EXHIBIT L

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Consulting Forensic Pathologist

EXPERT OPINION

KATHLEEN MEGAN FOLBIGG

1. My name is Johan DUFLOU.

2. **TRAINING, STUDY AND EXPERIENCE:** I am a specialist forensic pathologist. My professional qualifications are:
   - Bachelor of Medicine, Bachelor of Surgery.
   - Master of Medicine in Forensic Pathology.
   - Fellow of the Royal College of Pathologists of Australasia.
   - Fellow of the Faculty of Forensic & Legal Medicine of the Royal College of Physicians (London).
   - Diploma in Aviation Medicine.

   I have also been trained in the early management of severe trauma and have been awarded a Certificate in Early Management of Severe Trauma (EMST) of the Royal Australasian College of Surgeons.

   I hold a number of professional appointments, including consulting forensic pathologist, senior forensic pathologist at the Forensic Medicine Centre in Canberra ACT, Clinical Professor in the Central Clinical School of the University of Sydney, and Conjoint Associate Professor at the National Drug & Alcohol Research Centre of the University of NSW. I previously worked at the Department of Forensic Medicine in Sydney, where amongst other roles I held the appointment of Clinical Director.

   I have performed many thousands of autopsies on persons who have died in a wide range of circumstances where the deaths have been referred for medicolegal investigation over a period in excess of 35 years. Such cases include a large number of deaths of infants and young children who have died in a range of circumstances including natural disease processes and accidental and inflicted violence.

Since the mid 1980's I have presented expert evidence on many topics in forensic pathology before Courts and other Tribunals in many jurisdictions in Australia and internationally, including on the topics of infant deaths.

As part of my academic appointments, I teach undergraduate and postgraduate students and other professional groups in the field of forensic medicine, including specifically on the topic of investigation of death.

I have also had published in excess of 120 peer reviewed scientific articles in many scientific and medical journals, including many research and other publications on topics which include the investigation of deaths in a wide range of circumstances, including specifically on the topic of sudden death in the young.

I enclose a copy of my resume (**ATTACHMENT 1**).

3. **ACKNOWLEDGEMENT:** I acknowledge that I have read the Expert Witness Code of Conduct set out in schedule 7 of the NSW Uniform Civil Procedures Rules 2005, and I agree to be bound by it. I have made all inquiries which I believe are desirable and appropriate (save for any matters identified explicitly in the report), and no matters of significance which I regard as relevant have, to my knowledge, been withheld from the court.

4. **PURPOSE OF REPORT:** I understand that pursuant to section 77(1)(a) of the *Crimes (Appeal and Review) Act 2001* an inquiry is being held into the convictions of Kathleen Megan Folbigg for three counts of murder, one count of manslaughter and one count of maliciously inflicting grievous bodily harm in respect of her four children on 21 May 2003. I have been asked to comment on aspects of the deaths of the four children, as detailed in a Letter of Instruction of Mr Stuart Gray of Cardillo Gray Partners, dated 24 January 2019 (**ATTACHMENT 2**). My review of the provided material commences at paragraph 7 of this report. My opinions, which are wholly based upon my specialised knowledge, commence at paragraph 46 of this report.

5. **MATERIAL RECEIVED:** I have received and considered the following material in relation to this case:
   - A Letter of Instruction of Mr Stuart Gray of Cardillo Gray Partners, dated 24 January 2019.
   - Petition to the Governor of New South Wales for a Review of the Convictions of Kathleen Folbigg;
   - Extracts of the Uniform Civil Procedure Rules, including UCPR 31.23 Code of Conduct, UCPR 31.27 Experts' reports, and UCPR Schedule 7 – Expert witness code of conduct.

- Supreme Court of Victoria Practice Note No. 2 (Expert Evidence in Criminal Trials), including Item 1 of the included Schedule: Forensic Pathology – Standard Questions to Address the Cause of Death in an Alleged Homicide.
- Autopsy reports:
    - Caleb Folbigg.
    - Laura Folbigg.
    - Patrick Folbigg.
    - Sarah Folbigg.
- Black Book: index and volumes 1, 2 and 3.
- Blue Book: index and volumes 1 and 2.
- Report of Professor Stephen Cordner.
- Report of Professor Michael Pollanen, dated 1 June 2015.
- Report of Professor Carola Vinuesa, dated 2 December 2018.
- An electronic bundle of documents titled "Briefing material", which includes:
    - NSW Biochemical Genetics Service testing of Patrick Folbigg in February 1991.
    - Letter of Senior Constable Joe Lavin dated 11 October 1999 to Dr Bridget Wilcken.
    - Expert Certificate of Dr Wilcken dated 14 January 2000, including results of laboratory genetic tests on the four children.
    - Correspondence between Dr Allison Colley and Dr Wilken regarding Caleb and Patrick in December 1991 and February 1992.
    - Report of Professor Peter Berry, dated November 2000.
    - Correspondence relating to IgG testing of Folbigg children in March 2003.
    - Letter of Dr Allan Cala relating to IL-10 gene theory, dated 19 March 2003.
    - Letter of Dr John Christodoulou regarding genetic causes of some SIDS cases, dated 18 February 2003.
    - Letter of Professor John Hilton regarding genetic testing of Folbigg children, dated 27 February 2003.
    - Transcript of evidence of Dr Wilcken on 16 April 2003.
    - Supplementary report of Professor Berry regarding IL-10 gene theory, dated 29 April 2003.
    - Report of Professor Cecelia Blackwell, dated 8 May 2014.
    - Various research articles and book chapters.
- An electronic bundle of documents titled "Cardiology Documents", which includes:
    - Extracts of medical records of Patrick and Laura Folbigg.

- Statement of paramedic Brian Wadsworth, dated 15 September 1999, including ECG tracing of Laura around time of death.
- Expert Certificate of Dr Brian Bailey, dated 26 March 2003.
- Expert Certificate of Dr Jason Bendall, dated 8 April 2003.
- Expert Certificate of Dr Owen Hughes, dated 15 April 2003.

- An electronic bundle of documents titled "Forensic Pathology Tender Bundle", which includes but is not limited to:
  - Pregnancy related medical records of Kathleen Folbigg.
  - Medical records of Folbigg children.
  - Autopsy reports and associated documentation of Folbigg children.
  - Expert Certificate of Professor Peter Herdson, dated 17 January 2002.
  - Reports of Professor Roger Byard, dated 18 October 2002 and 14 April 2003.
  - Report of Professor Anthony Busuttil, dated 6 November 2002.
  - Expert Certificate of Dr Allan Cala, dated 28 March 2003.
- An electronic file titled "Forensic Pathology Documents."
- An electronic file titled "Second tranche – further set of documents relevant to forensic pathology, SIDS-SUDI, genetics or other."
- An electronic file titled "Genetics tender bundle."
- Additional report of Dr Allan Cala, dated 26 November 2018.
- Letter of Ms Amber Richards dated 7 January 2019.
- A large number of research publications for consideration.

6. I held the appointment of Deputy Director of the Department of Forensic Medicine, Glebe, at the time of the autopsies and compilation of the reports of Sarah and Laura Folbigg, and I was the Acting Clinical Director of the Department of Forensic Medicine at the time of the trial of Ms Folbigg. I have had a number of conversations with both Professor Hilton and Dr Cala in relation to the deaths of all the Folbigg children and have examined the microscopy slides, reports and various other material relating to the deaths as part of those conversations. I have also been asked to provide comment and advice of an administrative nature in relation to this matter in my role as Clinical Director of the Department of Forensic Medicine prior to and following release of the report of Professor Cordner.

REVIEW OF PROVIDED MATERIAL

7. Following is a limited review of medical aspects of the investigation of the deaths of the four Folbigg children, acknowledging that all four deaths have to a variable extent been investigated in detail on a number of occasions, and that this review is a summary of identified important aspects in each case. The review is not meant to be exhaustive, given the volume of material provided.

