincidence evidence, I find that the only conclusion reasonably open is that Ms Folbigg deliberately obstructed Patrick's airways which resulted in his ALTE.

74. I have no reasonable doubt as to the guilt of Ms Folbigg for the malicious infliction of grievous bodily harm upon Patrick Allen Folbigg on 18 October 1990.

Murder of Patrick Allen Folbigg

75. On the medical evidence in isolation, I find that it was possible that Patrick's death was attributable to encephalopathy in his brain in the sense that the encephalopathy caused a seizure, which in turn caused death. I also find that it was reasonably possible that Patrick's death was caused by an asphyxial event, by which I mean an event leading to obstruction of his airways, and which in context was some obstruction from a cause other than a seizure. There was no reasonable possibility that an infection or a genetic disorder was responsible for Patrick's death.

76. Taking into account the non-medical evidence, including the diary entries made by Ms Folbigg and her lies and obfuscation, and the tendency and coincidence evidence, I find that the only conclusion reasonably open is that Ms Folbigg smothered Patrick.

77. I have no reasonable doubt as to the guilt of Ms Folbigg for the murder of Patrick Allen Folbigg on 13 February 1991.

Murder of Sarah Kathleen Folbigg

78. On the medical evidence in isolation, I find that it is reasonably possible that Sarah's death was caused by an asphyxial event, by which I mean an event leading to obstruction of her airways, including smothering. There was no reasonable possibility that an infection or a genetic disorder was responsible for Sarah's death.

79. Taking into account the diary entries made by Ms Folbigg and her lies and obfuscation, the evidence of Mr Folbigg indicating Ms Folbigg's fraught relationship with Sarah, and the tendency and coincidence evidence, I find that the only conclusion reasonably open is that Ms Folbigg smothered Sarah.

80. I have no reasonable doubt as to the guilt of Ms Folbigg for the murder of Sarah Kathleen Folbigg on 30 August 1993.

Murder of Laura Elizabeth Folbigg

81. On the medical evidence in isolation, I find that it is a reasonable possibility the myocarditis found in Laura's heart at autopsy was fatal. It is also reasonably possible that her death was caused by an asphyxial event, by which I mean an event leading to obstruction of her airways, including smothering. There was no reasonable possibility that an infection or a genetic disorder was responsible for Laura's death.

82. Taking into account the rarity of myocarditis as a cause of death in children of Laura's age, the diary entries made by Ms Folbigg, her lies and obfuscation, the evidence of Mr Folbigg indicating the difficulties Ms Folbigg was having with Laura, and the tendency and coincidence evidence, I find that the only conclusion reasonably open is that Ms Folbigg smothered Laura.

83. I have no reasonable doubt as to the guilt of Ms Folbigg for the murder of Laura Elizabeth Folbigg on 1 March 1999.

Final comments

84. The trial judge in his sentencing remarks expressed his own agreement with the verdict of the jury. In the first appeal to the Court of Criminal Appeal the three judges in dismissing the appeal noted there "was ample evidence" to justify the findings of the jury.[9] In the second appeal to the Court of Criminal Appeal the three judges who heard the appeal described the Crown prosecution case as "overwhelming".[10] The three judges of the High Court who heard the special leave application obviously agreed when dismissing that application. I fully agree with that description as the evidence stood at the trial.

85. My observations of the consideration given to this matter by intermediate and High Court justices is not to suggest that my findings in this Inquiry were in any sense foreclosed. They were not, and particularly so given the extensive investigations conducted by, and evidence received in, the Inquiry to enable careful forensic scrutiny of Ms Folbigg's convictions. None of that material was before the appeal courts in their decisions between 2003 and 2007.

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9 *R v Folbigg* [2005] NSWCCA 23, [143].

10 *Folbigg v R* [2007] NSWCCA 371, [64].

86. However, the significant investigations conducted by the Inquiry into the four deaths and the ALTE have failed to identify a reasonable natural explanation for the five events, individually or together, as outlined above and in the previous chapters of this Report.

87. The investigations of the Inquiry have instead produced evidence that reinforces Ms Folbigg's guilt. I find Ms Folbigg's evidence and the listening device transcripts, neither of which were before the jury, when considered in light of her interview with police, show that Ms Folbigg has been in many respects untruthful, unbelievable and made deliberate attempts to obscure the fact that she committed the offences of which she was convicted.

88. It remains that the only conclusion reasonably open is that somebody intentionally caused harm to the children, and smothering was the obvious method. The evidence pointed to no person other than Ms Folbigg.

89. The evidence at the Inquiry does not cause me to have any reasonable doubt as to the guilt of Kathleen Megan Folbigg for the offences of which she was convicted. Indeed, as indicated, the evidence which has emerged at the Inquiry, particularly her own explanations and behaviour in respect of her diaries, makes her guilt of these offences even more certain.

Report of the Inquiry into the convictions of Kathleen Megan Folbigg

July 2019

Volume 1: Chapter 10

Chapter 10: Sentence

Introduction

1. Pursuant to s 82(2)(b) of the CAR Act I may refer the matter to the Court of Criminal Appeal for review of the sentence imposed if I am of the opinion that there is a reasonable doubt as to any matter that may have affected the nature or severity of Ms Folbigg's sentence.

2. At the conclusion of Ms Folbigg's evidence, psychiatric reports recently prepared by Dr Michael Diamond and Dr Michael Giuffrida (see below), together with reports previously prepared by psychiatrists at the time of trial by Dr Michael Giuffrida, Dr Bruce Westmore and Dr Yvonne Skinner, were received into evidence. This was because I formed the view that Ms Folbigg's evidence about the diaries had rendered expert opinion about her mental state relevant.

3. The recent report of Dr Diamond contained an opinion as to a diagnosis of "Complex Post-traumatic Stress Disorder" ("Complex PTSD"). This diagnosis had not been made in the reports tendered before the sentencing judge in determining Ms Folbigg's sentence and subsequently considered by the Court of Criminal Appeal.[1]

4. In light of this diagnosis, those assisting the Inquiry obtained a further opinion of Dr Giuffrida. Dr Giuffrida prepared a report dated 13 May 2019, expressing his opinions about Dr Diamond's diagnosis of Ms Folbigg.

5. In this chapter I examine the psychiatric reports tendered and form an opinion as to whether there is a reasonable doubt as to any matter that may have affected the nature or severity of Ms Folbigg's sentence.

2003 Report of Dr Michael Giuffrida

Early assessments of Ms Folbigg

6. Dr Giuffrida assessed Ms Folbigg on two occasions at Mulawa Correctional Centre in his capacity as Visiting Medical Officer Psychiatrist to Corrections Health on 22 May 2003 and 5 June 2003, arising from concerns as to possible risks of self-harm.[2]

1 Transcript of the Inquiry, 1 May 2019 T811.28-812.3.

2 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 2.

7. At the time of both assessments Ms Folbigg was 35 years old, had been separated from Mr Folbigg for three years, and was being held in isolation in an induction unit at Mulawa Correctional Centre where an assessment was being made regarding longer term placement.3 Dr Giuffrida noted the following from his brief mental state examination of Ms Folbigg on 22 May 2003:

> *Remarkably calm and detached and able to speak at length without distress at any point, strikes me as being affectless in this situation. Spoke clearly and coherently without any hint of thought disorder, delusional ideas or particular preoccupation other than details of her offences. I found her remarkably lacking in the expression of grief in relation to these.*[4]

8. At the assessment on 5 June 2003, Ms Folbigg agreed with Dr Giuffrida that she came across as being emotionally detached and noted that her mother and foster sister always said that she "built a brick wall around her emotions".[5] She explained that she had always coped with conflict and crises in this way.[6]

Engagement

9. Dr Giuffrida was formally engaged by Ms Folbigg's representatives to produce a "comprehensive psychiatric report" in advance of sentence.[7]

10. Dr Giuffrida was briefed with a summary of facts from police, Ms Folbigg's diaries as tendered at trial, a selection of "Defence extracts which were not tended [sic] at the trial",[8] Ms Folbigg's medical records and her Family and Community Services file.[9]

11. He examined Ms Folbigg for the purposes of preparing a report on 19 June 2003, 31 July 2003, 12 August 2003 and 14 August 2003 and referred in particular to two long sessions,[10] "each of about two hours discussing her relationship with each of her children and her husband."[11]

12. Following these sessions, Dr Giuffrida prepared a report dated 27 August 2003, which was ultimately tendered before the sentencing Judge.[12]

Diagnosis and conclusions

13. Dr Giuffrida described his early assessment of Ms Folbigg in May 2003 as revealing no psychiatric disorder or anything to indicate any underlying personality disorder, with the exception of the apparent detachment regarding the death of her children.[13]

14. Following his further sessions with her, Dr Giuffrida came to the following conclusions:

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3 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 2.
4 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 3.
5 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 4.
6 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 4.
7 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 1.
8 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 18.
9 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) pp 1-2.
10 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) pp 1, 4.
11 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 4.
12 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003).
13 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 3.

a. Although at times Ms Folbigg could engage warmly and responsively, there was "always a somewhat blunted, distant even remote quality to her ability to relate."[14] In light of her otherwise graphic descriptions of the deaths of her children, he found it "highly significant" that there was a remarkable inertness of emotional response to such discussions about their deaths and he was unable to elicit any symptoms suggestive of her reliving the events.[15]

b. He could find no evidence of any disorganisation of thinking, formal thought disorder, over-valued or delusional ideas or perception abnormality.[16]

c. She was of at least average verbal intelligence with no evidence of development disability.[17]

d. There was no clear evidence of psychotic illness, "remarkably little" to suggest any serious personality disorder and a "remarkable absence" of historical features or the core criteria for psychopathy.[18]

e. She had a history of pervasive depression, sometimes called a chronic dysthymia, which seemed to become more intense and long lasting after the death of each child. He concluded this "probably represents Ms Folbigg's particular expression of grief and bereavement".[19]

f. He did not consider Ms Folbigg suffered from a psychotic level of depression, but that it was serious and persistent enough to have strongly contributed to a state of mind that led to her killing her children.[20]

g. Her response to the death of her children was characterised by "an extraordinary absence of any of the normal mourning or bereavement signs" and did not reveal the symptoms expected of post-traumatic stress disorder ("PTSD").[21]

h. He described Ms Folbigg's case as a "very significant phenomenon" following the trauma she experienced as a young girl.[22] This resulted in a profound and probably irreversible impairment of her capacity to develop any meaningful emotional bonding and attachment, which "contributed in some part at least to her total inability to relate, care for and protect her own children".[23]

Report of Dr Bruce Westmore

Engagement

15. Dr Westmore first examined Ms Folbigg on 13 September 2002, and 21 January 2003, although on the documents available the purpose of these assessments is unclear.[24]

16. Following Ms Folbigg's convictions, Legal Aid briefed Dr Westmore to "psychiatrically re-examine Kathleen".[25] In his report dated 16 June 2003 which was tendered at sentencing, Dr Westmore noted that if he assumed Ms Folbigg did kill her children or was responsible for their deaths, he would have to "ask myself why these things occurred."[26]

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14 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 14.
15 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 14.
16 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 14.
17 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) pp 14, 20.
18 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 20.
19 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 19.
20 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 20.
21 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 20.
22 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 20.
23 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003) p 22.
24 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 1.
25 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 1.
26 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 2.

17. Dr Westmore refers to being briefed with "a large number of documents relating to the trial and its outcome", which included as least some of the diary entries.[27]

Diagnosis and conclusions

18. At the time of assessment in June 2003, Dr Westmore did not consider Ms Folbigg suffered from a major depressive illness and noted there were no psychotic features evident.[28]

19. He did not consider her history to be consistent with the diagnosis of Munchausen syndrome by proxy but thought that it would be reasonable to assume that she "suffers from a severe personality disorder with anger and impulse control being central difficulties."[29]

20. It was Dr Westmore's view that at the time of the offending that Ms Folbigg's mind was not distorted or disturbed by postpartum depression and she did not suffer any other clearly identifiable psychiatric illness which led her to behave aggressively towards her children.[30]

21. Dr Westmore concluded:

> *Based on the assumption that she was indeed responsible for the death of her children, it is probable in my view that she displaced onto the children her own anger and frustration with the difficulties she was having with her partner. It is unclear to me to what extent childhood difficulties played any immediate role in her behaviours although her childhood history is likely to have influenced her personality development…*
>
> *Her own concerns about not being a good or adequate mother, combined with her personality difficulties and vulnerability and her problems dealing with emotions such as anger and depression and frustration are all likely in combination to have led her to feel she could not cope with the children and subsequently her acting towards them in a way in which caused their deaths.*[31]

Report of Dr Yvonne Skinner

Instructions and briefing material

22. Dr Skinner was briefed by the Office of the DPP prior to Ms Folbigg's trial to prepare a report presenting her: "opinion as to whether an unbalance of mind arose from birth or lactation in the accused, as opposed to any other abnormality or character defect".[32]

23. She was briefed with material from trial including a statement of the Crown prosecution case and witness statements, as well as Ms Folbigg's diaries, ERISP and DOCS file.[33] She prepared a report dated 22 January 2003 which was tendered at sentence.[34]

24. Dr Skinner did not have the opportunity to examine Ms Folbigg.

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27 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 4.
28 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 4.
29 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) pp 5-6.
30 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 6.
31 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003) p 6.
32 Letter from ODPP to Dr Yvonne Skinner (6 December 2002) p 1.
33 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 1.
34 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003).

Diagnosis and conclusions

25. Dr Skinner concluded that she was unable to find any evidence to suggest that Ms Folbigg was suffering from a mental illness or mental disorder, or that she was suffering from a significant degree of depression.[35]

26. Dr Skinner acknowledged that Ms Folbigg had an "emotionally disturbed childhood" characterised by an "unsatisfactory foster placement, institutional placement and later a foster placement that proved more satisfactory".[36] In considering the significance of Ms Folbigg's chaotic early childhood, Dr Skinner stated:

> *Most psychiatrists would agree the background history of such disturbance would lead to personality problems or possibly psychiatric disorder, but studies show that there is no recognisable link between such childhood emotional disturbance and a particular psychiatric disorder or psychological condition.*[37]

27. Dr Skinner was not able to find any evidence that Ms Folbigg suffered from a postpartum psychiatric disorder, nor any other psychiatric condition that might have affected her judgment or ability to cope.[38]

Consideration of psychiatric evidence at sentence

28. At sentence, the psychiatric reports of Drs Skinner, Giuffrida and Westmore were tendered before the sentencing judge.[39] The sentencing judge also had the benefit of the oral evidence of Dr Westmore.[40]

29. Noting that Dr Skinner did not examine Ms Folbigg, and the limited scope of her brief regarding the availability of a psychiatric defence before trial, the sentencing judge determined Dr Skinner's report to be of limited assistance.[41]

30. However, his Honour accepted the evidence of Drs Giuffrida and Westmore, which he summarised as follows:

    a. by 18 months of age Ms Folbigg was a seriously disturbed and regressed little girl, and by this stage was severely traumatised;[42]

    b. antisocial personality disorder was not an appropriate diagnosis in Ms Folbigg's case;[43]

    c. Ms Folbigg was not psychotic;[44]

    d. the overall theme of the diaries is of a woman always coping at the margins of her capacity to bond, relate to, provide for and care for her children, a woman roused easily to panic and readily defeated by any perception on her part that she might fail to provide for her children;[45]

    e. the stresses on Ms Folbigg of looking after a young child were greater than those which would operate on an ordinary person because she was psychologically damaged and barely coping;[46]

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35 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 13.
36 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 13.
37 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 13.
38 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 13.
39 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003); Exhibit BB, Report of Dr Bruce Westmore (25 August 2003); Exhibit BD, Report of Dr Michael Giuffrida (17 August 2003).
40 *R v Folbigg* [2003] NSWSC 895, [71].
41 *R v Folbigg* [2003] NSWSC 895, [50].
42 *R v Folbigg* [2003] NSWSC 895, [51].
43 *R v Folbigg* [2003] NSWSC 895, [56].
44 *R v Folbigg* [2003] NSWSC 895, [57].
45 *R v Folbigg* [2003] NSWSC 895, [66].
46 *R v Folbigg* [2003] NSWSC 895, [91].

f. throughout these events Ms Folbigg was depressed and suffering from a severe personality disorder, and her capacity to control her behaviour was severely impaired;[47]

g. throughout her marriage Ms Folbigg was affected by the abuse perpetrated on her during the first 18 months of her life and the effects of this included an inability to form a normal, loving and forbearing relationship with her children;[48]

h. her depression went unrelieved and on occasions turned itself into anger;[49]

i. Ms Folbigg's mental state and her anxiety about it left her unable to shrug off the irritations of unwell, wilful and disobedient children, and she was not fully equipped to cope;[50] and

j. on occasions she appeared cool, detached, self-interested and unaffected by the fate of her children but in truth she suffered remorse which she could not express.[51]

31. The sentencing judge considered that the above findings provided "significant mitigation of [Ms Folbigg's] criminality"[52] so as to avoid the imposition of the maximum penalty of life imprisonment. He instead sentenced her to an effective head sentence of 40 years' imprisonment with a non-parole period of 30 years.[53]

Ms Folbigg's appeal against sentence

32. Ms Folbigg appealed to the Court of Criminal Appeal against both conviction and sentence. In respect of her sentence appeal, Sully J, Dunford and Hidden JJ agreeing, confirmed that the sentencing judge's findings in respect of the objective criminality of the offending were open to him, particularly in light of the evidence of Dr Westmore and Dr Giuffrida.[54] Sully J considered that it was important to note that the:

> *Psychological damage to which Barr J refers to in paragraph 91… was not trifling or peripheral damage, but was serious, deep-seated damage caused over a period of some years commencing when the appellant was a baby. The details make sad and shocking reading. It is unnecessary now to rehearse all of the ugly and distressing particulars.*[55]

33. Ms Folbigg's appeal against sentence was allowed on the grounds that:

a. there was an identifiable error in Barr J's method of cumulation that resulted in offering Ms Folbigg a "prospect… so crushingly discouraging as to put at risk any incentive that she might have to apply herself to her rehabilitation";[56] and

b. the overall result of a head sentence of 40 years and a non-parole period of 30 years was so crushing it appeared to be a "life sentence by a different name".[57]

34. Accordingly, Ms Folbigg was re-sentenced on two counts to result in an effective head sentence of 30 years with a non-parole period of 25 years.[58]

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47 *R v Folbigg* [2003] NSWSC 895, [94].
48 *R v Folbigg* [2003] NSWSC 895, [95].
49 *R v Folbigg* [2003] NSWSC 895, [95].
50 *R v Folbigg* [2003] NSWSC 895, [95].
51 *R v Folbigg* [2003] NSWSC 895, [96].
52 *R v Folbigg* [2003] NSWSC 895, [93].
53 *R v Folbigg* [2003] NSWSC 895, [100].
54 *R v Folbigg* [2005] NSWCCA 23, [169].
55 *R v Folbigg* [2005] NSWCCA 23, [171].
56 *R v Folbigg* [2005] NSWCCA 23, [186].
57 *R v Folbigg* [2005] NSWCCA 23, [189].
58 *R v Folbigg* [2005] NSWCCA 23, [191].