8. **DEATH OF CALEB FOLBIGG:** Caleb died on 20 February 1989 at the age of 19 days. The pregnancy had been largely uneventful, with the exception of an episode of fainting or fitting on the part of Mrs Folbigg ("Kathleen") on 15 December 1988 – no cause for this was identified. Labour was complicated by production of meconium stained liquor on artificial rupture of membranes and use of Kielland's forceps. Birth weight was 3.28 kg, and Apgar scores were 9 and 9 at one and five minutes. There was initial persistent mild respiratory distress requiring supplemental oxygen administration. Caleb underwent a number of investigations for this while in hospital. Specifically, he had a chest x-ray done which reportedly showed possible minimal pneumomediastinum. His respiratory distress appeared to resolve over the following days while still in hospital. Caleb was fed by bottle, and he appeared to have some initial difficulty with breathing while being fed. Both mother and baby were discharged home on day 5 in a satisfactory state. He was seen by a doctor on 17 February 1989 for mild stridor and a diagnosis of possible congenital laryngeal stridor due to a floppy larynx was made.[1]

9. On 19 February 1989, Caleb was given a feed and put down to sleep in a room adjacent to the parents' bedroom. He was checked by his mother at about 22:00 hours, and appeared to be well. At 02:45 hours he was checked again and found to be unresponsive. She found he was cold to touch and there was a small amount of blood stained froth around the mouth. Caleb's father, Mr Craig Folbigg ("Craig"), responded to Kathleen's screams, and found him to be still warm. He commenced CPR and an ambulance was called. Ambulance officers found Caleb either warm or cold to touch and with either pallor around the nose and mouth or a degree of blue/purple discolouration around and within the mouth. The airway appeared obstructed to

[1] Described by Dr Springthorpe in his letter to Dr Leeder on 21 March 1989 as follows: *"I was able to tell them that though Caleb had undoubted congenital laryngeal stridor (almost certainly on the basis of laryngomalacia) there was nothing abnormal in the way of laryngeal webs or cysts found at autopsy and the cause of his demise remains a mystery."*

ambulance officers. They continued attempted resuscitation for a further period of time prior to declaring Caleb deceased. The initial recorded cardiac rhythm was asystole.

10. A coronial autopsy was performed later in the day, with the following pertinent observations made by Dr Cummings:
    - Morphologically normal infant (length 55 cm, weight 3970 g).
    - No external signs of injury.
    - No abnormalities identified on naked eye internal examination.
    - Large quantity of curdled milk in stomach.
    - Microscopy showed incomplete aeration of lungs with some extravasated red blood cells and eosinophilic exudate in alveolar spaces. Other organs within normal limits.
    - Toxicology: negative.
    - Cause of death: SUDDEN INFANT DEATH SYNDROME.

11. **DEATH OF PATRICK FOLBIGG:** Patrick died on 13 February 1991 at age 8 months after a lengthy illness. He had been born on 3 June 1990 by normal vaginal delivery at 39 weeks following a largely uneventful pregnancy. Apgar scores were 7 and 8 at one and five minutes respectively. He had a birth weight of 3410 g, and the post-natal course in hospital was unremarkable. The infant was bottle fed from birth. Both Kathleen and Patrick were discharged home on 8 June 1990. A number of investigations were performed electively on 14 June 1990 in the form of a sleep study including an ECG and serum electrolyte levels, and a barium swallow. The barium swallow showed no evidence of reflux, but the presence of contrast in the nose suggested uncoordinated swallowing. Over the following months, he was taken to his general practitioner for standard infant related problems, none of which raised concerns.

12. On 17 October 1990, Kathleen placed Patrick in his bed at around 20:30 hours. He was checked by Craig at around 22:30 hours, and was heard to cough by Kathleen at around 03:00 hours. At 04:30 hours, Kathleen heard Patrick gasping and she saw his lips were blue. He was making minimal respiratory effort and he was unresponsive and floppy. Craig woke to Kathleen's screaming and he commenced CPR. After a period of time, Patrick made a high pitched cry and he appeared to commence breathing. Ambulance arrived at 04:41 hours and continued resuscitation, noting he was having respiratory difficulty with a tracheal tug and costal recession. He was taken to hospital where resuscitation of the patient continued.

13. Initial examination of the infant at this time revealed no evidence of trauma. He was seen to be arching his back and neck, and a medical record entry on admission to hospital indicates he had been observed to have done this in the past as well. An x-ray of the lungs revealed possible bronchiolitis. An ECG and an EEG were both reported as normal. Provisional diagnoses of mild bronchiolitis, epileptic seizure and "near SIDS" were made. That evening Patrick had a generalised seizure, and was administered diazepam followed by phenobarbitone. Seizures were difficult to control, were both generalised and right sided focal, and appeared to be associated with low fevers. Investigations including a cranial ultrasound, lumbar puncture, a metabolic screen and viral studies were all normal, while a CT scan of the brain showed hypodense regions in both the temporal and occipital lobes and was interpreted as being compatible with encephalitis. He was administered acyclovir for suspected herpes encephalitis, and after 3 days was seizure free. Patrick was discharged on 20 October 1990 with diagnoses of seizure disorder probably due to viral encephalitis, and bronchiolitis. Medication prescribed included phenytoin and phenobarbitone, both for seizure control.

14. Patrick was re-admitted to hospital on 4 November 1990 following a seizure lasting about 90 minutes. He was diagnosed as having an upper respiratory tract infection and adenovirus was cultured from an eye swab. A repeat EEG showed progressive deterioration with multifocal epileptogenic foci identified, and a repeat CT scan showed cerebral atrophy and areas of dystrophic calcification. Numerous other investigations were performed, including a rectal biopsy, extensive biochemical and metabolic tests, and viral and bacterial tests, all of which were recorded as normal. The possibility of shaken baby syndrome was suggested but not pursued further. Patrick had further admissions to hospital on 14 November 1990 and 22 December 1990.

15. On 12 February 1991, Patrick was observed to have a fever, but he appeared to sleep well during the night. He woke as usual the following morning and appeared to be his usual self to Craig. Craig went to work, and at 10:00 hours was called by Kathleen in relation to a sudden deterioration of their son. Craig arrived a short time later and commenced resuscitation, and a short time later ambulance arrived and paramedics took over. They observed Patrick to be blue and unresponsive. He was assessed as having a normal skin temperature and his buccal mucosa was described as pale. There may have been shallow respiration or apnoea at this time. Patrick was taken to hospital where resuscitation was continued and the initial heart rhythm on ECG was recorded as asystole. Despite continued resuscitation, Patrick was unable to be revived and he

was declared dead at 10:40 hours on 13 February 1991. The cause of death was given as ASPHYXIA due to AIRWAY OBSTRUCTION due to or as a consequence of EPILEPTIC FITS.

16. A non-coronial/hospital autopsy was performed at the Newcastle Mater Hospital later in the day, with the following pertinent observations made by Drs Bishop and Singh-Khaira:
    - Normally formed infant for age; body weight 8.57 kg, head circumference 44 cm, body length 53 cm.
    - No evidence of injury.
    - Frothy mucoid secretions in airways with a degree of pulmonary congestion.
    - Thymus described as enlarged at 30 g (mean normal weight 37.21 g at age 8 months).[2]
    - Neuropathologic examination of fixed brain:
        - Atrophy of both occipital lobes with cystic degeneration at grey-white junction.
        - Areas of firm white matter in occipital, frontal and parietal lobes.
    - Neuropathology microscopy opinion:
        - Old infarcts and gliosis in the form of old laminar necrosis most severe in the parieto-occipital area.
        - Multifocal neuronal atrophy consistent with epileptic seizures.
        - Chronic inflammation of involving leptomeninges either non-specific or related to cortical infarcts or related to encephalitis.
        - Linear cortical calcification likely a consequence of laminar cortical necrosis.
    - Microbiology: Mixed organisms identified on blood culture; lung tissue negative for bacteria, viruses and mycoplasma.
    - Toxicology: Phenobarbitone and carbamazepine levels within therapeutic range.
    - Cause of death: ASPHYXIA due to AIRWAYS OBSTRUCTION due to or as a result of EPILEPTIC FITS due to or as a result of ENCEPHALOPATHIC DISORDER (UNDERLYING CAUSE NOT DETERMINED ON INVESTIGATION).

17. **DEATH OF SARAH FOLBIGG:** Sarah died on 30 August 1993, at the age of 10 months. She was born on 14 October 1992 at 39 weeks gestation by normal vaginal delivery. The pregnancy had been complicated by vaginal bleeding requiring hospital admission on 21 February 1992. At birth, Sarah weighed 3.02 kg, she had a head circumference of 34.5 cm, and Apgar scores of 9 and 10 at one and five minutes respectively. Initially Sarah was breastfed, but this was changed

[2] Collins K, Byard R. 2014. Forensic pathology of infancy and childhood. Springer, New York.

to bottle feeding following discharge from hospital. Sarah slept on an apnoea blanket; there were reportedly some difficulties with use of the blanket, and its use ceased in around mid-August 1993 at about 10 months. On 5 November 1992, a sleep study was performed, showing very few sleep apnoeas, some periodic breathing, and quite long episodes of hypoventilation. Blood tests performed on that day also showed dicarboxylic aciduria without significant ketosis. At the age of 3 months and 4½ months, Sarah was examined by her paediatrician who found she was progressing well. Further sleep studies were arranged and reported as normal.

18. On 18 August 1993, Sarah was prescribed antibiotics in the form of flucloxacillin for a cold like illness. The medication was reportedly discontinued on 26 August. On 29 August 1993, Sarah was put to bed as usual at around 21:00 hours. According to Craig she had a cold like illness at this time. Kathleen reportedly appeared agitated that evening, and was upset because Sarah initially did not want to go to sleep. Craig put Sarah to bed, and she was heard to be snoring some short time later. Kathleen heard Sarah turn over in her sleep at around midnight. Kathleen went to the toilet at about 01:30 hours and could not hear Sarah breathing. She was a blue colour, cool to touch, and there was a discharge around the nose. CPR was commenced by Craig, and an ambulance was called. On attendance of the paramedics, Sarah was observed to be blue around the mouth, with vomit and mucus in her mouth and an obstructed airway. Her skin temperature was described as both normal and cold, and her buccal mucosa was pale. She was unresponsive, was not breathing and was in asystolic cardiac arrest. She was taken to Maitland Hospital where she was declared deceased at 04:30 hours.

19. An autopsy was performed by Professor John Hilton on 31 August 1993, with the following pertinent observations recorded:
    - Normally formed well-nourished female.
    - Body weight 9.44 kg, body length 71 cm.
    - Perimortem injuries:
        - A 1.5 cm scratch on the antero-lateral aspect of the right upper arm.
        - Two tiny punctate abrasions, one immediately below the lower lip on the left side, the other slightly to the left side of the midline of the chin.
    - Congested/haemorrhagic uvula of normal size, placed anterior to the epiglottis producing an obstructive element in the airway. Microscopy of uvula – marked congestion. Microscopy of larynx – light mixed lymphocytic infiltrate.
    - Gastric contents in airways.

- Both lungs showed focal areas of collapse. Microscopy – a light interstitial acute inflammatory infiltrate around occasional bronchioles, and multiple neutrophils localised to interstitium and a lymphoid deposit in another lung section.
- Occasional minor petechial haemorrhages present on surfaces of lungs and heart.
- Occasional petechiae on the surface of and within the thymus.
- Modest cervical and mesenteric lymph node enlargement.
- Stomach contents: A moderate quantity of curdled milk and possibly egg white.
- Neuropathology: Normal brain on naked eye and microscopic examination.
- Toxicology: Negative for common drugs and poisons.
- Microbiology: Multiple samples taken for bacterial and viral investigations. No pathogenic organisms isolated.
- Biochemistry: Non-contributory.
- Cause of death: SUDDEN INFANT DEATH SYNDROME.

20. **DEATH OF LAURA FOLBIGG:** Laura died on 1 March 1999 at the age of 1 year 8 months. Prior to conception, both Craig and Kathleen had attended the sleep disorders unit at Westmead Children's Hospital, where concerns were expressed in relation to familial respiratory obstruction, especially during sleep. Laura was born on 7 August 1997 at term by spontaneous vaginal delivery. Her birth weight was 3.26 kg, and she had Apgars of 9 and 10 at one and five minutes respectively. Laura was kept in hospital for two weeks, during which time she underwent a range of investigations. A sleep study done by Dr Sefton diagnosed mild central apnoea but no evidence of obstruction or bradycardia. A range of biochemical, metabolic and other blood and urine tests were performed, all reported as normal. She was breast fed for the first two weeks, then bottle fed. Laura was discharged home after both parents were trained in CPR and use of a home cardiorespiratory monitor.

21. A second sleep study was performed on 2 October 1997. This was interpreted as showing moderate central apnoea of infancy with a mild improvement since the previous study, although an obstructive component was now documented. Recommendations were made that all SIDS-protective factors should continue to be observed and that serious consideration be given to hospitalization of the child if she developed an upper respiratory tract infection. A third sleep study, done on 3 February 1998, was interpreted as Laura having normal sleep breathing without evidence of upper airway obstruction. In subsequent consultation with Dr Seton, Laura's sleep breathing remained normal. Nevertheless, it was recommended that use of the apnoea monitor

continue. The monitor was eventually returned to the Children's Hospital at around Laura turned 1 year.

22. Laura was seen by her general practitioner on multiple occasions during her life, generally for minor childhood illnesses, including flu like symptoms, upper respiratory tract infections, superficial burns and various rashes and fevers.

23. On waking on 1 March 1999, Laura was found to have a runny nose and "congestion" of her chest, and she was being given Demazin for this. Craig went to work as usual, and at about 11:00 hours both Kathleen and Laura attended Craig's work for about 30 minutes. They then returned home and Laura went to bed while Kathleen went to feed and play with the dog. She heard Laura cough and about 5 to 15 minutes later when she went to check on her found her to be unresponsive in bed, slightly blue and cool to touch. An ambulance was called, arriving at 12:14 hours. Paramedics found Laura with a normal skin temperature, cyanosed and unresponsive. The oral cavity and airway were described as either clear or obstructed. Her initial cardiac rhythm was bradycardia followed by a number of abnormal heart rhythms.[3] Resuscitation was continued by the paramedics, and Laura was taken to Singleton Hospital where she was found to be asystolic, not breathing, and with fixed dilated pupils. Despite further resuscitative efforts, Laura failed to respond, and she was declared dead at 12:45 hours. Hospital personnel found no injuries on the body.

24. A crime scene examination of the home was performed, revealing a number of small dark stains on the pillow of her bed. A screening test was positive for blood, but confirmatory testing for human blood was unsuccessful.

25. An autopsy was performed by Dr Allan Cala commencing at 21:00 hours on 1 March 1999, with the following pertinent observations recorded:
    - Morphologically normal female.
    - Body weight 11.52 kg, body length 80.5 cm.
    - Injuries:
        - A 5 x 3 mm ovoid brown bruise on the left knee.
        - A 12 x 10 mm brown ovoid bruise on the right distal lower extremity.

[3] MFI 34 – ECG tracing made by ambulance personnel, Blue Book page numbers 820-829.

- Heart: weight 62 g (mean normal weight for body weight: 66 g, range 43 – 101 g),[4] 8 mm diameter area of haemorrhage on the posterior surface of the left atrium. Microscopy: *"Within the myocardium is a moderately dense infiltrate of lymphocytes which have aggregated in certain areas particularly subendocardially and along the superficial surface of the myocardium, although further sections show large aggregates in the central area of the left ventricle. In these areas, there are large clusters of lymphocytes surrounding degenerate myocytes. Myocytolysis is present. No viral inclusions are seen. The appearances are of myocarditis, which is probably viral in aetiology."*
- Lungs: Focally haemorrhagic with areas of collapse. Microscopy: increased lymphocytes in the interstitium and some alveoli. Widespread areas of haemorrhage, with oedema fluid, foamy macrophages and some fibrin.
- Thymus: Petechial haemorrhages. Microscopy shows focal cortical haemorrhages.
- Spleen: Normal. Microscopy showed many germinal centres with appearances of a probable viral infection.
- Stomach: A small quantity of milky type fluid admixed with vegetable type material.
- Neuropathology: No significant pathology identified.
- Toxicology: No common drugs or poisons detected. No chlorpheniramine detected.
- Microbiology: No pathogenic organisms identified.
- Cause of death: UNDETERMINED.

26. **LETTER OF Dr CALA:** In a letter dated 19 June 2001, Dr Cala opined that haemosiderin in lung tissue is considered by some forensic pathologists to indicate previous mechanical obstruction of the airway or deliberate smothering, but that this is not a view widely held by the forensic pathology community. With reference to the myocarditis in Laura, he provided comment that the inflammation was *"light in amount and patchy in distribution"* and that this is an incidental finding. The heart also appeared normal on naked eye examination. With reference to the possibility of smothering being the cause of death of all the children, Dr Cala opined that it was a possible cause of death for all four children.