2019 report of Dr Michael Diamond

Instructions

35. Dr Michael Diamond was instructed by Ms Folbigg's representatives in the Inquiry to prepare an expert report specifically addressing the following:

> *History taken by you;*
>
> *Diagnosis;*
>
> *Prognosis;*
>
> *Please advise whether our client's treatment to date has been appropriate;*
>
> *What is your experience in treating and assessing individuals exposed to traumatic instances or circumstances?*
>
> *Please read the diary material provided to you. In light of your diagnosis, if any, and your experience with the treatment and assessment of individuals exposed to traumatic instances, in your opinion, are the diary entries influenced or impacted by any psychological illness from which Ms Folbigg was suffering at the time of writing them?*
>
> *What is survivor guilt?*
>
> *Do you have experience in treating individuals labouring under 'survivor guilt'? If so, please detail that experience.*
>
> *Taking into account your answers to questions 7 and 8 above, in your opinion, were Ms Folbigg's entries in her diaries influenced by 'survivor guilt'? Please provide reasons for your answer.*
>
> *Insofar as your diagnosis differs from Drs Skinner or Westmore, please advise why if you are able; and*
>
> *Any further comments you wish to make.*[59]

Material provided and assessments conducted

36. In preparing his report, Dr Diamond was briefed with over 1,000 pages of material, including the previous psychiatric reports of Drs Westmore, Skinner and Giuffrida, documents relevant to and extracts from the evidence heard at trial, and contemporary material such as Ms Folbigg's Justice Health records.[60]

37. Dr Diamond assessed Ms Folbigg on 25 and 27 March 2019, for extended periods and produced a report dated 16 April 2019.[61]

[59] Exhibit BA, Report of Dr Michael Diamond (16 April 2019), letter of instruction, p 2.
[60] Exhibit BA, Report of Dr Michael Diamond (16 April 2019), letter of instruction, p 1.
[61] Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 1.

Commentary on previous psychiatric reports

Report of Dr Skinner

38. Dr Diamond was critical of Dr Skinner for not clearly describing what he refers to as Ms Folbigg's "history of significant early life disruption of attachments and bonds".[62] He recorded that Dr Skinner's primary concern was excluding any evidence of psychiatric condition capable of producing cognitive disturbance that could impair Ms Folbigg's functioning to the extent that she would have a defence to the charges faced.[63]

39. In response to Dr Skinner's assertion that:

> *studies show that there is no recognisable link between… childhood emotional disturbance and a particular psychiatric disorder of psychological disorder*[64]

Dr Diamond dismissed these studies as "not current".[65]

Report of Dr Westmore

40. Dr Diamond noted that Dr Westmore's report was prepared in the context of guilty verdicts, and as such his focus was on attempting to gain a better understanding of the pathogenesis of Ms Folbigg's offending.[66] The questions put to Ms Folbigg during Dr Westmore's assessment assumed her guilt, and as a result she was at times unable to respond.[67]

41. According to Dr Diamond, Dr Westmore's reporting of Ms Folbigg's early life experience was inconsistent with the account Dr Diamond obtained directly from Ms Folbigg.[68] Dr Diamond claimed Dr Westmore failed to explore Ms Folbigg's "significant early life experiences",[69] but later credited Dr Westmore with acknowledging that Ms Folbigg's "early life experiences are likely to have influenced her personality development".[70]

42. Dr Diamond is critical of Dr Westmore's decision to "extrapolate" in respect of the view individuals who are over-controlled may be prone to episodes of extreme anger, and suggests Dr Westmore makes speculative comments about how Ms Folbigg's observed features may have expressed themselves in a way that resulted in the murder of her children.[71]

Report of Dr Giuffrida

43. Dr Diamond described Dr Giuffrida's report as "comprehensive"[72] and describes Dr Giuffrida's account of Ms Folbigg's history to be consistent with his own.[73]

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62 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 26.
63 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 30.
64 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003) p 13.
65 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 30.
66 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 30.
67 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 31.
68 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) pp 35-36.
69 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 26.
70 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 31.
71 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 31.
72 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 32.
73 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 33.

44. Dr Diamond considered that Dr Giuffrida assessed Ms Folbigg against the backdrop of her having been convicted. He reported that Dr Giuffrida did not consider the "distinct probability that she was suffering trauma related psychiatric illness, currently identified as Complex Post Traumatic Stress Disorder" despite identifying the diagnostic features associated with this condition.[74]

45. On Dr Diamond's assessment, Dr Giuffrida acknowledged the significant and prolonged trauma in the early life of Ms Folbigg to the extent that it has influenced her personality but did not associate it with the features that are commonly observed in abused children who develop Complex PTSD as a pervasive, long term psychiatric disorder.[75]

Diagnosis and conclusions

46. Dr Diamond considered Ms Folbigg's affect at the time of assessment to be unusual, describing her as relating pleasantly but in a "superficial talkative way" and at times she was "emotionally blunted to the point of being detached and disassociated".[76]

47. He assessed her thought processes as rational with no evidence of perceptual distortion, delusional material, hallucinations or persecutory ideation and she presented with at least average intellect. She also showed adequate ability to reason and exhibited sound judgment.[77]

48. Dr Diamond considered it inevitable that Ms Folbigg has been affected by the trauma of her early childhood experiences so as to reflect this in her personality.[78] While she has deep-seated personality vulnerabilities, particularly in establishing and maintaining relationships, she does not exhibit pervasive and severe dysfunction to the point of being able to diagnose a personality disorder.[79]

49. In Dr Diamond's view Ms Folbigg has had episodes of mood disturbance sufficient to make a diagnosis of Persistent Mood Disorder (Dysthymia) and at times has had episodes of Major Depressive Disorder.[80]

50. He opined that the significant and pervasive psychiatric diagnosis is that of Complex PTSD. Dr Diamond did not define this disorder, but noted Ms Folbigg has:

> *lifelong symptoms of emotional detachment, emotional numbing, difficulty trusting, engaging with others and experiencing periods of severe detachment to the point of dissociation*[81]

following the severe disruption of and violence in childhood.[82]

51. Dr Diamond was asked to comment on how his diagnosis differs from those advanced by Dr Skinner and Dr Westmore. He observed the differences in their diagnostic views arise because they considered different aspects of Ms Folbigg's presentation and were asked to address different issues.[83]

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74 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 36.
75 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 36.
76 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 23.
77 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 24.
78 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 39.
79 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 40.
80 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 40.
81 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 39.
82 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 38.
83 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 43.

52. Dr Skinner and Dr Westmore conducted their assessments seeking to identify any severe psychiatric illnesses that could account for Ms Folbigg's role in her children's deaths. While Dr Diamond agreed that he could find no evidence that Ms Folbigg has suffered from psychotic illness, severe mood disorder consistent with homicidal conduct or any other brain injury that might affect her conduct so as to carry out homicidal acts, he noted that neither Dr Skinner or Dr Westmore explored the possibility of a severe life-affecting condition such as Complex PTSD.[84] He considered this to be supported by Ms Folbigg's habitual ways of relating, her communication patterns, and her subjective emotional distress and trauma.[85]

53. Dr Diamond notes that "the significant history regarding her underlying primary psychiatric condition is contained in the DOCS records primarily",[86] discounting a difference in briefing material as a reason for differing diagnoses.

2019 Report of Dr Michael Giuffrida

Engagement

54. Following receipt of Dr Diamond's report, those assisting the Inquiry instructed Dr Michael Giuffrida to review Dr Diamond's report and prepare a short report outlining:

    a. a definition of a diagnosis of Complex PTSD;

    b. whether his opinions as expressed in 2003 in relation to diagnosis have changed or remained the same and why; and

    c. any differences between his opinions and those of Dr Diamond, and to the extent possible, the reasons for those differences.[87]

55. Dr Giuffrida prepared a report dated 10 May 2019.[88]

Definition of Complex PTSD

56. In his report Dr Giuffrida recorded that what may constitute Complex PTSD has presented as a controversial issue for more than 30 years.[89]

57. He confirmed that there is no specific reference to Complex PTSD in the mental disorders section of the International Classification of Diseases.[90] Similarly, while the fifth edition (published in 2013) of the Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association ("DSM V") provided an updated set of diagnostic criteria for PTSD, there is no specific reference to Complex PTSD.[91]

58. However, Dr Giuffrida acknowledged that since the late 1970s there have been a large number of studies which consider and support the concept of Complex PTSD as being:

> *A valid entity to understand the more complex patterns of the clusters of symptoms and behaviours associated with the more extreme forms of trauma suffered particularly by young children and adolescents.*[92]

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84 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 43.
85 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 43.
86 Exhibit BA, Report of Dr Michael Diamond (16 April 2019) p 43.
87 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019), letter of instruction, p 2.
88 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019).
89 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 2.
90 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 2.
91 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 2.
92 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 3.

59. In particular, Dr Giuffrida referred to a 2005 paper by van der Kolk et al which noted that PTSD has only ever captured a limited aspect of posttraumatic psychopathology, particularly in children.[93] The paper refers to a "DSM-IV Field Trial" which was conducted between 1990 and 1992 and found that:

> *Trauma, particularly trauma that is prolonged, that first occurs at an early age and that is of an interpersonal nature, can have significant effects on psychological functioning above and beyond PTSD symptomology. These effects include problems with dysregulation, aggression against self and others, dissociative symptoms, somatization and character pathology.*[94]

60. Dr Giuffrida suggested the following symptoms may also be prominent: impulsive outbursts of anger, self-destructive and suicidal behaviour, aberrant or deviant sexual behaviour, substance abuse, loss of trust, a pattern of revictimisation, risktaking behaviour, amnesia or dissociative type experiences and a sense of hopelessness and loss of beliefs.[95]

Whether Dr Giuffrida's opinion as expressed in 2003 has changed

61. Dr Giuffrida confirmed that in his 2003 report he had prepared a detailed mental state examination to exclude the likelihood that Ms Folbigg had killed one or more of her children as a result of a common psychiatric disorder, having reference to the available literature and his own experience.[96] In particular, he excluded a Borderline or Antisocial personality disorder, or that Ms Folbigg was labouring under delusional beliefs or dissociative phenomena.[97]

62. He was able however to identify a "very clear and strong history of a pervasive depression",[98] that could best be described as "chronic Dysthymia which may have at times reached the intensity of a Major Depressive Episode" which he says was "strongly confirmed by my reading of her diaries."[99]

63. In his 2019 report in relation to PTSD, Dr Giuffrida opined that as a child Ms Folbigg almost certainly did experience some of the Category A symptoms of PTSD as a result of the traumatic events of violence between her mother and father.[100] He also stated that she probably does satisfy some of the Category D symptoms of PTSD including persistent and exaggerated negative beliefs or expectations and feelings and behaviours of detachment and estrangement from others.[101]

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93 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 3; Bessel A van der Kolk, Susan Roth, David Pelcovitz, Susanne Sunday and Joseph Spinazzola, 'Disorders of Extreme Stress: The Empirical Foundation of a Complex Adaptation to Trauma' (2005) 18(5) *Journal of Traumatic Stress 389.*

94 Bessel A van der Kolk, Susan Roth, David Pelcovitz, Susanne Sunday and Joseph Spinazzola, 'Disorders of Extreme Stress: The Empirical Foundation of a Complex Adaptation to Trauma' (2005) 18(5) *Journal of Traumatic Stress 389, 394-395.*

95 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 2.

96 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 6.

97 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) pp 7, 9.

98 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 7.

99 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 9.

100 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 9.

101 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 10.

64. Dr Giuffrida concluded:

> *In short, Ms Folbigg's mental state does satisfy some but certainly not all of the diagnostic criteria of Posttraumatic Stress Disorder… I would firm up my opinion expressed in my report of 2003 that Ms Folbigg has suffered from a pervasive Depression which probably persists and that she shows significant features of a Posttraumatic Stress Disorder both in terms of the symptoms at least to a limited extent and in terms of the high likelihood that she was subject to extreme traumatic events of early childhood in causing such a Posttraumatic Stress Disorder.*[102]

Review of Dr Diamond's diagnosis

65. Dr Giuffrida stated that he agrees with Dr Diamond's diagnosis of Complex PTSD "in terms of the causality of the condition".[103] He considered it appears:

> *Highly likely that Ms Folbigg was the victim of repeated continuous early childhood sexual, physical and emotional abuse and neglect and almost certainly observed extreme domestic violence and possibly the murder of her mother.*[104]

66. However, Dr Giuffrida identified two difficulties with Dr Diamond's diagnosis:

    a. the concept remains somewhat controversial and in any case it involves a drawing together of a constellation of co-morbidities which the DSM V and the International Classification of Diseases has not recognised as a separate independent entity of its own right;[105] and

    b. Dr Diamond does not provide a formal list of reasons for such a diagnosis. In addition to the anxiety and depression experienced by Ms Folbigg, with Complex PTSD there is commonly a history of self-harm and suicidal ideation and attempts, features of Dissociative Identity disorder or Dissociative Amnesia, body image disturbances, emotional dysregulation and Borderline personality disorder features. These symptoms were not readily apparent in Ms Folbigg in 2003 or from Dr Diamond's most recent interview of her.[106]

[102] Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 10.
[103] Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 10.
[104] Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 10.
[105] Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 10.
[106] Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 10.

Submissions regarding Ms Folbigg's mental state

Submissions of counsel assisting

67. Counsel assisting submitted that neither Dr Diamond's nor Dr Giuffrida's 2019 report contains new or fresh evidence about a matter that may have affected the nature or severity of Ms Folbigg's sentence.[107]

68. In counsel assisting's submission, the factors that gave rise to a diagnosis by Dr Diamond of Complex PTSD were before the sentencing Judge. He expressly referred to and accepted the evidence from Dr Westmore and Dr Giuffrida that Ms Folbigg had been traumatised by the events of her childhood, suffered from a personality disorder, depression and anxiety, and that her mental state made it difficult for her to cope and bond, relate to, provide for and care for her children. Further, on appeal, Sully J found that the findings of the sentencing judge in relation to Ms Folbigg's psychological profile were amply open to him.[108]

69. Counsel assisting also emphasised the following points made by Dr Giuffrida in relation to the diagnosis by Dr Diamond:

    a. in Dr Giuffrida's view, Ms Folbigg's mental state satisfies some but not all of the diagnostic criteria for PTSD;

    b. the fact that many common features of Complex PTSD are not present in Ms Folbigg; and

    c. that the concept of Complex PTSD remains somewhat controversial and there is no specific reference to it in DSM V or the International Classification of Diseases.[109]

70. Counsel assisting accordingly submitted that the evidence before the Inquiry, including the report of Dr Diamond, does not give rise to a reasonable doubt as to any matter that may have affected the nature or severity of Ms Folbigg's sentence.[110]

Submissions of others with leave

71. Submissions in respect of sentence were not made by Ms Folbigg or Mr Folbigg.

72. The DPP adopted the submissions of counsel assisting in respect of sentence, submitting that there is no new matter arising in the evidence before the Inquiry, including the report of Dr Diamond, that may affect the nature or severity of Ms Folbigg's sentence.[111]

Findings

73. The report of Dr Diamond and the updated report of Dr Giuffrida were not available to the sentencing judge or to the Court of Criminal Appeal when it dealt with the appeal against sentence.

74. In my view, those reports do not, however, contain any relevantly new knowledge of the longstanding and deep-seated psychological problems of Ms Folbigg. Simply saying that she has Complex PTSD adds nothing to the understanding of her symptoms.

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107 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 10, [194].

108 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 10, [198]-[199].

109 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 10, [196]; Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019) p 10.

110 Submissions of counsel assisting the Inquiry (17 May 2019) Chapter 10, [203].

111 Submissions of the ODPP to the Inquiry (24 May 2019) p 48.

75. One of the submissions made in Ms Folbigg's appeal against sentence was that the sentences did not allow adequately, or indeed at all, for the personal and psychological profiles of Ms Folbigg. This argument was not successful and I see no reason to depart from that decision.

76. Both Drs Diamond and Giuffrida noted Ms Folbigg's lifelong symptoms of emotional detachment and inability to form meaningful bonds. That might explain how she came to commit these offences, but it does not excuse them, and to the extent it was relevant, it was given full effect by the sentencing judge and the Court of Criminal Appeal.

77. I am of the opinion that there is no reasonable doubt as to any matter that may have affected the nature or severity of Ms Folbigg's sentence.

Report of the Inquiry into the convictions of Kathleen Megan Folbigg

July 2019

Volume 1: Addendum

Addendum

Further Information Received Following Closure Of The Evidence And Submissions:

The CALM2 Genetic Variant In Sarah, Laura And Kathleen Folbigg

June 2019 research paper regarding International Calmodulin Registry and CALM3 variant

1. The evidence in the Inquiry closed on 1 May 2019.[1] On 21 June 2019 Professor Vinuesa sent to the Inquiry a further statement, specifically in relation to the CALM2 variant. The statement attached a paper published in June 2019 ("the June 2019 paper"),[2] which reported a family with a variant in the CALM3 gene (p.Gly114Trp or p.G114W) affecting the same amino acid as was identified in the CALM2 gene in Ms Folbigg, Sarah and Laura (p.Gly114Arg or p.Gly114R).

2. The statement also attached a letter dated 20 June 2019 addressed to Professor Vinuesa from one of the authors of the June 2019 paper, Professor Peter Schwartz, Director at the Centre for Cardiac Arrhythmias of Genetic Origin at the Istituto Auxologico Italiano in Milan, Italy.[3] The circumstance in which Professor Schwartz's letter to Professor Vinuesa came about appears to be that Professor Vinuesa provided to Professor Schwartz the Canberra report, authored by her and Professor Cook in relation to the genetic testing of the Folbigg family and tendered in the Inquiry.[4]

3. In his letter Professor Schwartz explained that the Registry of Calmodulinopathy referenced in the June 2019 paper included a family with an asymptomatic mother carrying the CALM3 variant, one child who died at age five from a cardiac arrest while playing, and another who died suddenly at age four.[5]

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1 At the close of the substantive hearings, at the request of Ms Folbigg's representatives, the Judicial Officer directed that Ms Folbigg's representatives had until 7 May 2019 to seek the tender of any further documents. Further documents were tendered at this point.

2 Exhibit BU, Lia Crotti et al 'Calmodulin Mutations and Life-Threatening Cardiac Arrhythmias: Insights from the International Calmodulinopathy Registry' (2019) *European Heart Journal* (advance).

3 Exhibit BT, Letter from Professor Peter Schwartz to Professor Carola Vinuesa (20 June 2019).

4 Exhibit AF, Joint report of Canberra genetics team (29 March 2019).

5 Exhibit BT, Letter from Professor Peter Schwartz to Professor Carola Vinuesa (20 June 2019) [2]-[3].

4. Professor Schwartz noted that the report he had seen (the Canberra report) only linked the CALM2 variant to a long QT syndrome phenotype, ignoring the possibility that the phenotype could be CPVT.[6] He noted that without an exercise stress test of Ms Folbigg, a diagnosis of CPVT was still "fully on the table". Professor Schwartz suggested the CALM2 variant and the absence of a stress test justified the re-opening of the Inquiry.[7]

5. It is apparent that Professor Schwartz was provided only with the Canberra report, and none of the evidence given in the Inquiry or the historical or recent clinical presentation information in relation to Ms Folbigg, including the April 2019 stress test conducted upon her and Professor Skinner's and Associate Professor Raju's opinions as to the results of that test.[8] Most significant of this material, in light of Professor Schwartz's view as to the potential diagnosis of CPVT, is Professor Skinner's opinion based on that stress test that Ms Folbigg does not have CPVT.[9]

Supplementary report of the Sydney team

6. Upon receipt of Professor Vinuesa's further statement, those assisting the Inquiry provided the Sydney team and Professor Skinner with the material received from Professor Vinuesa. Professors Skinner and Kirk and Dr Buckley provided a short report dated 5 July 2019 ("the Supplementary Sydney report").[10]

7. In the Supplementary Sydney report it was noted that although the June 2019 paper referred to a different gene (CALM3) than the one found in Sarah, Laura and Ms Folbigg (CALM2), the findings are relevant because the three CALM genes code an identical protein.[11] Professors Skinner and Kirk and Dr Buckley considered the information is relevant to interpretation of the potential clinical significance of the p.Gly114Arg variant, "increasing the likelihood that this variant is pathogenic and that it might be relevant to the deaths of Sarah and Laura".[12] They therefore considered afresh the classification of this variant in light of the new information available in the June 2019 paper.[13]

8. The Supplementary Sydney report considered that in light of the new information, the ACMG Guidelines criteria PP2 (missense variant in a gene that has a low rate of benign missense variation), and PM5 (novel missense change at an amino acid residue where a different missense change determined to be pathogenic has been seen before), should be applied.[14] As a result, if the clinical information was not taken into account, the addition of PM5 would mean that the variant would now be classified as likely pathogenic.[15]

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6 Exhibit BT, Letter from Professor Peter Schwartz to Professor Carola Vinuesa (20 June 2019) [4].

7 Exhibit BT, Letter from Professor Peter Schwartz to Professor Carola Vinuesa (20 June 2019) [6].

8 Exhibit BH, Further cardiac testing of Kathleen Folbigg (18 April 2019); Exhibit BL, Report of Associate Professor Hariharan Raju (18 April 2019).

9 Exhibit BK, Letter from Professor Jonathan Skinner to the Inquiry (30 April 2019) p 1; Exhibit BJ, Further report of Professor Jonathan Skinner (24 April 2019) pp 3-4.

10 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019).

11 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [2.1].

12 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [4].

13 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [4].

14 Exhibit AC, Genetics tender bundle, ACMG Guidelines; Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [1.2], [2.5].

15 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [2.5].

9. However, the Supplementary Sydney report considered that the clinical information is essential to the interpretation of the significance of the variant.[16] The Supplementary Sydney report observed, by reference to the clinical tests reviewed by two specialists in inherited cardiac conditions, that the clinical information was in conflict with the genetic evidence.[17] The Supplementary Sydney report concluded that the fact Ms Folbigg is alive (at age 52), has never had a cardiac arrest, and produced exercise test results within normal limits are strongly against a hypothesis of concealed CPVT.[18]

10. The Supplementary Sydney report recognised it would be "theoretically possible" to have mosaicism in one tissue which is not reflected in another, and that this could explain the absence or greatly attenuated cardiac phenotype in Ms Folbigg.[19] However, the Supplementary Sydney report concluded that based on the available genetic data, this was very unlikely and that testing of tissue was unlikely to be of value.[20] The Supplementary Sydney report also said that there are no functional studies for this particular protein validated to clinical standards.[21]

11. The Supplementary Sydney report identified four possible interpretations of the information regarding the variant:

    a. the variant could be pathogenic and the sole cause of the deaths of Sarah and Laura;[22]

    b. the variant could be pathogenic and related to, but not the sole cause, of the deaths of Sarah and Laura (such as if the children had experienced an asphyxial event which may or may not normally have been sufficient to cause their deaths, but which, through adrenergic stimulation induced a cardiac arrhythmia that would not otherwise have happened and led to their deaths);[23]

    c. the variant could be pathogenic but unrelated to the deaths of Sarah and Laura;[24] or

    d. the variant could be benign.[25]

12. The Supplementary Sydney report concluded that following application of the ACMG Guidelines, the CALM2 variant in Ms Folbigg, Sarah and Laura remains classified as a variant of uncertain significance, due to the conflict between the clinical and genetic evidence.[26]

13. The Supplementary Sydney report noted uncertainty as to which of the possibilities identified above was most likely. The Supplementary Sydney report did observe, however, that:

    a. either of the first two possibilities would require at least two different causes of death of the Folbigg children, given the absence of the CALM2 variant in Caleb and Patrick; and

    b. the first possibility would require "an exceptional clinical scenario" which is "outside the range that has previously been reported in association with variants in this group of genes".[27]

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16 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [2.8].
17 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [4].
18 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [1.6], [2.4].
19 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [2.4.2].
20 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [2.4.2], [4].
21 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [2.6].
22 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [3.1].
23 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [3.2].
24 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [3.3].
25 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [3.4].
26 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [4].
27 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [4].

Submissions of counsel assisting

14. Upon receipt of the Supplementary Sydney report, counsel assisting provided further written submissions on 8 July 2019.[28]

15. Counsel assisting emphasised that while the information in the June 2019 paper regarding the CALM2 variant changed the criteria applied by the Sydney team in the classification process, it has not changed the results of the genetic testing conducted by the Inquiry, or the conclusions reached by the Sydney team and Professor Skinner in their evidence.[29]

16. In counsel assisting's submission, the ultimate conclusion remains that CALM2 is a variant of uncertain significance in light of the relevant clinical information.[30] Counsel assisting submitted I should accept the opinions of Professors Skinner and Kirk and Dr Buckley regarding the importance of taking into account the relevant clinical information, particularly in circumstances where the genetic information is in conflict with that clinical information, and that there is no further testing that can be done that would be of value.[31]

17. By contrast, Professor Schwartz's assessment was formed in the absence of any of the clinical information in relation to Ms Folbigg or the Folbigg children, and in particular without the benefit of the exercise test conducted on Ms Folbigg in April 2019. The view he expressed was based on incomplete information.

18. Counsel assisting also drew attention to the differences between the clinical phenotype of the family referenced in the June 2019 paper and the Folbigg family.[32]

19. In the submission of counsel assisting, I should be satisfied that there has been no change in the results of the genetic testing conducted by the Inquiry and no variant has been identified in the Folbigg children during that testing process as being pathogenic or likely pathogenic. Counsel assisting submitted that it follows that it remains the case that there is no reasonable possibility that the death of any of the Folbigg children or Patrick's ALTE was caused by a recognised genetic variant.[33]

Letter in response to the Supplementary Sydney report

20. Ms Folbigg's representatives were given an opportunity to provide submissions in reply by 12 July 2019. On 12 July 2019 the Inquiry instead received a further expert report in the form of a letter, together with further written submissions on behalf of Ms Folbigg.

21. The letter was dated 11 July 2019 and recorded it was:

> *drafted by Professor Carola Vinuesa and Dr Todor Arsov and reviewed, added to, and endorsed by Professors Peter Schwartz, Matthew Cook and Michael Overgaard.*[34]

22. Professor Overgaard is a professor of protein science and Head of Department at the Department of Biochemistry and Bioscience, Aalborg University in Denmark.[35] Together with Professor Schwartz and others, he was an author of the June 2019 paper which Professor Vinuesa drew to the Inquiry's attention, identifying a pathogenic CALM3 variant manifesting in a family with a non-affected parent carrier and two child deaths.

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28 Further submissions of counsel assisting the Inquiry (8 July 2019).
29 Further submissions of counsel assisting the Inquiry (8 July 2019) [14].
30 Further submissions of counsel assisting the Inquiry (8 July 2019) [15].
31 Further submissions of counsel assisting the Inquiry (8 July 2019) [17].
32 Further submissions of counsel assisting the Inquiry (8 July 2019) [19].
33 Further submissions of counsel assisting the Inquiry (8 July 2019) [20].
34 Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [7.1].
35 Exhibit BW, Curriculum Vitae of Professor Michael Overgaard.

23. The letter recorded that:

> *In reviewing and endorsing this response, Professors Schwartz and Toft Overgaard were provided with the following exhibits in addition to the Supplementary Report by the Sydney team:*
>
> *a) AH – ECG of Kathleen Folbigg dated 17 May 2011*
>
> *b) AE – Pedigree of Kathleen Folbigg (family) dated 8 October 2018*
>
> *c) AF – Joint report of Canberra team dated 29 March 2019*
>
> *d) Z – Joint report of Sydney genetics team dated 29 March 2019*
>
> *e) Y – Expert report of Professor Jon Skinner dated 31 March 2019*
>
> *f) AX – Written response to joint expert report of Vinuesa and Cook dated 9 April 2019*
>
> *g) AY – Written reply to response of Kirk and Buckley dated 12 April 2019*
>
> *h) BL – Letter from Dr Hariharan Raju dated 18 April 2019*
>
> *i) BK – Letter from Professor Jon Skinner dated 30 April 2019.*[36]

24. The letter opined that the Supplementary Sydney report resiled from a classification of the CALM2 variant identified in Ms Folbigg, Sarah and Laura as likely pathogenic according to the ACMG criteria "based on a series of assertions relating to the clinical presentation of Kathleen Folbigg and her children", which the letter authors considered to be incorrect.[37]

25. In respect of Ms Folbigg the letter opined there is:

> *insufficient evidence that Ms Folbigg is unaffected by the G114R variant at this stage. Regardless, a finding that she is "healthy" is not a basis for overriding the ACMG criteria which support "likely pathogenic" interpretation of this variant.*[38]

26. The letter's authors concluded, by reference to the Schwartz diagnostic criteria for long QT syndrome and Professor Overgaard's opinion that the CALM2 variant identified in Ms Folbigg and Sarah and Laura "is likely to cause a cardiac condition amidst the CPVT/LQTS/IVF spectrum",[39] that:

> *there remain uncertainties regarding the clinical phenotype of Kathleen Folbigg. In addition, even if she lacks clinical manifestations there are precedents that mean this cannot exclude pathogenicity of the variant in her offspring.*[40]

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[36] Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [7.2].

[37] Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [6.1].

[38] Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [6.2].

[39] Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [4.4].

[40] Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [4.5].

27. The letter referred to mosaicism, digenic causes, and environmental triggers as explanations for incomplete penetrance in an apparently health carrier of a mutation, which can be lethal in a related person.[41] The letter considered that these explanations are "widely accepted by modern genetics", yet "remarkably still not captured by the ACMG Guidelines".[42]

28. In respect of the children the letter opined:

> *in the absence of conclusive phenotypic information about the children, there remains no factual basis for dismissing the significance of the CALM2 variant based on segregation analysis.*[43]

29. The letter particularised that the authors did not consider death a phenotype, and that the phenotype that caused death in each child remains ambiguous. This was said to be supported "by the relevant and inconsistent clinical and pathological analyses of the Folbigg children", though the analyses relied upon were not particularised.[44]

30. In responding to the Supplementary Sydney report's reference to an "exceptional clinical scenario" being required for the variant to be pathogenic, the letter firstly suggested the Sydney report was wrong to assert that variants in calmodulin have still not been reported as a cause of sudden infant death syndrome, being sudden death of an infant during sleep. The letter stated:

> *the facts recorded in the Registry of Calmodulinopathies (Crotti et al. Eur Heart J 2019) are that there are five cases of sudden death/cardiac arrest while asleep caused by CALM 1/2/3 mutation.*[45]

31. The letter did not, however, suggest there were any reported case of sudden death or cardiac arrest while asleep in an *infant.*

32. The letter went on to suggest, secondly, that:

> *whether the clinical scenario is "exceptional" or not, has no bearing on the interpretation of the pathogenicity of the variant. Even if this variant had not met the criteria to be classified as "likely pathogenic" per the ACMG criteria, we would maintain our position that applying the conservative and rigid ACMG criteria on the research end of the clinical/diagnostic genetics spectrum is not appropriate. This is also the view of the creators of the ACMG criteria, as outlined in the published ACMG Guidelines… Notwithstanding the stringency of the ACMG criteria the G14R variant has now been classified by both teams as likely pathogenic when adhering to the criteria.*[46]

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41 Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [3.2]-[3.5].

42 Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [3.2].

43 Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [6.4].

44 Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [2.2].

45 Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [5].

46 Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [6.6].

33. The letter noted that:

> *there is still general agreement that we are dealing with an exceptional clinical scenario. Rare genetic variants are by definition responsible for exceptional clinical scenarios.*[47]

34. The letter concluded by opining that:

> *based on the available facts we cannot reasonably exclude, and we think it is likely, that the two female Folbigg children died as a result of the CALM2 G114R variant, while the two male children died from different causes that could also be genetic.*[48]

35. This opinion appeared to rely on Professor Schwartz's and Professor Overgaard's work establishing that variable clinical expressivity including variable age of onset, depending on the precise amino acid substitution, "is very much the case for calmodulin mutations".[49] The letter opined that the Supplementary Sydney report made an assumption in concluding "the variant is of a type that (if pathogenic) would not usually be expected to cause death at such a young age", whereas the current uncertainty as to expressivity (and thus pathogenicity) should have been acknowledged.[50]

36. This opinion also appeared to rely on the letter's authors' assessment that:

> *We are not aware of any facts to refute that the two female Folbigg children died as a result of the CALM2 G114R variant, while the two male children died from different causes, whether genetic or otherwise.*[51]

Ms Folbigg's submissions

37. The further submissions of Ms Folbigg addressed the Supplementary Sydney report and the letter in response.[52]

38. The submissions suggested that upon application of the ACMG Guidelines, the addition of the PM5 criteria "provides a likely explanation for the sudden death of two of the children", such that I would have a reasonable doubt about the guilt of Ms Folbigg with respect to Sarah's and Laura's deaths.[53]

39. The submissions suggested that the Supplementary Sydney report gave clinical information "undue weight outside of the ACMG criteria" to conclude the variant was not likely pathogenic, without providing a reason for their opinion justifying departure from the ACMG Guidelines.[54] On that basis it was submitted that I would reject the Supplementary Sydney report, or give it no weight.[55]

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47 Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [6.5].

48 Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [6.7].

49 Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [2.5].

50 Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [2.5].

51 Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [2.3].

52 Further submissions of Ms Folbigg (12 July 2019).

53 Further submissions of Ms Folbigg (12 July 2019) [5]-[6].

54 Further submissions of Ms Folbigg (12 July 2019) [7]-[9].

55 Further submissions of Ms Folbigg (12 July 2019) [10].

40. The submissions were critical of what were said to be "unqualified views" expressed by Professor Skinner in his oral evidence and in the Supplementary Sydney report, in relation to the lack of any report of a calmodulin variant presentation in a SIDS – sudden infant death while sleeping – case. This was said to be incorrect because there are reported deaths during sleep caused by calmodulin variants.[56] The submissions went on to suggest that such reports included sudden death of infants,[57] though this was inconsistent with the limited reference in the responsive letter to sudden deaths during sleep without reference to age.

Findings regarding further information about the CALM2 variant

41. It is necessary to deal at some length with the further information received regarding the CALM2 variant since the close of the evidence and submissions. This information included opinions expressed by Professors Schwartz and Overgaard who were not called to give evidence before the Inquiry due to the timing of publication of that research. Those opinions were not in the form of sworn evidence and were not tested by cross-examination.

42. On the basis of the evidence received during the hearings, in the Report I found in respect of the CALM2 variant found in Ms Folbigg, Sarah and Laura:

> *I prefer the expertise and evidence of Professors Skinner and Kirk and Dr Buckley* [that the variant was classified as a variant of uncertain significance rather than likely pathogenic]. *Having regard to the conflict between the genetic and clinical information in respect of Ms Folbigg's cardiac presentation and in respect of the manner of the children's deaths, I find there is no reasonable possibility that this variant caused the death of Sarah or Laura.*[58]

43. The ACMG Guidelines call for the application of clinical information in the assessment of pathogenicity. The difference in classification between the Sydney and Canberra teams appears to arise from different approaches to, and judgments about, the clinical information available to them.