27. Dr Cala provided further written opinion in the form of an Expert Certificate dated 28 March 2003, after he was shown video footage of Laura playing on the day prior to her death. He

[4] Scholz D, Kitzman D, Hagen P, et al. Age-related changes in normal human hearts during the first 10 decades of life. Part I (Growth): A quantitative anatomic study of 200 specimens from subjects from birth to 19 years old. Mayo Clinic Proceedings 1988;63:126-136.

opined that Laura appeared entirely well and had she been suffering from significant myocarditis at the time she would have been short of breath on even slight exertion, may have been vomiting and would have been expected to be disinterested in play. He concluded that he was now of a firmer view than before that the myocarditis played no role whatsoever in the death, and was an incidental finding.

28. **REPORT OF Dr BERRY:** Dr Berry examined the microscope slides of the four children and made the following additional findings:
    - There is haemosiderin (using a Perl's stain) in macrophages in lung tissue of Caleb. There is no stainable iron in lung tissue of Sarah and Laura.
    - Sections of heart tissue of Laura are described as follows: *"Heart muscle shows a patchy but widespread interstitial mononuclear infiltrate in the right and left ventricle. There is no definite myocyte necrosis."*

    Dr Berry reached the following conclusions in his report:
    - Caleb: The finding of haemosiderin in Caleb's lungs would not allow SIDS as a cause of death. Suffocation is suspected on the basis of the autopsy and the diary entry. The presence of haemosiderin however has little weight placed on it because there is no indication of prior collapses.
    - Patrick: ALTE's are not part of the usual natural history of SIDS. The cause of death is best given as Unascertained.
    - Sarah: Taken in isolation, the cause of death resembles SIDS but she was older at time of death than most. The cause of death in isolation would be SIDS with misgivings.
    - Laura: Inflammation of the heart can cause death by congestive heart failure or by sudden arrhythmia. *"Most pathologists would have accepted it as the cause of death."* However, the presence of inflammation in the heart does not necessarily mean it was responsible for death. Taken in isolation, the cause of death would be given as Myocarditis.
    - Taken together: There were no pathological features of deliberate injury, thus if death was the result of the actions of a carer the most likely mechanism is suffocation. Suffocation in young children is often unaccompanied by external and internal signs. Intrathoracic petechiae are commonly found in suffocation, but are also often seen in SIDS. It is probable that Caleb, Patrick, Sarah and possibly Laura were suffocated by the person who found them lifeless.

29. **REPORT OF PROFESSOR HERDSON:** In his report of 17 January 2002, Professor Herdson concluded as follows:
    - Taken in isolation, the cause of death of Caleb is undetermined but apparently consistent with SIDS.
    - The history of an ALTE with subsequent abnormalities is most unusual for a death due to SIDS, and the cause is better given as Undetermined.
    - When taken in isolation, the cause of death of Sarah could be SIDS.
    - With reference to Laura, there is myocarditis and this is consistent with her previous illness, but is probably incidental to death.
    - When considered together, the first unexplained death may be attributed to SIDS, the second undetermined, and the third homicide until proven otherwise.[5]

30. **REPORT OF PROFESSOR BYARD:** In his report of 18 October 2002, Professor Byard opined as follows:
    - Sequential death of four children in the same family is exceedingly rare, and homicide or inherited anomaly must always be considered.
    - Changes of suffocation are often non-specific.
    - In this case, both Patrick and Laura had unequivocal abnormal findings at autopsy. The former had chronic brain damage and poorly controlled epilepsy and the latter had established myocarditis – both are well-known and accepted causes of death. Additionally, Caleb had clinical evidence of airways compromise and Sarah had autopsy evidence of airway narrowing.
    - If the deaths were presented as individual and isolated deaths in separate families, the causes of death would be as follows:
        - Caleb: Undetermined, given various omissions in the examination and his history of breathing problems (laryngomalacia).
        - Patrick: Epilepsy against a background of possible encephalitis.
        - Sarah: Undetermined, on the basis of the autopsy finding of a narrowed airway.
        - Laura: Myocarditis.
    - When taken in combination, all four death would be given as undetermined with previously listed conditions. The deaths, by definition, cannot be viewed as being due to SIDS.

[5] Historically termed Meadow's law, after Professor Sir Roy Meadow in the United Kingdom, alternatively DiMaio's rule after Dr Vince DiMaio an imminent forensic pathologist in Texas.

31. Professor Byard provided a further report on 14 April 2003, in relation to the video footage of Laura prior to death and the cardiac microscopy slides. He opined that Laura appeared normal and not unwell in the video footage, although he placed little significance on the footage. With reference to the microscopy slides of the heart, he stated that myocarditis was present in all seven microscopy slides. Death due to myocarditis can have no prior symptoms, both as determined by his person study of such cases and from review of medical literature on the topic. He also opined that myocarditis can be incidental to death, but to exclude this as a cause of death a precise alternative cause of death would need to be given.

32. **REPORT OF PROFESSOR BUSUTTIL:** In his report of 6 November 2002, Professor Busuttil opined as follows:
    - It is highly unusual to have four deaths of siblings over a period of 8 years.
    - Attempts at suffocating a child will result in internal bleeding into the lung tissue, and if there has been a period of survival, haemosiderin laden macrophages may be seen.
    - Caleb: The death should not have been attributed to SIDS because of clinically diagnosed upper airway obstruction. Imposed airway obstruction cannot be completely excluded.
    - Patrick: The death should not have been attributed to SIDS or asphyxia. There was a serious brain condition which could be due to encephalitis or due to imposed airway obstruction, and which resulted in life threatening convulsions.
    - Sarah: The death has appearances typical of SIDS. The uvula abnormality may have produced some airway obstruction.
    - Laura: There is myocarditis which may be completely incidental or may have caused sudden unexpected death.
    - *"It certainly cannot be said, indeed beyond reasonable doubt, that these deaths were irrefutably due to imposed or induced airway obstruction, as by suffocation."*

33. **EVIDENCE OF Dr SPRINGTHORPE:** Dr Springthorpe, a paediatrician provided the following evidence:
    - Caleb's stridor had nothing to do with his death. Obstruction of the airway by laryngomalacia, webs or cysts was totally excluded.
    - The cause of Caleb's death remains a mystery.
    - It is possible to smother an infant with a pillow over the face and leave no marks whatsoever.

34. **EVIDENCE OF PROFESSOR HILTON:** Professor Hilton provided the following opinions in his verbal evidence on 14 April 2003:

- The death was not considered frankly suspicious prior to autopsy.
- Punctate abrasions below the mouth could be due to application of minor force to that area by the child or another person.
- Pulmonary oedema and haemorrhage is consistent with an asphyxial mode of death, including deliberate smothering, as well as many other mechanisms. There is nothing specific about these findings. It is a finding which has been recorded in smothering.
- Petechial bleeding into the lungs is thought to be the result of negative pressure formation while attempting to inhale.
- Petechial haemorrhages on the heart and thymus can be seen in cases of asphyxiation.
- There was an unusually positioned uvula which was lying in front of the epiglottis, and which had the potential to occlude the airway. It was not clear whether this would have occluded the airway during life and it is possible it was an artefact.
- Microscopy of the upper airways tissues and the lungs showed inflammation and congestion, consistent with a mild respiratory tract infection.
- Microbiological testing reveled contaminants, and possibly pathogen organisms in the form of streptococcus bacteria. When taken together with the autopsy findings it is difficult to determine whether this caused death, but generally inflammation would be more than what was seen in cases where it caused death.
- The diagnosis of SIDS was based on a combination of positive and negative findings.
- SIDS can occur over the age of one year, although the vast majority are in infants under that age.
- The possibility of intentional suffocation should be considered if there has been a previous unexpected or unexplained death of a sibling, or there has been an ALTE of a sibling while in the care of the same person who cared for the deceased, or there has been death following ALTE's in a sibling while in the care of the same person who cared for the deceased.
- Professor Hilton disagrees that the cause of death should have been given as unknown or undetermined causes.
- Professor Hilton disagreed that the presence of the punctate abrasions under the mouth and the scratch on the arm should have excluded a diagnosis of SIDS.
- It may be difficult to differentiate between SIDS and accidental or deliberate suffocation with a soft object.
- On balance, the cause of death in this case is SIDS.