44. The Sydney team applied a clinical standard, allowing only for known disease associations with the particular phenotype of the children (sudden death in infancy during sleep) and Ms Folbigg, which they did not consider to be uncertain.

45. The Canberra team applied a research-based standard, allowing for known disease associations with a broader phenotype, noting they did not consider the phenotype of either the children or Ms Folbigg to be certain.

46. I accept that Professors Schwartz is an expert in his field as concern the study of calmodulin genetic variants. I accept, based on this expertise, that there is variable clinical expressivity including variable age of onset for calmodulin mutations, and there is current uncertainty and ongoing learning as to such expressivity.

47. For the purpose of considering whether the Inquiry should be reopened to receive, in the format of hearings, further evidence about the CALM2 variant, I am prepared to accept the opinion in the letter from Professor Vinuesa and others that the CALM2 variant identified in Ms Folbigg and Sarah and Laura "is likely to cause a cardiac condition amidst the CPVT/LQTS/IVF spectrum".[59]

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56 Further submissions of Ms Folbigg (12 July 2019) [12]-[13].

57 Further submissions of Ms Folbigg (12 July 2019) [14]-[15].

58 See Chapter 7, [167].

59 Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [4.4].

48. I note in this regard that the Supplementary Sydney report identified four possible interpretations of the information regarding the variant, including that it could be pathogenic and the sole cause of their deaths, or that it could be pathogenic and related to but not the sole cause of their deaths, or that it could be pathogenic but unrelated to their deaths, or that it could be benign. These possibilities each appear to me to be consistent with the opinion in the letter from Professor Vinuesa and others as to likelihood of the variant causing a relevant cardiac condition (as opposed to their deaths as they occurred).

49. In considering the expanded understanding of CALM genetic variants as it relates to the deaths of Sarah and Laura, I note the following findings of the June 2019 paper:

    a. the International Calmodulinopathy Registry includes only 74 patients;

    b. the clinical hallmarks of pathogenic calmodulin variants (primarily CPVT and LQTS) are "extremely rare and severe";

    c. the trigger for arrhythmic events in 81 per cent of the 74 patients was adrenergic stimuli, and mostly (62 per cent) associated with exertion;

    d. sudden cardiac death occurred in only 27 per cent of the 74 patients;

    e. only 13.5 per cent or 10 of the 74 patients were asymptomatic for any cardiac event;

    f. the median age at a first event was four years old;

    g. all CALM-CPVT patients were symptomatic with median age of onset of 6.0 years;

    h. 78 per cent of CALM-LQTS patients had life-threatening arrhythmias with median age of onset of 1.5 years; and

    i. the 10 year cumulative mortality rate for the 74 patients was 27 per cent.[60]

50. These findings demonstrate not only the extreme rarity of pathogenic calmodulin variants, but the even more extreme rarity of sudden cardiac deaths during sleep caused by such variants in previously asymptomatic carriers.

51. I note Sarah and Laura each died aged under two years, during a period of sleep and had not been symptomatic for any cardiac condition.

52. The conclusion by Professor Skinner and the Sydney team as to classification as a variant of uncertain significance turned on the conflict between the genetic and clinical information not only as they assessed it in relation to Ms Folbigg, but more importantly in my view, as was uncontroversial in respect of the manner of Sarah's and Laura's deaths.

53. It remains the case per Professor Skinner's evidence, that there is no reported case of a death of such nature – asymptomatic sudden cardiac death in infancy during a sleep period – being associated with a calmodulin variant. If so associated, Sarah and Laura's deaths would be the first and second reported cases of their kind. The letter prepared by Professor Vinuesa agreed with the Supplementary Sydney report that this would be an "exceptional clinical scenario".

54. I am satisfied that no further available genetic related testing would materially alter the effect of the available information.

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[60] Exhibit BU, Lia Crotti et al 'Calmodulin Mutations and Life-Threatening Cardiac Arrhythmias: Insights from the International Calmodulinopathy Registry' (2019) *European Heart Journal* (advance) 2, 4-5, 9.

55. I note the views of Professor Skinner and the Sydney team of the importance of the clinical information in relation to Ms Folbigg, Sarah and Laura and their conclusion that the CALM2 variant remains classified as a Variant of Uncertain Significance (Class 111). Professor Vinuesa disagrees and says it is likely pathogenic. This conflict in views and the debate it generates will no doubt continue, however, it is not necessary for it to be resolved in order for me to decide if the Inquiry should be re-opened.

56. The report of Professor Vinuesa states "we cannot reasonably exclude, and we think it is likely" the two children died as a result of the CALM2 G114R variant.[61] I take that to mean a real or plausible possibility in the context of this report and I agree that as a result of the June 2019 paper, it is now plausible that Sarah and Laura may have had a cardiac condition and that raises a possibility it caused their deaths. That, of course, is on the basis of considering the genetic evidence in isolation. However, in determining cause of death the Inquiry must consider that evidence in the context of the whole of the evidence before the Inquiry, and in doing that, the fact that all the experts agree this genetic explanation is an "exceptional clinical scenario" is a relevant matter to consider.[62]

57. This understanding creates for consideration a possible cause of death in the same way as the evidence of the pathologists' evidence created a possible cause of death for Laura, namely myocarditis. Such possibilities should be evaluated in the same way, namely together with all the other evidence in the Inquiry.

58. In relation to Sarah, taking into account additionally the diary entries made by Ms Folbigg and her lies and obfuscation, the evidence of Mr Folbigg indicating Ms Folbigg's fraught relationship with Sarah, and the tendency and coincidence evidence, I remain of the view that the only conclusion reasonably open is that Ms Folbigg smothered Sarah.

59. In relation to Laura, taking into account additionally the rarity of myocarditis as a cause of death in children of Laura's age, the non-medical evidence including the diary entries made by Ms Folbigg and her lies and obfuscation, the evidence of Mr Folbigg indicating the difficulties Ms Folbigg was having with Laura, and the tendency and coincidence evidence, I remain of the view that the only conclusion reasonably open is that Ms Folbigg smothered Laura.

60. Even on the basis of accepting the opinion of Professor Vinuesa that it is now plausible that Sarah and Laura Folbigg may have had a cardiac condition, and that that raises a possibility it caused their deaths, I do not consider the Inquiry should be re-opened for the purpose of holding further hearings about the CALM2 variant identified in Sarah and Laura. For the reasons above, the further information received since the close of the evidence and submissions does not raise in my mind any reasonable doubt of Ms Folbigg's guilt of these offences.

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61 Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [6.7].

62 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (5 July 2019) [3], [4]; Exhibit BW, Response from Professors Carola Vinuesa, Peter Schwartz, Matthew Cook, Michael Overgaard and Dr Todor Arsov to Supplementary report of Professors Jonathan Skinner, Edwin Kirk and Dr Michael Buckley (11 July 2019) [6.5].

Report of the Inquiry into the convictions of Kathleen Megan Folbigg

July 2019

Annexures

Annexure A: The Direction

Direction pursuant to section 77(1)(a) of the *Crimes (Appeal and Review) Act 2001*

WHEREAS it appears that there is a doubt or question as to part of the evidence in the proceedings leading to the conviction of Kathleen Megan Folbigg on 21 May 2003 of the following offences:

1. the manslaughter of Caleb Folbigg on 20 February 1989;
2. maliciously inflicting grievous bodily harm upon Patrick Folbigg on 18 October 1990, with intent to do grievous bodily harm;
3. the murder of Patrick Folbigg on 13 February 1991;
4. the murder of Sarah Folbigg on 30 August 1993; and
5. the murder of Laura Folbigg on 1 March 1999;

AND WHEREAS that doubt or question concerns evidence as to the incidence of reported deaths of three or more infants in the same family attributed to unidentified natural causes;

PURSUANT to section 77(1)(a) of the *Crimes (Appeal and Review) Act 2001*, I direct that an inquiry be conducted into the said convictions in accordance with the provision of Part 7 of the said Act; and

PURSUANT to section 81(1)(a) of the said Act I hereby appoint the Honourable Reginald Oliver Blanch AM QC, formerly a judicial officer within the meaning of the *Judicial Officers Act 1986*, to conduct an inquiry into the said convictions, having particular regard to the said evidence, with the powers and authorities conferred on a commissioner by Division 2 of Part 2 of the *Royal Commissions Act 1923* (except for section 17).

SIGNED AT SYDNEY this 22nd day of August 2018

General David Hurley AC DSC (Ret'd)

His Excellency, Governor of New South Wales

Annexure B: Advertisements

The Newcastle Herald (6 October 2018)

Public Notices

NSW GOVERNMENT

Inquiry

Directed under section 77(1)(a) of the *Crimes (Appeal and Review) Act 2001*

Kathleen Megan Folbigg

Pursuant to section 77(1)(a) of the *Crimes (Appeal and Review) Act 2001* his Excellency, the Governor of New South Wales, has directed that an inquiry be held into the conviction of Kathleen Megan Folbigg on 21 May 2003 of offences involving the deaths of her four children, and malicious infliction of grievous bodily harm upon one of the children, between 20 February 1989 and 1 March 1999.

The Inquiry has been directed due to a doubt or question concerning evidence in the case as to the incidence of reported deaths of three or more infants in the same family attributed to unidentified natural causes.

The Honourable Reginald Oliver Blanch AM QC, a former judicial officer within the meaning of the *Judicial Officers Act 1986*, has been appointed to conduct the Inquiry, with the powers and authorities conferred on a commissioner by Division 2 of Part 2 of the *Royal Commissions Act 1923* (except for section 17).

Any person who considers that he or she has information which may assist the Inquiry should contact:

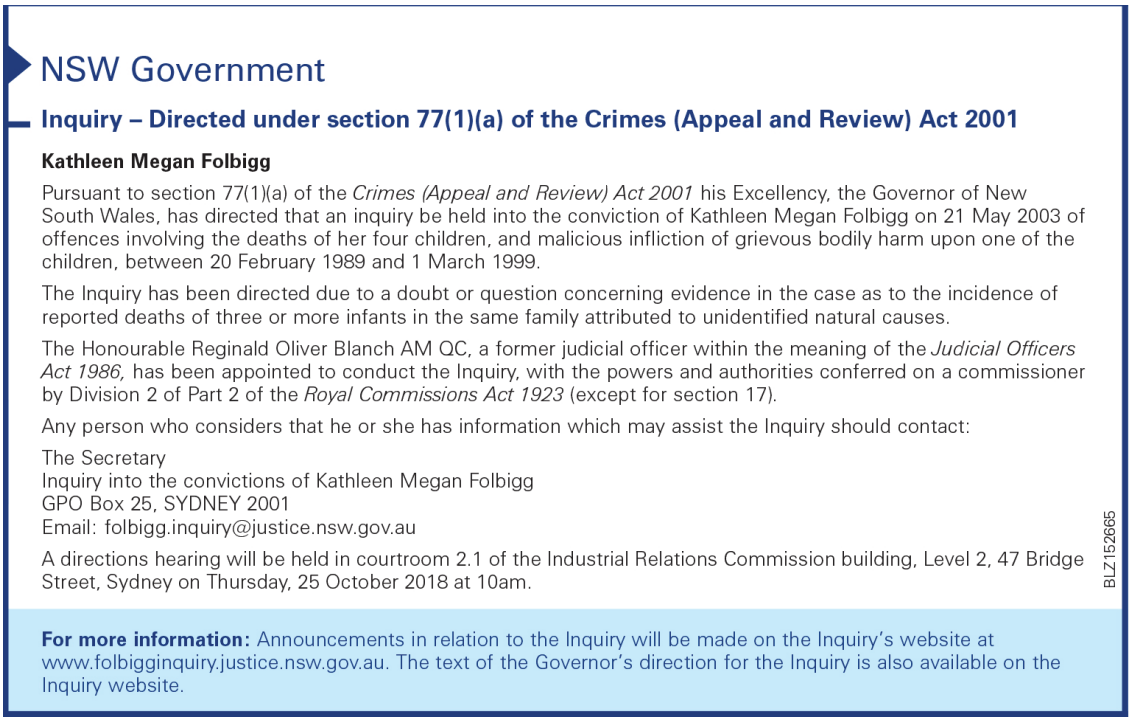
The Secretary
Inquiry into the convictions of Kathleen Megan Folbigg
GPO Box 25, SYDNEY 2001
Email: folbigg.inquiry@justice.nsw.gov.au

A directions hearing will be held in courtroom 2.1 of the Industrial Relations Commission building, Level 2, 47 Bridge Street, Sydney on Thursday, 25 October 2018 at 10am.

BLZ152665

Announcements in relation to the Inquiry will be made on the Inquiry's website at www.folbigginquiry.justice.nsw.gov.au. The text of the Governor's direction for the Inquiry is also available on the Inquiry website.

The Sydney Morning Herald (10 October 2018)



NSW Government

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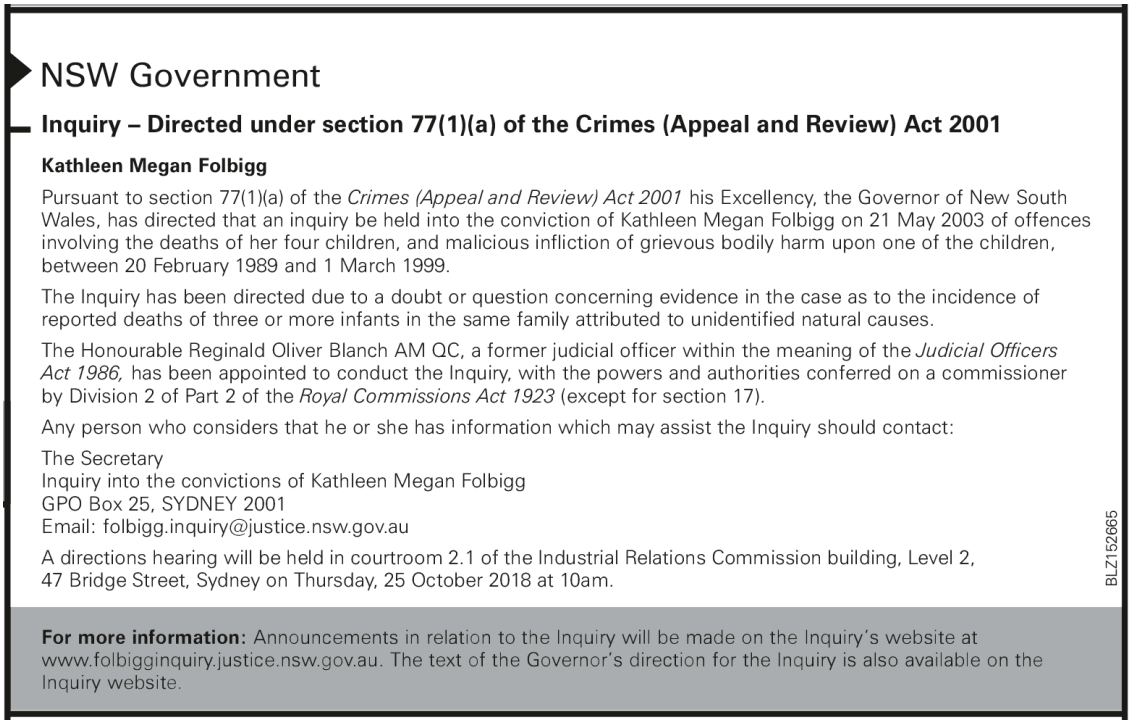
The Daily Telegraph (6 October 2018)



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The Australian Medical Journal (15 November 2018)

NSW GOVERNMENT

Inquiry

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The Inquiry has been directed due to a doubt or question concerning evidence in the case as to the incidence of reported deaths of three or more infants in the same family attributed to unidentified natural causes.

The Honourable Reginald Oliver Blanch AM QC, a former judicial officer within the meaning of the *Judicial Officers Act 1986*, has been appointed to conduct the Inquiry, with the powers and authorities conferred on a commissioner by Division 2 of Part 2 of the *Royal Commissions Act 1923* (except for section 17).

Any person who considers that he or she has information which may assist the Inquiry should contact:

The Secretary
Inquiry into the convictions of Kathleen Megan Folbigg
GPO Box 25, SYDNEY 2001
Email: folbigg.inquiry@justice.nsw.gov.au

BLZ152665

Announcements in relation to the Inquiry will be made on the Inquiry's website at www.folbigginquiry.justice.nsw.gov.au.

Annexure C: The experts at trial and in the Inquiry

Trial

Dr Brian Bailey

1. Dr Brian Bailey was a consultant cardiologist with a special interest in sudden unexpected death due to long QT syndrome.[1] He was provided Laura's cardiac rhythm tracing records, her post-mortem report and other medical documents by the investigating police. He gave evidence at trial for the Crown in relation to Laura's agonal rhythm and myocarditis.[2]

Dr Susan Beal AM

2. Dr Susan Beal was a paediatrician at the Women's and Children's Hospital in Adelaide. She had studied SIDS for over 35 years and published widely in that domain, including an article on the recurrence of SIDS in a family.[3] She was also an epidemiologist, studying patterns of disease.[4]

3. Dr Beal gave oral evidence at trial for the Crown in relation to each of the four children and prepared a statement dated 8 December 1999.[5]

Professor Peter Berry

4. Professor Peter Berry was a Consultant Paediatric Pathologist (recently retired at the time of trial) at the Bristol Royal Hospital for Sick Children, and Professor of Paediatric Pathology at the University of Bristol.[6] He was co-editor of a book describing the most recent study on SIDS at that time and had a particular interest in the investigation and causes of sudden unexpected death in infancy.[7]

5. Professor Berry was briefed by the prosecution at trial with the children's medical records, microscopic tissue slides, and post-mortem reports. He prepared two reports dated November 2000 and 29 April 2003 and gave oral evidence at trial for the Crown.[8]

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1 5 May 2003 T1098.34.
2 5 May 2003 T1098.39-1099.5.
3 28 April 2003 T974.27-57.
4 24 April 2003 T943.54-56; 28 April 2003 T975.19-976.3.
5 Exhibit H, Forensic pathology tender bundle, Statement of Dr Susan Beal (8 December 1999).
6 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Berry (November 2000).
7 1 May 2003 T1054.15-1055.8.
8 Exhibit H, Forensic pathology tender bundle, Report of Professor Berry (November 2000) and Further report of Professor Berry (29 April 2003).