- There is no specific autopsy test for asphyxia.
- She died from a cessation of breathing, and it is not possible to state how she stopped breathing.
- Signs of intentional suffocation include petechial haemorrhages on the face, eyelids and eyes, damage to the frenulum, and bruising of the inside of lips, none of which were found in Sarah.
- Ten month old children can scratch their body and face. The injuries could also be the result of resuscitation.
- The amount of force required to cause the abrasions is minimal.
- The presence of petechiae in chest organs tends to favour a diagnosis of SIDS over intentional suffocation, and are much more commonly seen in SIDS than in deliberate suffocation.
- Injury to the mouth need not necessarily be seen in cases of deliberate suffocation with a pillow.

35. Professor Hilton was recalled on 24 April 2003, and gave the following additional evidence:
    - Microscopy slides of Laura's heart showed fairly extensive inflammation of the heart muscle, using a scale of 1 to 10, it would probably be around 5 or 6. It was more than moderate intensity.
    - At the time of death Laura was suffering from myocarditis, and it could have caused death.
    - Myocarditis may be incidentally found in causes where there is another unequivocal cause of death. There was no such other cause of death in this case, and the only pathology identified was myocarditis.
    - Myocarditis usually has clinical manifestations prior to death, but it can also be entirely silent clinically prior to death.
    - Laura's known activities prior to death do not exclude myocarditis as a cause of death.
    - The presence of myocarditis does not exclude deliberate suffocation as a cause of death.
    - The punctate abrasions on Sarah's face do not have characteristics of deliberate suffocation.
    - Suffocation with a hand with a ring with jewels would more likely result in scratches than punctate abrasions. Use of a pillow with a button would unlikely cause punctate abrasion. A soft toy would highly unlikely have caused punctate abrasion.
    - No photographs were taken at the time of Sarah's autopsy. This was standard practice at the time.
    - The punctate abrasions likely occurred not more than a few hours prior to death.

36. Professor Hilton was recalled a second time on 7 May 2003 as a result of him having found photographs taken at the time of Sarah's autopsy. He provided the following opinions:
    - Photographs confirm the descriptions given in the autopsy report.
    - The displaced uvula is most likely a dissection artefact. If this was thought to have caused death, the cause of death would have been given as respiratory obstruction due to a displaced uvula, and not as SIDS.

37. **EVIDENCE OF Dr CALA:** Dr Cala gave verbal evidence on 15 and 16 April 2003, with the following opinions expressed:
    - Asphyxia is any condition resulting in death whereby there is failure terminally to either adequately oxygenate tissue or failure to take carbon dioxide away from tissue. It is death from a failure to breath.
    - The only injuries on Laura's body were bruises on the legs, which were probably several days old. There were no facial injuries, and no petechial haemorrhages on the face or eyelids.
    - Focal collapse of lung tissue and lung petechiae are nonspecific, but are also consistent with asphyxiation.
    - It is not possible to differentiate SIDS from smothering at autopsy. Both infants who have been smothered and those who have died of SIDS may have petechiae on the organs of the chest. Eyelid petechiae and injuries to the face and mouth may or may not be seen in cases of smothering.
    - It may take 20 to 30 seconds or possibly even longer to smother a child of 19 months of age to either unconsciousness or death. Death may follow a short time after the onset of unconsciousness.
    - The myocarditis observed in Laura is consistent with the after effects of a cold or flu and Dr Cala does not believe it played a role in causing death.
    - The heart was normal macroscopically and on microscopy the inflammation was *"quite patchy and rather mild in the sense that although the inflammation existed it was of a rather low amount as opposed to other cases that I've seen where the inflammation was much heavier in the heart and in other organs."*
    - Inflammation of this severity may be seen in a person who has died of totally unrelated causes.
    - Following review of a video taken of Laura 23 hours prior to death, Dr Cala opined that *"it is quite unlikely that she has died as a result of the effects of myocarditis."*

- Death due to rhythm disturbance is *"in all likelihood, very unlikely"* in Laura's case, and it is not a reasonable possibility that she died of myocarditis.
- The cause of death was undetermined, which can include death due to natural causes which cannot be found, and other inflicted causes of death such as suffocation, and accidental causes of death.
- SIDS is an invented term and not a natural disease, although it is a natural process.
- Laura did not die of SIDS because she was too old at 20 months, even in the setting of a family history.
- Dr Cala would not make a diagnosis of SIDS as a cause of death if a sibling had previously died from SIDS. Dr Cala would not have given SIDS as the cause of death in the case of Sarah, considering there had been two previous deaths.
- Neither Sarah nor Laura had obstructive apnoea on sleep studies.
- Dr Cala suspects that all children died in the same way that Laura did, and that although not able to be proved medically that they were deliberately smothered. The post mortem findings for all four children are consistent with deliberate smothering, and there is no other entity that could account for all four deaths apart from that.
- If the death of each child was considered in isolation or the only one known about, smothering would be suspected by Dr Cala. Smothering has to be suspected in all cases in the death of a child who dies suddenly because it leaves no trace and it can be difficult to prove.
- The circumstances in which the children were found, their sudden deaths, together with the negative death scene examination by police and negative autopsies link the four cases, because there are no positive features in smothering.
- Dr Cala has never read of a child who has died of a floppy larynx.
- In Caleb's case if he had done the autopsy without any knowledge of what happened to the other children subsequently he would have given the cause of death as undetermined.
- Caleb likely died from a catastrophic asphyxiating event of unknown causes.
- Patrick's ALTE is consistent with him having been deliberately smothered, and it is a possibility that his ALTE was the result of an acute catastrophic asphyxiating event.
- Considering Patrick's case in isolation, the cause of death at autopsy would have been given as undetermined. It is possible that Patrick died as a result of deliberate smothering or an acute catastrophic asphyxiating event of unknown causes.

- Considering Sarah's case in isolation, the cause of death at autopsy would have been given as undetermined. It is possible that Sarah died as a result of deliberate smothering. Professor Hilton has not been able to exclude deliberate or accidental trauma.
- Sarah's cause of death was not SIDS because of her age and the injuries to the face.
- It is probable that Sarah died of an acute and catastrophic asphyxiating event of unknown cause.
- All four cases should have been diagnosed as having died of undetermined cause, all died in circumstances consistent with deliberate smothering, they all possibly died from an acute and catastrophic asphyxiating event of unknown cause and there was no natural cause of death that could account for all four deaths and the ALTE.
- On cross examination, Dr Cala agreed there was no evidence of manual asphyxiation despite a thorough examination of Laura. There was no evidence of a struggle by the child.
- Because there are no positive signs of suffocation or asphyxiation, it can almost never be ruled out at autopsy.
- It is theoretically possible that the marks on Sarah's face are the result of resuscitation, although marks on the face are uncommon in resuscitation.
- Children who die of myocarditis die suddenly and unexpectedly in a small percentage of cases.
- Normal or routine histologic examination of heart does not include sampling of the cardiac conduction system. Examination of the conduction system would have required retention of the heart following the autopsy.
- Myocarditis cannot be excluded as a cause of death, when the case is looked at in isolation, but it would not have been given as a cause of death because the inflammation was not particularly heavy, there was no evidence of heart failure, and there was evidence of a viral infection in other organs.

38. **EVIDENCE OF PROFESSOR HERDSON:** Professor Herdson presented the following evidence:
    - He was not aware of any reports of death in an infant due to laryngomalacia.
    - Sudden infant death at 19 days is uncommon.
    - It is virtually impossible to differentiate SIDS from accidental or deliberate suffocation.
    - Professor Herdson opined he can be certain that Caleb died from a sudden catastrophic asphyxiating event of unknown cause.
    - Patrick's ALTE was consistent with being deliberately or accidentally smothered, and the ALTE is due to a sudden catastrophic asphyxiating event of unknown cause.

- The cause of Sarah's death is either SIDS or undetermined but it is consistent with smothering and appears to be the result of a sudden acute catastrophic asphyxiating event of unknown causes.
- The significance of the myocarditis in Laura's heart is not known, but he favours it being incidental and not the cause of death.
- The presence of myocarditis would likely have meant that Laura needed more oxygen than normal for her heart to function.

39. **EVIDENCE OF PROFESSOR BYARD:** Professor Byard produced a report in relation to myocarditis and its significance at autopsy. He gave verbal expert evidence on 7 May 2003, and opined as follows:
    - The cause of Caleb's death is undetermined and not SIDS because no death scene examination was performed and accidental asphyxia has therefore not been able to be excluded, there was no detailed examination of the brain, and there was abnormality with his voice box but the larynx was not examined microscopically. It does however not exclude the possibility of death due to SIDS.
    - Death due to laryngomalacia is probably very rare.
    - The autopsy findings in suffocation can be similar to that of SIDS.
    - The presence of haemosiderin in lungs in infant deaths is not indicative of suffocation in that it is seen in around 20% of SIDS cases.
    - The cause of Patrick's ALTE is not known, but it could be an aborted SIDS, or a problem with breathing, or encephalitis or a manifestation of epilepsy.
    - It is not clear in the photographs whether Sarah's uvula was normal or not.
    - Death due to SIDS cannot be excluded in the case of Sarah.
    - Laura had established widespread myocarditis with necrosis of myocytes.
    - It is not possible to tell how many people with common viral infections have myocarditis.
    - Incidental myocarditis is uncommon in cases of death due to other causes.
    - Deliberate suffocation cannot be excluded as the cause of death in all four cases.
    - Professor Byard was unable to think of any reports of three or more children who have died or had an ALTE.
    - Determination of cause of death is part based on the pathology found and part based on the circumstances leading up to death.
    - It is unlikely that a floppy larynx had anything to do with the death of Caleb.
    - An asphyxiating event is the most likely explanation for haemosiderin in Caleb's lungs.

- An ALTE caused by a first epileptic seizure would be highly unusual.
- Based on South Australian research, statistically speaking there is a greater chance of Laura having died of a cause not related to myocarditis than of myocarditis.
- It is not a reasonable conclusion to say that all four children died from the same natural cause.
- Professor Byard is not aware of any case where four children in one family have died of four different natural causes.
- Persons who die of epileptic seizures do not need to have physical evidence of a seizure at autopsy.
- Death due to natural causes cannot be excluded in any of the four children.

40. **REPORT OF PROFESSOR CORDNER:** Professor Cordner produced a lengthy report with appendices in April 2015. The report considers multiple aspects of death medicolegal death investigation, including the investigation of infant deaths generally and those of the Folbigg children specifically. The report also considers the diagnoses of asphyxia, sudden infant death syndrome (SIDS), acute life-threatening event (ALTE) and myocarditis. I have previously written a brief and limited review of Professor Corner's report in my briefing note to Ms Foster-Curry (**ATTACHMENT 3**), and I do not propose to repeat that review in this document.

41. **REPORT OF PROFESSOR POLLANEN:** Professor Pollanen, Chief Forensic Pathologist for the province of Ontario in Canada performed a peer review of Professor Cordner's report and issued a report of that review on 1 June 2015. Professor Pollanen largely agrees with the observations made by Professor Cordner, and he makes the following comments in relation to the report and in relation to death investigation generally:
    - Unexplained deaths in infancy should not be considered homicides, in the absence of satisfactory proof of there being actions by other persons. Meadow's Rule (alternatively DiMaio's Rule) is flawed and an oversimplification.
    - Two of the deaths have a satisfactory natural cause of death.
    - A recourse to statistics in determining cause and manner of death is likely to mislead.
    - There are no specific signs of asphyxia at autopsy, and defaulting to a diagnosis of asphyxia is flawed. There are however circumstances where recognisable patterns associated with an asphyxiating event may be present.
    - The term "consistent with" should be avoided in the legal context because of the potential to mislead.

- Manner of death (natural, accident, homicide, suicide) should not be substituted for cause of death. In the Folbigg matter, *"a tacit judgment about a homicidal manner of death has informed the decision about the cause of death."*
- There is no forensic pathology evidence in these cases to sustain a determination of homicidal mechanical asphyxia.
- The views of Dr Cala at the time he gave his evidence were not opinions outside the norm of forensic pathologists (in America) at the time.
- There are no positive pathological findings in relation to the deaths of Caleb and Sarah.
- Myocarditis is definitely present in Laura. It is a well-recognised cause of unexpected natural death.
- Patrick has significant brain damage and a history of a seizure disorder. Sudden unexpected death in epilepsy (or SUDEP) can explain this death.
- *"A homicidal conclusion for the Folbigg case in toto is not supported by the medical evidence."*

42. **REPORT OF Dr CALA DATED 26 NOVEMBER 2018:** In his additional report, Dr Cala makes a number of comments:
    - Some doctors do not believe SIDS "runs in families" while others do; some question whether a diagnosis of SIDS can occur twice or even more in a family, whist others hold this view.
    - SIDS has recently fallen into disuse in favour of sudden unexplained death in infancy or SUDI.
    - Caleb: There was blood and froth around Caleb's mouth prior to any attempted resuscitation. When taken together with other factors, the death is not satisfactorily explained by SIDS.
    - Patrick: *"Although I cannot definitely prove smothering has occurred, I am concerned that smothering could explain Patrick's ALTE and his sudden death."*
    - Sarah: The cause of death should not be given as SIDS because of the previous two death, and the abnormality of the uvula.
    - Laura: Myocarditis does not adequately explain this child's death. Smothering remains a possible explanation for her sudden death.
    - It is almost certainly the case that if any of the Folbigg children had died in 2018 that DNA testing would have been performed.
    - Smothering of infants can result in a range of abnormalities, including no injuries.
    - Making a diagnosis of SIDS twice in one family (with an intervening ALTE related death) is "both astonishing but very troubling."

- *It remains my opinion 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."*

43. **GENETICS TESTING:** There was consideration of a genetic or inherited cause of the deaths from February 1991 onwards, i.e after the death of Patrick. Essentially, multiple tests available between 1991 and 2003 were performed, and no definite genetic abnormality, inborn error of metabolism or other condition was identified by geneticists which have been shown to be associated with familial sudden death.

44. Further genetic testing in the form of whole exome sequencing analysis was performed in 2018 on a specimen of Kathleen's DNA, and in a report dated 2 December 2018 Professor Vinuesa identified two genetic abnormalities associated with cardiac pathology:
    - A novel mutation in the CALM2 gene. Previously reported mutations in this gene have been associated with Long QT syndrome and sudden death in infancy and childhood.
    - A very rare mutation in the MYH6 gene. Previously reported mutations in this gene have been associated with a variety of cardiac abnormalities, including atrial septal defect, hypertrophic cardiomyopathy, sick sinus syndrome and fatal cardiac arrhythmia.

    I understand that further genetic testing is being conducted on specimens of the four children, but the results of those tests are not available at the time of writing of this report.

45. **CRAIG FOLBIGG:** Craig has reportedly been diagnosed with obstructive sleep apnoea and at the time of the events in question was a smoker. I understand that he limited his smoking to outdoors.

OPINIONS

46. I have been asked a series of questions in the Letter of Instruction, which I transcribe and answer as follows:

1. **Provide an opinion on the standard of the autopsies conducted in each case bearing in mind best practice at the time of each autopsy. ANSWER:** I have practised in Australasia as a specialist forensic pathologist since November 1988, and was employed as a staff specialist forensic pathologist at the time of the deaths of all four Folbigg children. There is no doubt that during that time there have been many changes in procedures relating to the investigation of infant deaths, including which cases should be referred for autopsy to a specialist forensic pathology centre (at the time in question, Glebe and Westmead), who should perform the autopsies, the extent of autopsy investigation conducted, the nature of the death scene examination, the range of ancillary tests performed, the extent of documentation, the means by which cause of death is determined, and which cases should be peer reviewed. I do not have a detailed recollection of the chronology of the various changes between 1989 and 1999, but it is possible that some documentation detailing various policies and standards relating to infant death investigation during this time may still be held by NSW Health, the Department of Forensic Medicine, and the Sudden Infant Death Advisory Committee (SIDAC) convened by the NSW State Coroner.

   I also recollect that in the early 1990's there was a country wide research project led by Professor Cordner (with Professor Hilton and I listed as co-researchers) and funded by the National SIDS Council of Australia which aimed to standardise the autopsy assessment of sudden infant deaths. Included in this project was the production of a protocol/minimum standard for the examination of all suspected SIDS cases, the National SIDS Autopsy Protocol, with use of that protocol commencing in NSW in around 1992 to 1993.