Professor Roger Byard AO PSM

6. Professor Roger Byard is a specialist forensic pathologist with expertise in sudden natural death in infancy and early childhood.[9] At the time of trial he was Clinical Professor in the Departments of Pathology and Paediatrics at the University of Adelaide and was employed as a Specialist Forensic Pathologist by the Forensic Science Centre in Adelaide.[10]

7. Professor Byard has published widely including on SUDI and is regarded as a world leader in paediatric forensic pathology. Together with Dr Jhodie Duncan he edited the 2018 publication *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future*, the most contemporary and comprehensive publication in the field at the time of the Inquiry.[11]

8. Professor Byard gave evidence at trial for the defence in relation to all four children and prepared two reports dated 18 October 2002 and 14 April 2003.[12]

Professor Anthony Busuttil

9. Professor Anthony Busuttil was Regius Professor of Forensic Medicine and Clinical Forensic Examiner at the University of Edinburgh. He prepared a report for trial at the request of Ms Folbigg's representatives considering the causes of death of the four children.[13] Professor Busuttil did not give evidence at trial.

Dr Allan David Cala

10. Dr Allan Cala is Senior Staff Specialist forensic pathologist at Newcastle Department of Forensic Medicine and formerly of the NSW Institute of Forensic Medicine at Glebe. At the time of trial he was Head of Pathology of the Forensic Science Service for South Australia.[14] Dr Cala carried out the post-mortem examination of Laura and prepared an interim report dated 1 March 1999 and a final autopsy report dated 13 December 1999.[15] He prepared a statement dated 28 March 2003 and gave evidence at trial for the Crown.[16]

Dr John Cash

11. Dr John Cash was a Visiting Medical Officer at Singleton Hospital at the time of trial.[17] He examined Laura several times, including at 1:00am on 22 June 1998 when she presented with a history of a slight upper respiratory infection for several days and a croupy cough.[18] He prepared a statement dated 9 March 1999 and gave evidence at trial for the Crown.[19]

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9 7 May 2003 T1195.19-1200.28.

10 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) p 2.

11 Exhibit D, Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018).

12 Exhibit H, Forensic pathology tender bundle, Report of Professor Roger Byard (18 October 2002) and Report of Professor Roger Byard (14 April 2003).

13 Exhibit H, Forensic pathology tender bundle, Report of Professor Anthony Busuttil (6 November 2002).

14 15 April 2003 T705.5-12.

15 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Laura (1 March 1999); Final autopsy report of Laura (13 December 1999).

16 Exhibit H, Forensic pathology tender bundle, Statement of Dr Allan Cala (28 March 2003).

17 Exhibit H, Forensic pathology tender bundle, 14 April 2003 T657.1-3; Exhibit H, Forensic pathology tender bundle, Statement of Dr John Cash (9 March 1999) [4]

18 14 April 2003 T657.5-19.

19 Exhibit H, Forensic pathology tender bundle, Statement of Dr John Cash (9 March 1999).

Dr David Cooper

12. Dr David Cooper was the Director of Paediatric Respiratory and Sleep Medicine at the Mater Children's Hospital in Brisbane and Associate Professor at the University of Queensland at the time of trial.[20] Dr Cooper conducted sleep studies on Patrick and Sarah and gave evidence for the Crown at trial in relation to the results.[21] He also gave evidence about infant sleep apnoea and the recurrence of SIDS in families. He prepared a statement dated 6 December 1999.[22]

Dr Royal Cummings

13. Dr Royal Cummings was the pathologist who conducted Caleb's autopsy. He prepared an interim autopsy report dated 20 February 1989 and a final autopsy report dated 9 May 1989.[23] He was deceased at the time of trial so did not give evidence.

Dr Joseph Dezordi

14. Dr Joseph Dezordi was a consultant paediatrician who examined Patrick when he was brought to the Mater Hospital at Newcastle on 18 October 1990 following his ALTE.[24] Dr Dezordi had trained in paediatrics for seven years and was completing advanced training in neonatology at the time of the trial.[25]

15. Dr Dezordi prepared a statement dated 17 March 2000 and gave oral evidence at the trial for the Crown.[26]

Dr David Drucker

16. Dr David Drucker was a Reader in Microbiology and Head of the Oral Microbiology section of University Dental Hospital of Manchester at the time of trial. He holds a PhD and DSc and published a research series on SIDS microbiology, including the discovery of an association between the IL-10 gene and SIDS. He did not give evidence at trial but prepared a report for the defence dated 18 February 2003 which was tendered at the Inquiry.[27]

Dr Virginia Friedman

17. Dr Virginia Friedman was a blood analyst at the Division of Analytical Laboratories of the New South Wales Department of Health at Lidcombe.[28] At the request of investigating police, she prepared a pathology report in relation to the stain found on Laura's pillow, identifying it as human blood.[29] She gave evidence for the Crown at trial.

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20 Exhibit H, Forensic pathology tender bundle, Statement of Dr David Cooper (6 December 1999) p 1; 14 April 2003 T585.34-42.
21 Exhibit H, Forensic pathology tender bundle, Statement of Dr David Cooper (6 December 1999) pp 2-3.
22 Exhibit H, Forensic pathology tender bundle, Statement of Dr David Cooper (6 December 1999).
23 Exhibit H, Forensic pathology tender bundle, Interim post-mortem report of Caleb (20 February 1989) and Final autopsy report of Caleb (9 May 1989).
24 9 April 2003 T446.25-42; Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000) pp 1-2.
25 9 April 2003 T446.8-18.
26 Exhibit H, Forensic pathology tender bundle, Statement of Dr Joseph Dezordi (17 March 2000).
27 Exhibit BM, Letter from Dr David Drucker to Legal Aid NSW (18 February 2003).
28 5 May 2003 T1153.3-6.
29 Exhibit E, trial Exhibit AF, Pathology report of Virginia Friedman (9 November 1989).

Dr Michael Giuffrida

18. Dr Michael Giuffrida is a forensic psychiatrist who prepared a psychiatric report for Ms Folbigg's sentencing proceedings at the request of her solicitor.[30] He did not give oral evidence at her sentencing proceedings.

Dr Richard Hawker

19. Dr Richard Hawker was a consultant paediatric cardiologist at The Children's Hospital, Westmead at the time of trial.[31] He prepared a statement dated 6 March 2003 in relation to Laura's agonal rhythm but did not give evidence at trial.[32]

Professor Peter Herdson

20. Professor Peter Herdson was a consultant forensic pathologist, Professor Emeritus of Pathology at the University of New Zealand, Auckland and Honorary Professor of Pathology at the University of Sydney. He was Director of Pathology at Royal Canberra Hospital when he was engaged by police to provide evidence at Ms Folbigg's trial.[33]

21. Professor Herdson prepared gave a report dated 17 January 2002 and gave oral evidence at trial for the Crown in relation to each of the four children.[34]

Professor John Miller Napier Hilton

22. Professor John Hilton had been Head of the Institute of Forensic Medicine at Glebe for 12 years at the time of trial.[35] He was Clinical Director of the Department of Forensic Medicine at the Sydney Area Health Service, and was Associate Professor of Pathology at the University of Sydney.[36] Professor Hilton continued to broadly consult in forensic medicine up until 2016 when he retired.[37]

23. Professor Hilton conducted the post-mortem examination of Sarah and was present when Dr Cala carried out the post-mortem examination of Laura. He prepared the final autopsy report of Sarah dated 25 November 1993 and gave evidence at trial for the Crown.[38]

Dr Owen Jones

24. Dr Owen Jones was a consultant paediatric cardiologist at the Sydney Children's Hospital.[39] He prepared a report at trial at the request of Ms Folbigg's solicitor and gave evidence in relation to Laura's cause of death and whether the children had any congenital heart abnormalities.[40]

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30 Exhibit BD, Report of Dr Michael Giuffrida (27 August 2003).
31 Exhibit H, Forensic pathology tender bundle, Statement of Dr Richard Hawker (6 March 2003).
32 Exhibit H, Forensic pathology tender bundle, Statement of Dr Richard Hawker (6 March 2003).
33 1 May 2003 T1033.5-24; Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Herdson (17 January 2002) pp 1-2.
34 Exhibit H, Forensic pathology tender bundle, Report of Professor Peter Herdson (17 January 2002).
35 14 April 2003 T615.25-46.
36 14 April 2003 T615.35-38; 24 April 2003 T906.13-16.
37 Transcript of the Inquiry, 19 March 2019 T62.9-25.
38 Exhibit H, Forensic pathology tender bundle, Final autopsy report of Sarah (25 November 1993).
39 8 May 2003 T1260.39-48.
40 Exhibit H, Forensic pathology tender bundle, Report of Dr Owen Jones (15 April 2003).

Dr Paul Innis

25. Dr Paul Innis was Laura's treating general practitioner from 14 August 1998 until February 1999, during which he saw her approximately 13 times.[41] Dr Innis gave a statement dated 15 March 1999 and gave evidence for the Crown at trial.[42]

Dr Alex Kan

26. Dr Alex Kan was visiting Senior Pathologist at the Children's Hospital, Westmead at the time of trial. He examined slides of Patrick's brain tissue and provided a neuropathology report to assist Dr Singh-Khaira in completing Patrick's post-mortem report.[43] He also gave evidence for the Crown at trial.

Dr Man Kit Lai

27. Dr Man Kit Lai was a staff specialist radiologist at the Mater Hospital in Newcastle at the time of trial.[44] Dr Lai prepared the two CT brain scans of Patrick and prepared two reports dated 23 October 1990 and 5 November 1990. He prepared a statement for police but did not give evidence at trial.[45]

Dr Christopher Marley

28. Dr Christopher Marley was a general medical practitioner who saw Ms Folbigg on various dates between 1987 and 1994, and both Patrick and Sarah on several occasions during each of their lifetimes.[46] He prepared a statement dated 9 March 1999 and gave evidence at trial for the Crown.[47]

Dr Janice Ophoven

29. Dr Janice Ophoven was a paediatric forensic pathologist based in Minnesota, USA with substantial experience in the investigation of deaths and injuries in childhood in Canada and the US.[48] She prepared a statement dated 6 October 2000, a report dated 1 December 2001 at the request of investigating police, and a second report dated 27 March 2003 at the request of the prosecution and did not give evidence at trial.[49]

Professor Robert Ouvrier

30. Professor Robert Ouvrier was a paediatric neurologist at the Department of Neurology, the Children's Hospital, Westmead.[50] He prepared a report for the police dated 28 October 2002 based on the children's medical records. In his report he set out his opinion on the cause of Patrick's ALTE and his death. He did not give evidence at trial.[51]

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41 15 April 2003 T665.37-39,T668.51-53; Exhibit H, Forensic pathology tender bundle, Statement of Dr Paul Innis (15 March 1999) [5].
42 Exhibit H, Forensic pathology tender bundle, Statement of Dr Paul Innis (15 March 1999).
43 11 April 2003 T559.32-37; Trial Exhibit AD, Histopathology report of Patrick (24 June 1991).
44 Exhibit H, Forensic pathology tender bundle, Statement of Dr Man Kit Lai (11 February 2000).
45 Exhibit H, Forensic pathology tender bundle, Statement of Dr Man Kit Lai (11 February 2000) [5].
46 Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Marley (9 March 1999) p 2.
47 Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Marley (9 March 1999).
48 Exhibit H, Forensic pathology tender bundle, Statement of Dr Janice Ophoven (6 October 2000) pp 1-2.
49 Exhibit H, Forensic pathology tender bundle, Statement of Dr Janice Ophoven (6 October 2000) and Report of Dr Janice Ophoven (1 December 2001) and Report of Dr Janice Ophoven (27 March 2003).
50 Exhibit H, Forensic pathology tender bundle, Report of Dr Robert Ouvrier (28 October 2002) p 2.
51 Exhibit H, Forensic pathology tender bundle, Report of Dr Robert Ouvrier (28 October 2002) p 2.

Dr Roger Pamphlett

31. Dr Roger Pamphlett was the neuropathologist who examined Sarah's brain on autopsy. He provided a neuropathology report which was annexed to the post-mortem report of Professor John Hilton.[52] He did not give evidence at the trial.

Dr Michael Rodriguez

32. Dr Michael Rodriguez was the neuropathologist who examined Laura's brain on autopsy. He provided a neuropathology report which was annexed to the post-mortem report of Dr Allan Cala.[53] He did not give evidence at the trial.

Dr Christopher Seton

33. Dr Christopher Seton was a sleep and respiratory physician with SIDS expertise, and Staff Specialist in the Sleep Disorders Unit at the (then) New Children's Hospital, Westmead at the time of trial.[54] Mr and Ms Folbigg were referred to him in August 1996 for advice about the potential risk of SIDS after the three previous deaths.[55] He saw Laura during her lifetime and conducted her sleep study.[56] Dr Seton prepared a statement dated 23 November 1999 and gave evidence at trial for the Crown.[57]

Dr Yvonne Skinner

34. Dr Yvonne Skinner was a consultant psychiatrist engaged by the prosecution in advance of Ms Folbigg's trial to prepare a report regarding Ms Folbigg's mental state and the issue of infanticide. She did not assess Ms Folbigg in person and she did not give evidence at trial.

35. Dr Skinner's report was tendered at the Inquiry and she did not give oral evidence at the Inquiry's hearings.[58]

Dr Gurpreet Singh-Khaira

36. Dr Gurpreet Singh-Khaira was a histopathologist with experience conducting post-mortem examinations who, together with Dr Jan Bishop, conducted the post-mortem examination of Patrick on 13 February 1991.[59] Two reports were produced, an interim autopsy report dated 14 February 1991 and a final autopsy report dated 2 September 1991.[60] He also gave evidence at trial for the Crown.

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52 Exhibit H, Forensic pathology tender bundle, Neuropathology report of Sarah (undated).
53 Exhibit H, Forensic pathology tender bundle, Neuropathology report of Laura (13 December 1999).
54 15 April 2003 T690.3-30.
55 Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Seton (23 November 1999) [4]-[6].
56 Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Seton (23 November 199) [7]-[12]; 15 April 2003 T691.45-692.22.
57 Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Seton (23 November 1999).
58 Exhibit BC, Report of Dr Yvonne Skinner (22 January 2003).
59 10 April 2003 T554.15-42.
60 Exhibit H, Forensic pathology tender bundle, Interim autopsy report of Patrick (14 February 1991) and Final autopsy report of Patrick (2 September 1991).

Dr Barry Springthorpe

37. Dr Barry Springthorpe was a consultant paediatrician who established the Child Development Unit in Newcastle in 1976, which had an emphasis on developmental problems, SIDS and child abuse, and also established the Suspected Child Abuse and Neglect Group.[61]

38. Dr Springthorpe saw Caleb twice, first on 2 February 1989 when Caleb was 14 hours old and again on 17 February 1989 when he was two weeks old.[62] He prepared a statement dated 6 December 1999 and gave evidence at trial for the Crown.[63]

Dr Christopher Walker

39. Dr Christopher Walker was the Director of the Emergency Medicine Department at the Mater Hospital in Newcastle when Patrick arrived on 13 February 1991. He pronounced Patrick's death. Dr Walker prepared a statement dated 18 January 2000 and gave evidence at trial for the Crown.[64]

Dr Bruce Westmore

40. Dr Bruce Westmore was a forensic psychiatrist who was engaged by Ms Folbigg's solicitor at trial. He examined her twice before her trial and a third time following conviction and prepared a report for her sentencing proceedings dated 16 June 2003.

41. Dr Westmore's report was tendered in the Inquiry but he did not give oral evidence.[65]

Professor Bridget Wilcken AM

42. Professor Bridget Wilcken was a clinical geneticist at the time of trial, employed at the (then) New Children's Hospital at Westmead as a Senior Staff Physician, and the Director of the New South Wales Newborn Screening Programme and the New South Wales Genetics Service.[66] She was involved in testing and analysing samples in relation to all four Folbigg children in December 1999.[67] She prepared a statement for the prosecutors and gave evidence at trial for the Crown in relation to her findings.

Dr Ian Arthur Wilkinson

43. Dr Ian Wilkinson was a consultant paediatric neurologist who examined Patrick between 18 October 1990 (the date of his ALTE) and 13 February 1991 (date of his death). At the time of trial he was Director of Medicine and a consultant paediatric neurologist at John Hunter Hospital, Newcastle.[68] He conducted a number of investigations into Patrick's encephalopathy during his presentations and prescribed his epilepsy medications.

44. Dr Wilkinson prepared two statements dated 12 March 1999 and a statement dated 8 October 1999 and gave evidence at trial for the Crown.[69]

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61 Exhibit H, Forensic pathology tender bundle, Statement of Dr Barry Springthorpe (6 December 1999) [3].
62 7 April 2003 T265.22-28, T266.6-8, T268.30-40, T269.25-28.
63 Exhibit H, Forensic pathology tender bundle, Statement of Dr Barry Springthorpe (6 December 1999).
64 Exhibit H, Forensic pathology tender bundle, Statement of Dr Christopher Walker (18 January 2000).
65 Exhibit BB, Report of Dr Bruce Westmore (16 June 2003).
66 Exhibit AC, Genetics tender bundle, Statement of Dr Bridget Wilcken (14 January 2000) p 1; 16 April 2003 T817.30-52.
67 Exhibit AC, Genetics tender bundle, Statement of Dr Bridget Wilcken (14 January 2000) p 2.
68 10 April 2003 T507.11-14.
69 Exhibit H, Forensic pathology tender bundle, Statement of Dr Ian Wilkinson (12 March 1999); Statement of Dr Ian Wilkinson (8 October 1999).