   The Department of Forensic Medicine at Glebe obtaining NATA/RCPA accreditation in around mid-1993. This accreditation would have included the production of multiple documents detailing minimum standards and record keeping requirements, and adherence to those standards would have been mandatory. I do not recollect if there were specific requirements in relation to infant death investigations at the time of that initial NATA/RCPA accreditation process.

Taking into account my limited recollection of autopsy standards and protocols
I consider the four autopsies as follows:

Caleb: The autopsy was done by Dr Cummings at the Newcastle Morgue. Dr Cummings was a specialist pathologist, and appointed as a "government medical officer" at the time. He would have done a considerable number of infant death autopsies in accordance with procedures in place at the time. There would have been no requirement to transport the body to Glebe, and had this taken place it would have been viewed as a most unusual action at the time. I am not aware of any detailed requirements for doing such autopsies, but it is my recollection that the majority of autopsies performed on suspected SIDS cases were done to a standard well below that of Dr Cummings, considering that at the time such autopsies were generally done in non-metropolitan Sydney by government medical officers with no specialist training in pathology. It is my opinion that the standard of the autopsy on Caleb, as documented, was that of forensic pathologists practicing in metropolitan Sydney at the time, noting that many investigations, including microbiological and viral testing, and microscopy on the brain, could have been done without much difficulty. I have previously examined the microscopy slides relating to this case, and consider the pathological descriptions as concordant with the observed microscopy. Haemosiderin in alveolar spaces was subsequently identified on special staining of the lung slides but I concur with Professor Byard that the presence of stainable iron is not indicative of cause of death in infants. The report documenting the autopsy findings is brief, but assuming all pathological processes have been documented, I am of the overall view that the autopsy has been performed to an adequate standard relevant at the time in question.

Patrick: The autopsy on Patrick was done as a non-coronial investigation, with the autopsy consented to by Patrick's parents. In my opinion, it is not unreasonable that a death certificate was issued in this matter, although it would have been my recommendation that the case be referred for coronial investigation and that a death certificate not be issued given a degree of uncertainty in relation to the cause of the ALTE and the cause of death. The autopsy was performed by Drs Bishop and Singh-Khaira, a specialist pathologist and a registrar in pathology respectively at the time. The brain microscopy slides were further examined by Dr Kan, a paediatric pathologist. The death was not considered a sudden unexpected infant death at the time, and as such any SIDS protocol operational at the time

would not necessarily have applied. Overall, the autopsy appears to have been done to a standard which was typical of "hospital" (or non-coronial) autopsies at the time, with detailed examination of the brain well in excess of what would usually be expected in a SIDS type autopsy at the time. Further, multiple samples were retained for various metabolic, genetic, viral and other investigations, likely in excess of what would be usually expected at the time in an autopsy of a case of sudden infant death. In summary, I am of the view that the autopsy has been conducted to an adequate standard operational at the time, and in some respects the autopsy significantly exceeded that standard.

Sarah: The National SIDS Autopsy Protocol was in operation at the time of the autopsy on Sarah and was a standard which was expected to be followed by all forensic pathologists at the Department of Forensic Medicine. However, a degree of latitude based on circumstances relevant to the case was acceptable, as long as any deviation from the standard could be justified by the autopsy pathologist. In this case, the Protocol was largely followed, with the one major exception being that it appears no x-rays were taken prior to autopsy. Professor Hilton has given an explanation for his deviation from the standard – there was no indication of trauma to the body, and direct visualisation of skeletal structures typically affected by compression of an infant such as the ribs is more reliable than x-rays. I agree with those comments of Professor Hilton, but am also of the view that x-rays of the body would have been a prudent test to perform as part of the autopsy. I have previously examined the microscopy slides relating to this case, and consider the pathological descriptions as concordant with the observed microscopy. Overall, appropriate ancillary testing has been performed and if the assumption is made that all pathological processes have been documented in the report, I am of the overall view that the autopsy has been performed to an appropriate standard relevant at the time in question.

Laura: In common with the autopsy on Sarah, this autopsy was conducted at the time of the National SIDS Autopsy Protocol, acknowledging that Laura was not an infant at the time of death and that the SIDS protocol would therefore not strictly apply in this case. Nevertheless, the standard of this autopsy was done in broad accordance with the Protocol again assuming that all pathological processes have been documented in the report, and all relevant ancillary examinations have been performed. I do not necessarily agree with Dr Cala that retention of the heart for further examination would have caused undue distress to the parents given he had retained the brain and dissected the face, but I accept as well that the

heart appeared normal macroscopically, and that routine examination of the cardiac conduction system was not done by all pathologists in paediatric sudden death cases at the time in question; subsequent publication of the TRAGADY guidelines recommended such examination.[6] I have previously examined the microscopy slides relating to this case, and am of the view that there is moderately extensive myocarditis characterised by mixed inflammatory, mainly lympho-histiocytic in appearance in many areas of the myocardium, with associated focal myocyte necrosis and degeneration of myocytes. In my opinion, the appearances are in accordance with that described in the autopsy report of Dr Cala, and I also agree with Dr Cala that most likely the cause of the myocarditis is a viral infection. I also clearly recall having seen the initial heart microscopy slides of the case at the time Dr Cala first received them, and having expressed a view in front of multiple other forensic pathologists present in the registrar's room at the time that there was no doubt that there was highly significant myocarditis present in this case.

I therefore am of the opinion that the autopsy has been done to a standard in broad accordance with the requirements of the National SIDS Autopsy Protocol, accepting that the Protocol would not apply in this case.

Although I am of the view that the autopsy on Laura was done in accordance with the protocol, and that specifically the microscopy description was in accordance with my observations and it appears those of other experts, there appears to have been a significant change of opinion by Dr Cala in relation to the severity of the cardiac pathology in the time between the report being issued and his evidence at the trial. Specifically, Dr Cala stated in his verbal evidence at the Trial, *inter alia* that the inflammation was *"quite patchy and rather mild in the sense that although the inflammation existed it was of a rather low amount…"* In my opinion, there is at least moderately extensive inflammation of the heart, with necrosis of myocytes, and I would definitely not characterise the inflammation as patchy and mild.

2. **Provide your opinions about the causes of death in each case. In providing these opinions, also list the factual matters you rely upon and the assumptions that you make. ANSWER:** I consider each case individually as follows:

[6] Skinner J, Duflou J, Semsarian C. Reducing sudden death in the young in Australia and New Zealand: the TRAGADY initiative (Editorial). Medical Journal of Australia 2008;189:539-540.

Caleb: The history provided is one of a sudden death during sleep, at some time between 22:00 hours and 02:45 hours during the night of 19/20 February 1989. Caleb had been sleeping in his own bed, but no further details of the sleep environment are provided. He was aged 19 days at the time of death. There was a prior medical history of congenital laryngeal stridor which was considered to almost certainly be on the basis of laryngomalacia, and a diagnosis made on chest x-ray after birth of possible pneumomediastinum. A small amount of blood-stained froth was observed by Kathleen around Caleb's mouth at the time she found him. The autopsy was recorded as showing no abnormalities, with the exception of some incomplete aeration of lung spaces and areas of intra-alveolar haemorrhage, and subsequent identification of haemosiderin in alveoli. The larynx and upper airway were described as unremarkable. No injuries were identified. Toxicology was negative.

Time of death has not been formally assessed at autopsy or by ambulance officers in this case but I note Caleb was described as cold to touch by Kathleen and either warm or cold to touch by ambulance officers. He also was observed to have a large quantity of curdled milk in his stomach at the time of autopsy. The time taken for the stomach to empty in infants is variable and a complex topic of investigation with variations dependent on multiple factors, but in general gastric emptying time of 1 to 2 hours is not unreasonable.[7] When taken together with the body temperature descriptions it appears to me very likely that Caleb died some short time after he was checked by his mother at 22:00 hours, and likely not around the time he was next checked by Kathleen at 02:45 hours.