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Dr Todor Arsov

45. Dr Todor Arsov is a Visiting Fellow at the Centre for Personalised Immunology at the Australian National University. He holds a PhD in biomedical sciences and a Master of Genetic Counselling.[70] Dr Arsov obtained the sample from Ms Folbigg which Professor Vinuesa analysed,[71] and assisted Professors Vinuesa and Cook prepare the Canberra team's joint report dated 29 March 2019.[72] He also prepared a pedigree of Ms Folbigg and gave evidence in the Inquiry.[73]

Dr Yeliena Baber

46. Dr Yeliena Baber is a forensic pathologist at the Victorian Institute of Forensic Medicine. She examined photomicrographs of sections of the slides of Laura's heart provided by Professor Cordner and prepared a report dated 18 January 2019 at the request of Ms Folbigg.[74]

Professor Cecelia Caroline Blackwell

47. Professor Caroline Blackwell is Conjoint Professor of Immunology and Microbiology at the School of Health, University of Newcastle. She has qualifications in microbiology and a PhD in medical microbiology.[75] She is a medical science researcher, but not a medical practitioner. She gave oral evidence in the Inquiry and prepared four statements.

48. The first statement was prepared in 2004 at the request of Legal Aid on behalf of Ms Folbigg.[76] It was annexed to and formed the draft of the second statement dated 5 March 2019 prepared at the request of those representing Ms Folbigg in the Inquiry.[77] Professor Blackwell provided an undated (third) statement to the Inquiry in March 2019 setting out the relevant medical advances which had been made since 2004.[78] The fourth statement by Professor Blackwell, dated 13 March 2019, concerned Caleb only and was prepared at the request of Ms Folbigg's representatives.[79]

Dr Heinrich Bouwer

49. Dr Heinrich Bouwer is a forensic pathologist at the Victorian Institute of Forensic Medicine. He examined photomicrographs of sections of the slides of Laura's heart provided by Professor Cordner and prepared a report dated 4 January 2019 at the request of Ms Folbigg.[80]

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70 Transcript of the Inquiry, 16 April 2019 T462.19-21, T462.25-26.
71 Exhibit AG, Report of Professor Carola Vinuesa (2 December 2018).
72 Exhibit AF, Joint report of Canberra genetics team (29 March 2019) p 6; Transcript of the Inquiry, 16 April 2019 T463.25-29.
73 Exhibit AE, Pedigree of Kathleen Folbigg (8 October 2018).
74 Exhibit AM, Seven expert reports from forensic pathologists of the VIFM .
75 Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure A.
76 Exhibit T, Report of Professor Caroline Blackwell (5 March 2019) Annexure A.
77 Exhibit T, Report of Professor Caroline Blackwell (5 March 2019).
78 Exhibit U, Further report of Professor Caroline Blackwell (undated).
79 Exhibit V, Further report of Professor Caroline Blackwell (13 March 2019).
80 Exhibit AM, Seven expert reports from forensic pathologists of the VIFM .

Dr Michael Francis Buckley

50. Dr Michael Buckley is a genetic pathologist and Clinical Director of the NSW Health South Eastern Area Laboratory Services at the Prince of Wales Hospital in Sydney. He holds a PhD in the field of molecular genetics.[81] The Inquiry engaged Dr Buckley to advise and assist in the task of arranging for genetic testing to be undertaken. He prepared a report dated 25 February 2019 addressing developments in genetic science from the time of trial to the Inquiry.[82]

51. He was also engaged to interpret the genetic sequencing data. Dr Buckley undertook the interpretation exercise together with Professor Edwin Kirk and Dr Alison Colley and produced a joint report dated 29 March 2019.[83] He prepared a further report together with Professors Skinner and Kirk dated 5 July 2019.[84] Dr Buckley gave oral evidence at the Inquiry.

Dr Michael Burke

52. Dr Michael Burke is a forensic pathologist at the Victorian Institute of Forensic Medicine. He examined photomicrographs of sections of the slides of Laura's heart provided by Professor Cordner and prepared a report dated 30 January 2019 at the request of Ms Folbigg.[85]

Professor Roger Byard AO PSM

53. Professor Byard was engaged by the Inquiry as an expert advisor in relation to the literature in the areas of forensic pathology and SUDI.

Dr Allan David Cala

54. Dr Cala sought and was granted leave to appear before and be represented in the Inquiry. He prepared a report dated 26 November 2018 and a further report dated 13 February 2019.[86] Dr Cala gave evidence in the Inquiry and provided written submissions to the Inquiry in relation to Laura dated 14 June 2019.

Emeritus Professor Robert Llewellyn Clancy AM

55. Professor Robert Clancy is a mucosal immunologist and Emeritus Professor of Pathology at the University of Newcastle.[87] Professor Clancy's field of specialised knowledge, mucosal immunology, concerns immune system responses that occur at mucosal membranes of the intestines, the urogenital tract and the respiratory system. He retired in February 2013.

56. Professor Clancy was engaged by those representing Ms Folbigg to prepare a report on the children's causes of death. He prepared a report dated 13 March 2019, a second report dated 17 March 2019 after reviewing the microbiology reports of Sarah and Laura,[88] and a third dated 27 March 2019 reviewing Professor Horne's report.[89] Professor Clancy gave evidence in the Inquiry's hearings.

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81 Exhibit Z, Joint report of Sydney genetics team, CV of Dr Michael Buckley; Transcript of the Inquiry, 15 April 2019 T372.33-38.
82 Exhibit AB, Report of Dr Michael Buckley (25 February 2019).
83 Exhibit Z, Joint report of Sydney genetics team (29 March 2019).
84 Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley (11 July 2019).
85 Exhibit AM, Seven expert reports from forensic pathologists of the VIFM .
86 Exhibit M, Report of Dr Allan Cala (26 November 2018); Exhibit N, Further report of Dr Allan Cala (13 February 2019).
87 Exhibit W, Report of Professor Robert Clancy (13 March 2019) CV of Professor Robert Clancy.
88 Exhibit W, Further report of Professor Robert Clancy (17 March 2019) p 1.
89 Exhibit AT, Further report of Professor Robert Clancy (27 March 2019) p 1.

Dr Alison Fiona Colley

57. Dr Alison Colley is a clinical geneticist and the Director of Clinical Genetics Services for South West Sydney Local Health District. She has trained in paediatrics as well as clinical genetics. She is a Conjoint Senior Lecturer at the University of New South Wales and a renowned dysmorphologist.[90]

58. She was engaged by the Inquiry to prepare a report summarising her previous involvement with respect to the children and to provide advice on genetic advances since 2003.[91]

59. Dr Colley was also engaged to provide interpretation of the genetic sequencing data along with Professor Edwin Kirk and Dr Michael Buckley and produced a joint report dated 29 March 2019.[92] She also gave evidence at the Inquiry.

Professor Matthew Cook

60. Professor Matthew Cook is Professor of Medicine at the Australian National University, and a practising clinical immunologist at Canberra Hospital. He is also Co-Director of the Centre for Personalised Immunology at the Australian National University, and Medical Director of the Canberra Clinical Genomics laboratory.[93] That laboratory is accredited to conduct bioinformatics analysis of DNA and RNA sequences, such as those produced by whole exome sequencing or whole genome sequencing.[94]

61. Professor Cook undertook the interpretation of th genetic sequencing data exercise together with Professor Vinuesa and produced a joint report dated 29 March 2019.[95] Professors Cook and Vinuesa were assisted in this task by Dr Todor Arsov, a visiting fellow at the Centre for Personalised Immunology. He was unable to give evidence at the Inquiry.

Professor Stephen Moile Cordner AM

62. Professor Stephen Cordner is Professor of Forensic Pathology (International) at Monash University and Head of International Programs at the Victorian Institute of Forensic Medicine. He authored the forensic pathology report annexed to the petition for this Inquiry.[96] This report examined the forensic pathology evidence at trial and the children's causes of death in light of modern understanding in forensic pathology.

63. In addition, Professor Cordner solicited opinions from seven forensic pathologists at the Victorian Institute of Forensic Medicine regarding Laura's myocarditis. He gave oral evidence at the Inquiry hearings.[97]

Dr Michael Diamond

64. Dr Michael Diamond is a psychiatrist who examined Ms Folbigg in custody at the request of Ms Folbigg's legal representatives for the purpose of the Inquiry. He prepared a report dated 16 April 2019 which was tendered at the Inquiry and did not give oral evidence at the hearings.[98]

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90 Exhibit Z, Joint report of Sydney genetics team, CV of Dr Alison Colley; Transcript of the Inquiry, 15 April 2019 T373.39-50.
91 Exhibit AA, Report of Dr Alison Colley (26 November 2018).
92 Exhibit Z, Joint report of Sydney genetics team (29 March 2019).
93 Exhibit AF, Joint report of Canberra genetics team, CV of Professor Matthew Cook; Transcript of the Inquiry, 15 April 2019, T366.19-25.
94 Exhibit AF, Joint report of Canberra genetics team (29 March 2019) p 4.
95 Exhibit AF, Joint report of Canberra genetics team (29 March 2019) p 6; Transcript of the Inquiry, 16 April 2019 T462.19-21, T462.25-26.
96 Exhibit Q, Report of Professor Stephen Cordner (undated).
97 Exhibit R, Letter from Professor Stephen Cordner to the Inquiry (8 March 2019).
98 Exhibit BA, Report of Dr Michael Diamond (16 April 2019).

Professor Johan Duflou

65. Professor Johan Duflou is a consultant forensic pathologist in private practice, part-time Specialist Forensic Pathologist at the Forensic Medicine Centre in Canberra, Clinical Professor of Pathology at the University of Sydney and Conjoint Associate Professor at the National Drug and Alcohol Research Centre, University of New South Wales.

66. He prepared a report dated 13 February 2019 examining the children's causes of death and gave evidence in the Inquiry at the request of Ms Folbigg's representatives.[99]

Professor Dawn Elder

67. Professor Dawn Elder is a consultant paediatrician, Deputy Dean and Head of the Department of Paediatrics at the University of Otago in Wellington.[100] Professor Elder has a PhD in the field of respiratory variability in infants and children and is trained in both neonatal medicine as well as paediatric sleep medicine. She was also co-investigator in the recent New Zealand case control SIDS study.

68. She was engaged by the Inquiry to prepare a report identifying and explaining advances in SIDS research since 2003 that are relevant to the causes of death of any of the children and/or the cause of the ALTE in respect of Patrick.[101] She prepared a report dated 15 February 2019 and gave evidence in the Inquiry.[102]

Associate Professor Michael Collingwood Fahey

69. Associate Professor Michael Fahey is a paediatric neurologist, clinical geneticist, Director of Paediatric Neurology and Head of Neurogenetics at the Monash Children's Hospital in Victoria.[103] He is also a neurologist at the Paediatric Rehabilitation Unit at Monash Children's Hospital and a neurogeneticist at the Neurogenetics Clinic at Royal Melbourne Hospital.[104]

70. He provided the Sydney genetics team with a list of 204 genes associated with childhood neurological disorders for analysis.[105] Associate Professor Fahey prepared a report following the whole genome sequencing of Patrick dated 30 March 2019, taking into account the conditions mentioned in Professor Ryan's report, as well as other relevant genetic variants.[106] He gave evidence in the Inquiry in relation to Patrick's ALTE and death.

Dr Joanna Glengarry

71. Dr Joanna Glengarry is a forensic pathologist at the Victorian Institute of Forensic Medicine. She examined photomicrographs of sections of the slides of Laura's heart provided by Professor Cordner and prepared a report dated 3 January 2019 at the request of Ms Folbigg.[107]

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99 Exhibit L, Report of Professor Johan Duflou (13 February 2019).
100 Transcript of the Inquiry, 18 March 2019 T16.13-15.
101 Exhibit K, Report of Professor Dawn Elder (15 February 2019) p 2.
102 Exhibit K, Report of Professor Dawn Elder (15 February 2019).
103 Transcript of the Inquiry, 17 April 2019 T581.49-50.
104 Transcript of the Inquiry, 17 April 2019 T582.15-17.
105 Transcript of the Inquiry, 17 April 2019 T588.6-9.
106 Exhibit AK, Report of Associate Professor Michael Fahey (30 March 2019).
107 Exhibit AM, Seven expert reports from forensic pathologists of the VIFM .

Dr Michael Giuffrida

72. Dr Giuffrida was engaged by the Inquiry to provide an updated report following receipt of the report of Dr Diamond.[108]

Professor Paul Goldwater

73. Professor Paul Goldwater is Professor of Infectious Diseases, clinical microbiologist and specialist in infectious diseases at the University of Adelaide. He was engaged by Ms Folbigg's representatives and prepared a peer review of the evidence given by Professors Blackwell and Clancy in the Inquiry. He did not give oral evidence, but his report was tendered at the Inquiry.[109]

Professor John Miller Napier Hilton

74. Professor John Hilton sought and was granted leave to appear before and be represented in the Inquiry. He prepared an affidavit dated 13 November 2018 and a report dated 22 January 2019.[110] He gave evidence in the Inquiry and provided written submissions to the Inquiry dated 18 June 2019 relating to Sarah and Laura.

Professor Rosemary Sylvia Claire Horne

75. Professor Rosemary Horne is Professor, Deputy Director and Senior Principal Research Fellow at the Ritchie Centre, Hudson Institute of Medical Research and Department of Paediatrics, Monash University. She holds a PhD on arousal responses from sleep as an underlying mechanism for SIDS.[111] Professor Horne was awarded a DSc for her work in SIDS research and sleep disorders in children, and a Distinguished Researcher Award by the International Society for the Study and Prevention of Infant Death for her research into understanding the mechanisms involved in SIDS.

76. She was engaged by the Inquiry to prepare a report identifying advances in the understanding of SUDI since 2003 relevant to the causes of death of any of the children and/or the ALTE in respect of Patrick, as well as cases of three or more sudden infant or childhood deaths from unexplained causes in the same family. She prepared a report dated 10 February 2019 and gave evidence in the Inquiry.[112]

Professor Edwin Phillip Enfield Kirk

77. Professor Edwin Kirk is a genetic pathologist and clinical geneticist at the NSW Health South Eastern Area Laboratory Services as well as Co-Head of the Centre for Clinical Genetics at the Sydney Children's Hospital. He is Conjoint Professor in the School of Women's and Children's Health, University of New South Wales and Chief Examiner in Genetic Pathology for the Royal College of Pathologists of Australasia. He has trained in paediatrics and clinical genetics, including training in inborn errors of metabolism. He has a PhD in cardiac genetics and was head of the Metabolic Diseases Service at Sydney Children's Hospital for 12 years. He provides a cardiac genetics clinical service which focuses on adults and children with cardiomyopathies and disorders of cardiac rhythm.[113]

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108 Exhibit BR, Report of Dr Michael Giuffrida (10 May 2019).

109 Exhibit AU, Report of Professor Paul Goldwater (29 March 2019).

110 Exhibit O, Report of Professor John Hilton (22 January 2019); Exhibit P, Affidavit of Professor John Hilton (13 November 2018).

111 Transcript of the Inquiry, 18 March 2019 T16.15-18, T18.13-19.19.

112 Exhibit J, Report of Professor Rosemary Horne (10 February 2019).

113 Exhibit Z, Joint report of Sydney genetics team, CV of Professor Edwin Kirk; Transcript of the Inquiry, 15 April 2019 T370.25-28, T370.35-47.

78. Professor Kirk was engaged by the Inquiry to assist in the interpretation of the genetic sequencing data. He undertook the interpretation exercise together with Dr Michael Buckley and Dr Alison Colley and produced a joint report dated 29 March 2019.[114] He prepared a further report together with Professor Skinner and Dr Buckley dated 5 July 2019.[115] Professor Kirk gave evidence in the Inquiry.

Dr Michael Lynch

79. Dr Michael Lynch is a forensic pathologist at the Victorian Institute of Forensic Medicine. He examined photomicrographs of sections of the slides of Laura's heart provided by Professor Cordner and prepared a report dated 8 January 2019 at the request of Ms Folbigg.[116]

Professor Michael Pollanen

80. Professor Michael Pollanen is Chief Forensic Pathologist for Ontario, Canada and Professor in the Department of Laboratory Medicine and Pathology at the University of Toronto. He prepared a peer review of Professor Cordner's report which formed part of the Petition giving rise to the Inquiry.[117] He did not give evidence at the Inquiry.

Associate Professor Hariharan Raju

81. Associate Professor Hariharan Raju is a cardiologist and electrophysiologist at Macquarie University Hospital. He holds a PhD in cardiology and won the Young Investigator Award at the Heart Rhythm Congress in 2013 for his research on the genetics of sudden death.

82. Associate Professor Raju was engaged by Ms Folbigg's representatives to conduct cardiac stress testing of Ms Folbigg. He prepared a report of his findings which was tendered at the Inquiry, but did not give oral evidence.[118]

Professor David Ranson

83. Professor David Ranson is Head of Forensic Services and Deputy Director of the Victorian Institute of Forensic Medicine. He examined photomicrographs of sections of the slides of Laura's heart provided by Professor Cordner and prepared a report dated 31 December 2018 at the request of Ms Folbigg.[119]

Professor William Rawlinson AM

84. Professor William Rawlinson is a Senior Medical Virologist and Director of Virology at South Eastern Sydney and Illawarra Health Service.[120] Professor Rawlinson prepared a short written statement setting out his opinion as to the viability of testing the children's tissue samples for viruses to assess their potential causes of death. His statement was tendered in the Inquiry and he was not called to give evidence. [121]

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114 Exhibit Z, Joint report of Sydney genetics team (29 March 2019).

115 Exhibit AX, Response from Professor Kirk and Dr Buckley to joint report of Canberra genetics team (9 April 2019); Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley dated 5 July 2019.

116 Exhibit AM, Seven expert reports from forensic pathologists of the VIFM .

117 Exhibit C, Report of Professor Michael Pollanen (1 June 2015).

118 Exhibit BL, Report of Associate Professor Hariharan Raju (18 April 2019).

119 Exhibit AM, Seven expert reports from forensic pathologists of the VIFM .

120 Exhibit X, Statement of Professor William Rawlinson (undated) CV of Professor William Rawlinson.

121 Exhibit X, Statement of Professor William Rawlinson (undated).

Professor Monique Ryan

85. Professor Monique Ryan was one of two paediatric neurologists who gave evidence in the Inquiry in relation to Patrick's ALTE and death. Professor Ryan is Professor of Paediatric Neurology and Director of Neurology at the Royal Children's Hospital in Victoria.[122] Professor Ryan was briefed by those representing Ms Folbigg and prepared a report dated 15 March 2019 concerning Patrick's encephalopathy and cause of death.[123]

Professor Peter Schwartz

86. Professor Peter Schwartz is Professor of Cardiology and Director of the Centre for Cardiac Arrhythmias of Genetic Origin, Instituto Auxologico Italiano in Milan, Italy. He is a world leader in genetic disorders leading to sudden cardiac death in infants and the young.

87. Professor Schwartz considered the pathogenicity of the CALM2 variant found in Sarah, Laura and Kathleen Folbigg in light of the findings in his June 2019 paper at the request of Professor Vinuesa. His letter to Professor Vinuesa dated 20 June 2019 outlining his views and his paper were tendered in the Inquiry.[124] Professor Schwartz also reviewed the supplementary report of Professor Vinuesa dated 11 July 2019 which was tendered in the Inquiry. He did not give evidence in the Inquiry (the hearings having concluded in May 2019).

Professor Jonathan Robert Skinner

88. Professor Jonathan Skinner is a consultant paediatric cardiologist and electrophysiologist working as a Senior Medical Officer at Starship Children's Hospital in Auckland, New Zealand. He is Honorary Professor in Paediatrics, Child and Youth Health at the University of Auckland and Chairman of the Genetics Council of the Cardiac Society of Australia and New Zealand.[125]

89. Professor Skinner prepared a report dated 31 March 2019 which addressed cardiac-related variants in the children's and Ms Folbigg's genes as reported by the Canberra genetics team and Sydney genetics team, and the cardiac clinical presentation of each of them.[126] He gave oral evidence at the Inquiry's hearings. He prepared three further reports dated 24 April 2019, and 30 April 2019 and 5 July 2019 (the latter together with Professor Kirk and Dr Buckley).[127]

Professor Maria Carola Garcia de Vinuesa de la Conta

90. Professor Carola Vinuesa is an Australian National Health and Medical Research Council Principal Research Fellow, and Professor of Immunology at the Australian National University. She is also the Chief Scientist at the Canberra Clinical Genomics laboratory of which Professor Matthew Cook is the medical director. Together with Professor Cook, she is also the Co-Director of the Centre for Personalised Immunology.[128]

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122 Transcript of the Inquiry, 17 April 2019 T581.7-8.

123 Exhibit AJ, Report of Professor Monique Ryan (15 March 2019).

124 Exhibit BU, Lia Crotti et al, 'Calmodulin Mutations and Life-Threatening Cardiac Arrhythmias: Insights from the International Calmodulinopathy Registry' (2019) *European Heart Journal* (advance); Exhibit BT, Letter from Professor Peter Schwartz to Professor Carola Vinuesa (20 June 2019).

125 Transcript of the Inquiry, 15 April 2019 T369.8-19.

126 Exhibit Y, Report of Professor Jonathan Skinner (31 March 2019).

127 Exhibit BH, Further cardiac testing of Kathleen Folbigg (18 April 2019); Exhibit BJ, Further report of Professor Jonathan Skinner (24 April 2019); Exhibit BK, Further report of Professor Jonathan Skinner (30 April 2019); Exhibit BV, Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley dated 5 July 2019.

128 Exhibit AF, Joint report of Canberra genetics team, CV of Professor Carola Vinuesa; Transcript of the Inquiry, 16 April 2019 T460.15-18.

91. She prepared a report dated 2 December 2018 at the request of Ms Folbigg's representatives concerning the results of whole exome sequencing undertaken at the Centre for Personalised Immunology.[129]

92. Professor Vinuesa undertook interpretation of the children's genetic sequencing data together with Professor Cook and produced a joint report dated 29 March 2019.[130] Professors Vinuesa and Cook were assisted by Dr Todor Arsov in this task.[131] Professor Vinuesa submitted a supplementary report dated 11 July 2019 assessing the pathogenicity of a variant found in Sarah and Laura in light of a June 2019 publication by Professor Schwartz. She also gave evidence in the Inquiry.

Dr Kathryn Waddell-Smith

93. Dr Kathryn Waddell-Smith is a genetic cardiologist at Flinders Medical Centre, South Australia and holds a PhD in cardiac genetics. Dr Waddell-Smith prepared a report dated 29 March 2019 at the request of Ms Folbigg's representatives addressing a number of questions in relation to cardiac genetic testing.[132] Dr Waddell-Smith's report was tendered and she did not give oral evidence at the Inquiry.

Professor Bridget Wilcken AM

94. Professor Wilcken is now Clinical Professor in Paediatrics and Child Health at the Sydney Medical School, Senior Staff Specialist at Sydney Children's Hospital, Centre for Clinical Genetics and Emeritus Consultant in the Biochemical Genetics and Newborn Screening, Western Sydney Genetics Service at the Children's Hospital, Westmead. Professor Wilcken prepared a report for the Inquiry dated 26 November 2018 but did not give oral evidence.[133]

Professor Noel Woodford

95. Professor Noel Woodford is Director of the Victorian Institute of Forensic Medicine and Chair of the Department of Forensic Medicine at Monash University. He examined photomicrographs of sections of the slides of Laura's heart provided by Professor Cordner and prepared a report dated 18 January 2019 at the request of Ms Folbigg.[134]

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129 Exhibit AG, Report of Professor Carola Vinuesa (2 December 2018).

130 Exhibit AF, Joint report of Canberra genetics team (29 March 2019) p 6; Transcript of the Inquiry, 16 April 2019 T462.19-21, T462.25-26.

131 Exhibit AF, Joint report of Canberra genetics team (29 March 2019) p 6; Transcript of the Inquiry, 16 April 2019 T462.19-21, T462.25-26.

132 Exhibit AV, Report of Dr Kathryn Waddell-Smith (29 March 2019).

133 Exhibit AC, Genetics tender bundle, Report of Professor Bridget Wilcken (26 November 2018).

134 Exhibit AM. Exhibit AM, Seven expert reports from forensic pathologists of the VIFM .

Annexure D: Expert reports and statements to the Inquiry

Expert	Report and/or statement	Exhibit
SIDS/SUDI		
Professor Dawn Elder	Report dated 15 February 2019	K
Professor Rosemary Horne	Report dated 10 February 2019	J
Forensic pathology		
Dr Allan Cala	Report dated 26 November 2018 Further report dated 13 February 2019	M N
Professor Stephen Cordner AM	Report annexed to the Petition for the Inquiry (undated) Letter to the Inquiry dated 8 March 2019	Q R
Professor Johan Duflou	Report dated 13 February 2019	L
Professor John Hilton	Affidavit dated 13 November 2018 Report dated 22 January 2019	P O
Professor Michael Pollanen	Report dated 1 June 2015 which formed part of the Petition for the Inquiry	C

Expert	Report and/or statement	Exhibit
Experts at the Victorian Institute of Forensic Medicine contacted by Professor Stephen Cordner in relation to Laura		
Dr Yeliena Baber	Report dated 18 January 2019	AM
Dr Heinrich Bouwer	Report dated 4 January 2019	
Dr Michael Burke	Report dated 30 January 2019	
Dr Joanna Glengarry	Report dated 3 January 2019	
Dr Matthew Lynch	Report dated 8 January 2019	
Professor David Ranson	Report dated 31 December 2018	
Dr Kathryn Waddell-Smith	Report dated 29 March 2019	
Professor Noel Woodford	Report dated 18 January 2019	
Genetics and cardiology		
Dr Todor Arsov	Pedigree of Ms Folbigg dated 8 October 2018	AE
	Joint report of Canberra genetics team dated 29 March 2019	AF
Dr Michael Buckley	Report dated 25 February 2019	AB
	Joint report of Sydney genetics team dated 29 March 2019	Z
	Response from Professor Kirk and Dr Buckley to joint report of Canberra genetics team dated 9 April 2019	AX
	Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley dated 5 July 2019	BV
Dr Alison Colley	Report dated 26 November 2018	AA
	Joint report of Sydney genetics team dated 29 March 2019	Z
Professor Matthew Cook	Joint report of Canberra genetics team dated 29 March 2019	AF
	Response from Canberra genetics team to response of Professor Kirk and Dr Buckley dated 12 April 2019	AY

Expert	Report and/or statement	Exhibit
Professor Edwin Kirk	Joint report of Sydney genetics team dated 29 March 2019	Z
	Response from Professor Kirk and Dr Buckley to joint report of Canberra genetics team dated 9 April 2019	AX
	Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley dated 5 July 2019	BV
Associate Professor Hariharan Raju	Report dated 18 April 2019	BL
Professor Peter Schwartz	Letter dated 20 June 2019	BT
Professor Carola Vinuesa	Report dated 2 December 2018	AG
	Joint report of Canberra genetics team dated 29 March 2019	AF
	Response from Canberra genetics team to response of Professor Kirk and Dr Buckley dated 12 April 2019	AY
	Response from Professor Carola Vinuesa, Dr Todor Arsov, Professor Peter Schwartz, Professor Matthew Cook and Professor Michael Overgaard to supplementary report of Professor Skinner, Kirk and Dr Buckley dated 11 July 2019	BW
Professor Bridget Wilcken AM	Report dated 26 November 2018	AC, tab 63
Neurology		
Associate Professor Michael Fahey	Report dated 30 March 2019	AK
Professor Monique Ryan	Report dated 15 March 2019	AJ
Microbiology/Immunology/Infection		
Professor Caroline Blackwell	Report dated 5 March 2019 (including report prepared in 2004 and updated in 2006, Annexure A)	T
	Further report (undated)	U
	Further report dated 13 March 2019	V
Professor Robert Clancy AM	Report dated 13 March 2019	W
	Further report dated 17 March 2019	W
	Further report dated 27 March 2019	AT
Professor Paul Goldwater	Report dated 29 March 2019 (redacted)	AU
Professor William Rawlinson AM	Statement (undated)	X

Expert	Report and/or statement	Exhibit
Psychiatry		
Dr Michael Diamond	Report dated 16 April 2019 (redacted)	BA
Dr Michael Giuffrida	Report dated 10 May 2019	BR

Annexure E: List of exhibits tendered in the Inquiry

Exhibit	Document	Date of tender
A	Governor of New South Wales, Direction pursuant to section 77(1)(a) of the *Crimes (Appeal and Review) Act 2001* dated 22 August 2018	25 October 2018, first directions hearing
B	Bundle of judgments: a. *R v Folbigg* [2002] NSWSC 1127 b. *R v Folbigg* [2003] NSWCCA 17 c. *Folbigg v The Queen* [2003] HCATrans 589 d. *R v Folbigg* [2003] NSWSC 895 e. *R v Folbigg* [2005] NSWCCA 23 f. *Folbigg v R* [2005] HCATrans 657 g. *Folbigg v R* [2007] NSWCCA 128 h. *Folbigg v R* [2007] NSWCCA 371	25 October 2018, first directions hearing
C	Expert reports of Professor Stephen Cordner (undated) and Professor Michael Pollanen dated 1 June 2015	12 December 2018, second directions hearing
D	Jhodie R Duncan and Roger W Byard (eds), *SIDS – Sudden Infant and Early Childhood Death: The Past, the Present and the Future* (University of Adelaide Press, 2018)	12 December 2018, second directions hearing
E	Exhibits tendered at the 2003 trial	12 December 2018, second directions hearing
F (Amended)	Complete set of trial transcripts, including voir dire and matters heard in the absence of the jury	12 December 2018, second directions hearing (evidence before the jury only) 11 February 2019, fourth directions hearing (full set tendered)
G	Further set of documents from 2003 trial	18 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
H	Forensic pathology tender bundle	18 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI

Exhibit	Document	Date of tender
J	Expert report of Professor Rosemary Horne dated 10 February 2019 including CV and letter of instruction	18 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
K	Expert report of Professor Dawn Elder dated 15 February 2019 including CV and letter of instruction	18 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
L	Expert report of Professor Johan Duflou dated 13 February 2019 including CV and letter of instruction	19 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
M	Expert report of Dr Allan Cala dated 26 November 2018 including CV and letter of instruction	19 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
N	Further expert report of Dr Allan Cala dated 13 February 2019 including letter of instruction	19 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
O	Expert report of Professor John Hilton dated 22 January 2019 including CV and letter of instruction	19 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
P	Affidavit of Professor John Hilton dated 13 November 2018	19 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
Q	Report of Professor Stephen Cordner (undated)	12 December 2018 (first tendered at second directions hearing) 20 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
R	Letter from Professor Stephen Cordner to the Inquiry dated 8 March 2019 including CV	21 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
S	Section of Patrick's medical records	21 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
T	Report of Professor Caroline Blackwell dated 5 March 2019 including CV and letter of instruction	22 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI

Exhibit	Document	Date of tender
U	Further report of Professor Caroline Blackwell (undated)	22 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
V	Further report of Professor Caroline Blackwell dated 13 March 2019	22 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
W	Report of Professor Robert Clancy dated 13 March 2019 including CV and letter of instruction and further report dated 17 March 2019	22 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
X	Statement of Professor William Rawlinson (undated) including CV	22 March 2019, substantive hearings relevant to forensic pathology and SIDS/SUDI
Y	Report of Professor Jonathan Skinner dated 31 March 2019 including CV and letter of instruction	15 April 2019, substantive hearings relevant to genetics, cardiology and neurology
Z (Amended)	Joint report of Sydney genetics team dated 29 March 2019 including CVs and letters of instruction	15 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AA	Report of Dr Alison Colley dated 26 November 2018 including letter of instruction	15 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AB	Report of Dr Michael Buckley dated 25 February 2019	15 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AC	Genetics tender bundle	15 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AD	Funnel diagram of genomic variant filtering process for hypothesis-free WGS testing (undated)	15 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AE	Pedigree of Kathleen Folbigg taken by Dr Arsov dated 8 October 2018	16 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AF (Amended)	Joint report of Canberra genetics team dated 29 March 2019 including CVs and letters of instruction	16 April 2019, substantive hearings relevant to genetics, cardiology and neurology

Exhibit	Document	Date of tender
AG	Report of Professor Carola Vinuesa dated 2 December 2018	16 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AH	ECG of Kathleen Folbigg dated 17 May 2011	16 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AJ	Report of Professor Monique Ryan dated 15 March 2019 including CV and letter of instruction	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AK	Report of Associate Professor Michael Fahey dated 30 March 2019 including CV and letter of instruction	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AL	Neurology tender bundle	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AM	Seven reports from forensic pathologists of the Victorian Institute of Forensic Medicine: a. Expert report of Professor David Ranson dated 31 December 2018 b. Expert report of Dr Yeliena Baber dated 18 January 2019 c. Expert report of Dr Joanna Glengarry dated 3 January 2019 d. Expert report of Dr Heinrich Bouwer dated 4 January 2019 e. Expert report of Dr Matthew Lynch dated 8 January 2019 f. Expert report of Professor Noel Woodford dated 18 January 2019 g. Expert report of Dr Michael Burke 31 January 2019	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AN	Index to literature referred to in substantive hearings relevant to forensic pathology and SIDS/SUDI	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AO	Policy Statement of the American Academy of Pediatrics, 'Distinguishing Sudden Infant Death Syndrome from Child Abuse Fatalities' (2001) 107(2) *Pediatrics* 437 (formerly trial MFI 24)	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AP	Summary of prosecution medical evidence concerning the deaths and ALTE (formerly trial MFI 39)	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AQ	Crown chronology of deaths and the ALTE for each of the Folbigg children (formerly trial MFI 40)	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology

Exhibit	Document	Date of tender
AR	Summary of Crown coincidence evidence (formerly trial MFI 41)	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AS	Written directions and list of questions to assist the jury (formerly trial MFI 42)	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AT	Further report of Professor Robert Clancy dated 27 March 2019	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AU	Report of Professor Paul Goldwater dated 29 March 2019	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AV	Report of Dr Kathryn Waddell-Smith dated 29 March 2019 including CV and letter of instruction	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AW	Gene lists from Sydney and Canberra genetics teams (undated)	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AX	Response from Professor Kirk and Dr Buckley to joint report of Canberra genetics team dated 9 April 2019	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AY	Response from Canberra genetics team to response of Professor Kirk and Dr Buckley dated 12 April 2019	17 April 2019, substantive hearings relevant to genetics, cardiology and neurology
AZ	Diaries tender bundle (amended to include 1992 diary of Ms Folbigg)	29 April 2019, Ms Folbigg's evidence
BA	Report of Dr Michael Diamond dated 16 April 2019	1 May 2019, Ms Folbigg's evidence
BB	Report of Dr Bruce Westmore dated 16 June 2003 (formerly Exhibit 1 on sentence)	1 May 2019, Ms Folbigg's evidence
BC	Report of Dr Yvonne Skinner dated 22 January 2003 (formerly Exhibit C on sentence)	1 May 2019, Ms Folbigg's evidence

Exhibit	Document	Date of tender
BD	Report of Dr Michael Giuffrida dated 27 August 2003 (formerly Exhibit 2 on sentence)	1 May 2019, Ms Folbigg's evidence
BE	Abdulaziz Zorgani et al, 'Detection of Pyrogenic Toxins of *Staphylococcus aureus* in Sudden Infant Death Syndrome' (1999) 25 FEMS *Immunology and Medical Microbiology* 103	1 May 2019, Ms Folbigg's evidence
BF	Index to literature referred to in substantive hearings relevant to genetics, cardiology and neurology	1 May 2019, Ms Folbigg's evidence
BG	Statement of Craig Folbigg dated 19 April 2019	1 May 2019, Ms Folbigg's evidence
BH	Raw test results of Kathleen Folbigg's exercise testing dated 18 April 2019	1 May 2019, Ms Folbigg's evidence
BJ	Further report of Professor Jonathan Skinner dated 24 April 2019	1 May 2019, Ms Folbigg's evidence
BK	Letter from Professor Jonathan Skinner dated 30 April 2019	1 May 2019, Ms Folbigg's evidence
BL	Report of Associate Professor Hariharan Raju dated 18 April 2019	1 May 2019, Ms Folbigg's evidence
BM	Report of Dr David Drucker dated 18 February 2003 including letter of instruction	1 May 2019, Ms Folbigg's evidence
BN	Diary entries relating to Caleb Folbigg dated 24 May 1989 and 18 July 1989	1 May 2019, Ms Folbigg's evidence
BO	Listening device transcripts (annexed to Exhibit BS): a. Transcription Number 1 - 22 July 1999, Master Tape L26 b. Transcription Number 2 - 23 July 1999, Master Tape B28 c. Transcription Number 3 - 23 July 1999, Master Tape L30 d. Transcription Number 4 - 24 July 1999, Master Tape B30 7:53am e. Transcription Number 5 - 24 July 1999, Master Tape L30 8:02am f. Transcription Number 6 - 26 July 1999, Master Tape L40 g. Transcription Number 7 - 27 July 1999, Master Tape L46	Post hearings

Exhibit	Document	Date of tender
BP	Laura's Blue Book dated 22 August 1997 – 1 June 1998	Post hearings
BQ	Statement of Detective Senior Constable Glen Ward dated 14 December 1999 annexing 41 x photographs	Post hearings
BR	Report of Dr Michael Giuffrida dated 10 May 2019 including CV and letter of instruction	Post hearings
BS	Statement of Detective Senior Constable Bernard Ryan dated 19 November 1999	Post hearings
BT	Letter from Professor Peter Schwartz to Professor Carola Vinuesa dated 20 June 2019	Post hearings
BU	Lia Crotti et al 'Calmodulin Mutations and Life-Threatening Cardiac Arrhythmias: Insights from the International Calmodulinopathy Registry' (2019) *European Heart Journal* (advance)	Post hearings
BV	Supplementary report of Professor Jonathan Skinner, Professor Edwin Kirk and Dr Michael Buckley dated 5 July 2019	Post hearings
BW	Response from Professor Carola Vinuesa, Dr Todor Arsov, Professor Peter Schwartz, Professor Matthew Cook and Professor Michael Overgaard to supplementary report of Professors Skinner, Kirk and Dr Buckley dated 11 July 2019	Post hearings
BX	Crown Notice of coincidence evidence dated 24 October 2002	Post hearings
BY	Crown Notice of tendency evidence dated 24 October 2002	Post hearings
BZ	Letter from Dr Christopher Seton to Dr David Sanders dated 30 April 1998	Post hearings
CA	Letter from Dr Christopher Seton to Dr Quentin King dated 27 August 1997	Post hearings
CB	Transcript of first meeting with expert geneticists dated 10 December 2018	Post hearings
CC	Transcript of second meeting with expert geneticists dated 4 February 2019	Post hearings
CD	Emails exchanged between Professor Skinner and Professor Vinuesa on 11 and 12 February 2019	Post hearings
CE	Letter from Dr Allan Cala to Ms Jane Culver, Office of the Director for Public Prosecutions dated 19 March 2003	Post hearings

Annexure F: List of witnesses who gave evidence before the Inquiry

Name	Transcript reference
ARSOV, Dr Todor	T459-579
BLACKWELL, Professor Cecelia Caroline	T311-352
BUCKLEY, Dr Michael Francis	T369-612
CALA, Dr Allan David	T57-309
CLANCY AM, Professor Robert Llewellyn	T311-352
COLLEY, Dr Alison Fiona	T369-612
CORDNER AM, Professor Stephen Moile	T57-309
DUFLOU, Professor Johan	T57-309
ELDER, Professor Dawn	T18-55
FAHEY, Associate Professor Michael Collingwood	T581-611
FOLBIGG, Kathleen Megan	T621-775, T777-809
GARCIA DE VINUESA DE LA CONTA, Professor Maria Carola	T459-579
HILTON, Professor John Miller Napier	T57-309
HORNE, Professor Rosemary Sylvia Claire	T18-55
KIRK, Professor Edwin Phillip Enfield	T369-612
RYAN, Professor Monique	T581-611
SKINNER, Professor Jonathan Robert	T369-546

Annexure G: Glossary of medical terms

A

Aetiology – The cause of a disease or the study of the causes of disease.[i]

Agonal rhythm – (also called dying heart pattern) is the name given to an ECG pattern that indicates progressive death of the ventricular myocardium.[ii]

AGRF – Australian Genome Research Facility

Amino acids – Small chemical building blocks that join together to form proteins: there are 20 common amino acids which join in different combinations to make up proteins.[iii]

Allele – There are usually two copies of a gene. These two copies are called alleles. In some cases, one or both alleles will be mutated or altered in some way.[iv]

ALTE – Apparent life threatening event (formerly known as near-miss SIDS). An episode that is frightening to the observer and that is characterised by some combination of apnoea, colour change, marked change in muscle tone, choking, or gagging. In some cases, the observer fears that the infant has died.[v]

Anoxia – Condition of the body in which oxygen is entirely absent from tissues[vi]

Apnoea – A complete cessation of breathing that lasts 10 seconds or greater. Sleep apnoea is a disorder in which breathing is repeatedly interrupted during sleep.[vii]

Artefact – Artificial product; in relation to autopsy, a sign or finding imitating pathology, disease, or injury occurring in life.[viii]

Assay – An investigative procedure in laboratory medicine for qualitatively assessing or quantitatively measuring the presence, amount, or functional activity of a target entity.[ix]

Autosomal dominant – A kind of monogenic condition where only one copy of a gene with a pathogenic variant is necessary for an individual to be affected.[x]

Autosomal recessive – A kind of monogenic condition where the person must have a pathogenic variant in both copies of a disease gene to be affected.[xi]

Aystotle/aystolic – (also called cardiac arrest) is a type of ventricular dysrhythmia that exists when there is no electrical activity in the ventricular myocardium. It is the only true arrhythmia (without rhythm).[xii]

B

Benign variant – A variant which does not usually cause any health or developmental concern.[xiii]

Biochemistry (biochemical) – Relating to the chemical substances present in living organisms and the reactions and methods used to identify or characterise them.[xiv]

Bradycardia – A type of arrhythmia characterised by slowing of the heart rate to 60 beats per minute or less.[xv]

Bronchiolitis – Inflammation of the bronchioles (smaller branches of bronchial passageways in the respiratory tract).[xvi]

C

Cardiac arrhythmia – Variation from the normal heart rate or regularity of the heartbeat, usually resulting from irregularities within the conduction system of the heart.[xvii]

Cardiorespiratory arrest – The cessation both of normal circulation of the blood due to failure of the heart and/or normal breathing.[xviii]

Cerebellum – The portion of the brain forming the largest segment of the rhombencephalon (hind brain).[xix]

Cerebral – Relating to or located in the hemispheres of the brain (cerebrum).[xx]

Cerebral oedema – Accumulation of excessive fluid in the substance of the brain.[xxi]

Cerebrum – The largest part of the brain, consisting of two hemispheres separated by a deep longitudinal fissure.[xxii]

Channelopathy (cardiac) – Abnormalities in the ion channels in myocardial cell membranes.[xxiii]

Coliform – Facultative anaerobic, nonsporulating, rod-shaped bacteria that produce acid and gas from the fermentation of lactose sugar. Usually occur in the intestinal tracts of animals, including humans. Examples include *E coli, E aerogenes* and *K pneumoniae*.[xxiv]

Congenital – Born with.[xxv]

Congestion – An excessive amount of blood in an organ or in tissue.[xxvi]

Contusion – Bruise.[xxvii]

Copy number variant ("CNV") – A phenomenon in which sections of the genome are repeated and the number of repeats in the genome varies between individuals in the human population.[xxviii]

CPVT – Catecholaminergic polymorphic ventricular tachycardia. A cardiac condition in which arrhythmias are triggered by physical or emotional exertion.[xxix]

Cyanosis – A bluish coloration of the skin due to the presence of deoxygenated haemoglobin in blood vessels near the skin surface, i.e. in life, a sign of oxygen deficiency.[xxx]

Cytokine – Any of a group of small, short-lived proteins that are released by one cell; to regulate the function of another cell, thereby serving as intercellular chemical messengers. They are best known for the roles they play in the immune system's defence against disease-causing organisms.[xxxi]

D

***De novo* variant** – A type of variant present for the first time in a child and not inherited from either parent.[xxxii]

Digenic inheritance – A phenotype which is caused by variants in two genes.[xxxiii]

DNA sequencing – Determining the pattern or order in which the nucleotide bases occur in a piece of DNA. This sequence is the genetic code.[xxxiv]

Dravet syndrome – A severe form of epilepsy that is part of a group of diseases known as SCN1A-related seizure disorders. The condition appears during the first year of life as frequent febrile seizures.[xxxv]

E

Epicardium – The protective outer layer of the wall of the heart.[xxxvi]

Epidemiology (epidemiological) – The study of the distribution and determinants of health-related states and events in populations, and the application of this study to the control of health problems.[xxxvii]

Epiglottis (epiglottic) – The flap of cartilage lying behind the tongue and in front of the entrance to the larynx (voice box) that keeps food from going into the trachea (windpipe) during swallowing.[xxxviii]

Encephalitis – Inflammation of the brain.[xxxix]

Encephalopathy – Any diffuse disease of the brain that alters brain function or structure.[xl]

Eosinophilic – Readily stained by eosin (an acidic dye).[xli]

Exome – The part of the genome composed of exons, the coding portions of genes, comprising about 1% of the human genome.[xlii]

F

Fibroblasts – The principle active cell of connective tissue. Fibroblasts are large, flat, elongated cells possessing processes extending out from the ends of the cell body.[xliii]

G

Gene – The basic unit of heredity; a segment of DNA which contains the information for a specific characteristic or function.[xliv]

Genome – The complete set of genes carried by an individual or a cell.[xlv]

Guthrie card – Pre-printed collection cards used to store blood collected from newborn babies for screening.[xlvi]

H

Haematoma – A collection of blood, generally the result of haemorrhage/internal bleeding; usually resulting from injury (e.g., bruises in skin) but indicative of more serious injury when located within organs, most critically inside the skull, where hematomas may place pressure on the brain.[xlvii]

Haemorrhage – The loss of blood from a ruptured blood vessel.[xlviii]

Haemosiderin – A particle representing an iron-storage complex that is formed by the breakdown of haemoglobin or an abnormal metabolic pathway of ferritin.[xlix]

Histology – The study of tissue sectioned as a thin slice, using a microtome (a mechanical instrument used to cut biological specimens into very thin segments for microscopic examination).[l]

Histopathology – A branch of pathology concerned with the study of the microscopic changes in diseased tissues.[li]

Heterozygous mutation – A mutation of only the maternal or paternal allele.[lii]

Holter test – Monitoring the patient's heartbeat continuously for long periods of time (24 to 72 hours) through a portable echocardiographic monitor.[liii]

Homeostatic control – The maintenance of a relatively stable internal environment by an organism in the face of a changing external environment and varying internal activity using negative feedback mechanisms to minimise an error signal.[liv]

Homozygous mutation – An identical mutation of both the paternal and maternal alleles.[lv]

Hunter syndrome – Also known as Mucopolysaccharidosis type II. An inherited disorder of carbohydrate metabolism that occurs almost exclusively in males. It is characterised by distinctive facial features, a large head, hydrocephalus, enlargement of the liver and spleen and hearing loss.[lvi]

Hypoxia – Condition of the body in which tissues are starved of oxygen.[lvii]

Hypoxic-ischemic encephalopathy – Brain damage caused by a lack of oxygen and blood flow to the brain.[lviii]

I

IgG deficiency – A deficiency in immunoglobin G (the most common type of antibody in human blood).[lix]

Inheritance patterns – How genetic variants are distributed in families.[lx]

Intercostal recession/retraction – Drawing-in of the intercostal muscles between each rib during breathing. A sign of respiratory distress.[lxi]

Interleukin-10 – An anti-inflammatory cytokine (small proteins used in cell signalling) coded by the IL-10 gene.[lxii]

Intracranial – Within or introduced into the skull.[lxiii]

Intrathoracic – Within the cavity of the chest.[lxiv]

Ischaemia/ischaemic – A restriction in blood supply to tissues causing a shortage of oxygen.[lxv]

L

Laryngomalacia – Congenital abnormality of the larynx cartilage that predisposes to dynamic supraglottic collapse during the inspiratory phase of respiration, resulting in intermittent upper airway obstruction and stridor.[lxvi]

Larynx (laryngeal) – Also known as the voice box, a structure in the neck involved in protection of the trachea (windpipe) and in sound production.[lxvii]

Likely benign variant – Variants for which there is 90% certainty of benignity.[lxviii]

Likely pathogenic variant – Variants for which there is 90% certainty of pathogenicity.[lxix]

Lividity (post-mortem) – A dark-blue staining of the dependent surface of a cadaver, resulting from the pooling and congestion of blood.[lxx]

LQTS – Long QT syndrome. A syndrome characterised by a prolongation in the depolarisation time-course of cardiac myocyte action potentials which can lead to fatal arrhythmias.[lxxi]

Lymphocyte – The primary cells of adaptive immune responses.[lxxii]

M

MCAD – Medium-chain acyl-CoA dehydrogenase. MCAD deficiency is an inherited metabolic disorder that prevents the body from converting certain fats to energy, particularly during periods without food.[lxxiii]

Micro-array testing – A genetic test usually done by obtaining a blood sample that looks for extra or missing DNA segments along chromosomes. Having more or less DNA than usual can cause health or developmental concerns.[lxxiv]

Missense variant – DNA changes that cause a different amino acid to be included in the protein.[lxxv]

Mitochondrial inheritance – Inheritance involving genes found only in mitochondrial DNA. [lxxvi]

Monogenic inheritance – Conditions caused by pathogenic variants in a single gene. One or both alleles of a gene may be affected depending on the type of inheritance.[lxxvii]

Mosaic/mosaicism – Where there is different DNA in different cells of the body.[lxxviii]

Muccopolysaccharidosis – Refers to a group of inherited conditions in which the body is unable to properly break down mucopolysaccharides (long chains of sugar molecules that are found throughout the body). There are seven distinct forms and numerous subtypes of mucopolysaccharidoses.[lxxix]

Multifactorial inheritance – A pattern of inheritance which results from the interaction of one or more genes with environmental factor(s).[lxxx]

Myocytolysis – A type of cellular necrosis involving significant damage to cardiac myocytes caused by myocardial strain.[lxxxi]

N

NATA – National Association of Testing Authorities.

NCIS – National Coronial Information System.

Neuropathologist – A pathologist who specialises in the diagnosis of diseases of the brain and nervous system by microscopic examination of the tissue and other means.[lxxxii]

Nucleotide – Also known as bases, they are the basic components of DNA. They are denoted by the letters **A** (**A**denine), **G** (**G**uanine), **C** (**C**ytosine) and **T** (**T**hymine). The sequence of these bases forms the genetic code.[lxxxiii]

O

Oedema – An abnormal build-up of fluid between tissue cells.[lxxxiv]

Oronasal – Of or relating to the mouth and nose.

P

Paraffin – A white, soft solid used as an embedding medium for tissue processing in histopathology to allow for tissue orientation.[lxxxv]

Pathogenic variant – A variation in a gene that makes it faulty.[lxxxvi]

Pathognomonic – Specifically characteristic or indicative of a particular disease or disorder.[lxxxvii]

Pathologist – A medical professional trained to examine tissues, cells, and specimens of body fluids for evidence of disease.[lxxxviii]

Petechial haemorrhage (petechiae) – Pinpoint haemorrhage; tiny purple or red spots that appear on the skin because of small spots of bleeding in the skin.[lxxxix]

Phenotype – The physical and/or biochemical characteristics of a person, an animal or other organism which are determined by their genetic make-up and/or environment.[xc]

Pneumomediastinum – The abnormal presence of air or another gas in the mediastinum. [xci]

Polysomnogram – A type of sleep study which records brain waves, oxygenation of blood, heart rate, breathing and eye/leg movements during sleep.[xcii]

Proband – An individual being studied or reported on. A proband is usually the first affected individual in a family who brings a genetic disorder to the attention of the medical community.[xciii]

Pulmonary – Concerning, affecting, or associated with the lungs.[xciv]

Pulmonary congestion – A condition characterized by the engorgement of the pulmonary vessels.[xcv]

Pulmonary pleura – The portion of the pleura (the delicate membranous covering of the lungs) that covers the surface of the lungs and dips into the fissures between its lobes.[xcvi]

R

Relative risk – The ratio of probability of an event in an exposed group to the probability of an event in a non-exposed group.[xcvii]

Rigor mortis – The stiffening of the muscles after death.[xcviii]

S

Stridor – Also known as noisy breathing. A medium-pitched respiratory sound, usually with inspiration, that represents resistance to airflow through the airway.[xcix]

Syncope/syncopal – Fainting.[c]

T

Tachycardia – A heart rate that is above the age-adjusted range of normal heart rates.[ci]

Tachypnoeia – Abnormally rapid breathing.[cii]

Thoracic – Involving or located in the chest.[ciii]

Thymus – A small glandular organ situated behind the top of the breastbone, consisting mainly of lymphatic tissue.[civ]

Torticollis – An abnormality in which the neck is in a twisted, bent position such that the head is pulled to one side and the chin points to the other.[cv]

Toxicology – The division of medical and biological science concerned with toxic substances, their detection, their avoidance, their chemistry and pharmacological actions, and their antidotes and treatment.

V

Variant (or mutation) – A variation in the DNA sequence of a genome. [cvi]

Variant of unknown significance – A variant the significance of which to the function or health of an organism is not known.[cvii]

VCGS – Victorian Clinical Genetics Service.

W

Whole exome sequencing – A genomic technique for sequencing all of the protein-coding genes in a genome.[cviii]

Whole genome sequencing – The process of determining the complete DNA sequence of an organism's genome at a single time.[cix]

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