In my opinion, the cause of death in this case can be reasonably attributed to **SUDDEN INFANT DEATH SYNDROME, CATEGORY 2**, where I use the SIDS definition of Krous et al[8] as follows *"The sudden unexpected death if an infant less than 1 year of age, with onset of the fatal episode apparently occurring in sleep, that remains unexplained a thorough investigation, including performance of a complete autopsy and review of circumstances of death and the clinical history"* and I subclassify the death as Category 2 on the basis of age at death below 21 days, and a history of a neonatal condition in the form of congenital laryngeal stridor which was considered to almost certainly be on the basis of laryngomalacia

[7] Gridneva Z, Kugananthan S, Hepworth A, et al. Effect of human milk appetite hormones, macronutrients and infant characteristics on gastric emptying and breastfeeding patterns of term fully breastfed infants. Nutrients 2017;9:5.

[8] Krous H, Beckwith J, Byard R, et al. Sudden Infant Death Syndrome and Unclassified Sudden Infant Deaths: A Definitional and Diagnostic Approach. Pediatrics 2004;114:234-238.

(although not specifically identified at autopsy). I note that contrary to evidence adduced during the Trial, severe laryngomalacia is a known cause of sudden death in infancy, with death having been attributed to either upper airway obstruction or heart failure in reported cases.[9] Additionally, Category 2 SIDS can be given where there is death of siblings while in custody of the same caregiver, noting that of course Caleb's siblings died after he did.

Patrick: Patrick died at 8 months. There was a normal pregnancy, and he was investigated after birth for risk factors for sudden death. These investigations were normal except for a barium swallow which suggested uncoordinated swallowing. At four months, Patrick was observed to cough and gasp during sleep following which he turned blue. CPR was commenced and he appeared to recover. Patrick was taken to hospital where he was extensively examined. There were no injuries. Possible bronchiolitis was diagnosed. While in hospital, Patrick had a generalised seizure and detailed further investigations were conducted, leading to a discharge diagnosis of seizure disorder probably due to viral encephalitis. Following discharge, Patrick's neurological function continued to deteriorate and he was admitted a number of times to hospital for control of seizures. On the day of his death, Patrick had a fever and during the day he had a cardiorespiratory arrest. Resuscitation was unsuccessful, and a death certificate was issued giving the cause of death as ASPHYXIA due to AIRWAY OBSTRUCTION due to or as a consequence of EPILEPTIC FITS.

The non-coronial autopsy identified severe brain damage in the form of laminar necrosis, gliosis and a leptomeningeal chronic lymphocytic infiltrate. No injuries were identified. The time of death was not assessed, but I note he was described as having a normal skin temperature by ambulance officers. Stomach contents was not described in the autopsy report. The cause of death, as determined by the autopsy pathologists, was given as ASPHYXIA due to AIRWAYS OBSTRUCTION due to or as a result of EPILEPTIC FITS due to or as a result of ENCEPHALOPATHIC DISORDER (UNDERLYING CAUSE NOT DETERMINED ON INVESTIGATION).

Time of death is not able to be determined from the information provided, but 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 Craig went to work to when the ambulance officers arrived, with a later time more likely than an early time.

[9] Baxter M. Congenital laryngomalacia. Canadian Journal of Anaesthetics. 1994;41:332-339.

In my opinion, the events leading up to the sudden collapse at age 4 months can be reasonably termed an acute life threatening event, or ALTE, which I define as "an episode that is frightening to the observer and that is characterised by some combination of apnea (central or occasionally obstructive), color change (usually cyanotic or pallid but occasionally erythematous or plethoric), marked change in muscle tone (usually marked limpness), choking, or gagging." [10]

The table reproduced below gives a list of causes of ALTE:[11]

Table 3 Differential diagnosis of apparent life-threatening event

Cardiac	Metabolic
Congenital heart disease	Inborn errors of metabolism
Arrythmia (long QT syndrome, WPW)	Reye's syndrome
Myocarditis	Nesideoblastosis
Cardiomyopathy	Hypocalcemia
	Hypomagnesemia
	Hypoglycemia
Child abuse	Neurologic
Munchausen syndrome by proxy (suffocation, intentional salt poisoning, medication overdose, physical abuse, head injury)	Malignancy
	Seizure disorder
	Febrile seizure
Smothering (unintentional or intentional)	Congenital brain malformations
	Craniostenostosis
	Hydrocephalus
	Central apnea
	Ventriculoperitoneal infection
	Neuromuscular disorders
	CNS bleeding
Gastrointestinal	Other
Gastroesophageal reflux	Developmental delay
Volvulus	Feeding difficulties
Intussusception	Medication
Laryngeal chemoreflex	Hypothermia
Gastroenteritis	Anemia
Incarcerated hernia	Idiopathic
	Anaphylaxis
	Food allergy
Infectious	Respiratory
Sepsis	Anatomical airway obstruction
Urinary Tract Infection	Infections of the respiratory tract
Upper Respiratory Tract Infection	Periodic breathing
Encephalitis/Meningitis	Breath holding spell
Pneumonia	Choking episode
	Foreign body

A cause of ALTE, including a cause of sudden deterioration of a person, with potential to cause sudden death, and listed in the table above is various reflex causes, including the gastro-oesophageal reflex and the laryngeal chemoreflex. In general, these can also be characterised as forms of vagal arrest where as a result of stimuli transmitted to various nerves in the body, including the vagus nerve, the trigeminal nerve and the glossopharyngeal nerve, a reflex cardiac arrest is mediated. In general, most of the forensic pathology research in this area is related to death due to neck compressions and the various

[10] Tieder J, Altman R, Bonkowsky J, et al. Management of Apparent Life-threatening Events in infants: a systematic review. Journal of Pediatrics 2013;163:94-9.
[11] Shah S, Sharieff G. An update on the approach to apparent life-threatening events. Current Opinions in Paediatrics 2007;19:288-294

mechanisms whereby it is achieved, but other medical specialties including anaesthetists and ENT surgeons are also exposed to the dangers of cardiac arrest following introduction of irritants to the upper airways.[12] Whether this applies to the events leading up to Patrick's ALTE, or the death of any of the other children is of course speculative, but there certainly is evidence that aspiration of gastric contents can be both distressing to observers and at times have profound consequences on the cardiovascular and neural physiology of an infant.

The events in the days leading up to the death of Patrick appear largely unremarkable with the possible exception of fever on the day of his death. The autopsy again identified no injuries on the body. There was severe brain pathology, typical of one or more episodes of hypoxia-ischaemia some time in the past. No definite cause for the brain damage was identified, although again there were concerns in relation to prior encephalitis. In my opinion, the cause of death in this case can be reasonably given as **THE CONSEQUENCES OF HYPOXIC-ISCHAEMIC ENCEPHALOPATHY BROUGHT ON BY AN ACUTE LIFE-THREATENING EVENT (ALTE) OF UNASCERTAINED CAUSE**.

Sarah: Sarah died at the age of 10 months. Pregnancy was normal, and her post-natal course was largely unremarkable with the exception of a sleep test done at 3 weeks of age which showed showing very few sleep apnoeas, some periodic breathing, and quite long episodes of hypoventilation. Subsequent sleep studies were normal. Sarah had a cold-like illness in the weeks leading up to death and she was heard to snore during sleep on the night of her death. She slept in her own bed. Kathleen found Sarah unresponsive, not breathing, blue in colour and cold to touch. Despite attempted resuscitation by Craig, paramedics and hospital staff, Sarah could not be revived.

An autopsy revealed the presence of a 1.5 cm scratch on the antero-lateral aspect of the right upper arm, and two tiny punctate abrasions, one immediately below the lower lip on the left side, the other slightly to the left side of the midline of the chin. No injuries to the mouth were identified. There was an abnormally placed congested/haemorrhagic uvula located anterior to the epiglottis producing an obstructive element in the airway. Petechiae were observed on the surfaces of the organs of the chest. Microscopy showed a degree of upper and lower airway inflammation and limited lymph node enlargement. Further

[12] Schrag B, Vaucher P, Bollmann M, Mangin P. Death caused by cardioinhibitory reflex: a systematic review of cases. Forensic Science International 2011;207:77-83..

ancillary tests were non-contributory. Professor Hilton gave the cause of death as SUDDEN INFANT DEATH SYNDROME.

Time of death has not been formally assessed as part of the ambulance attendance or autopsy, but Sarah's skin temperature was described by ambulance officers 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. Again, taking into account the temperature of the body and the stomach contents, this would suggest that Sarah died closer to the time she was put to bed by Craig at around 21:00 hours, rather than when found by Kathleen at around 01:30 hours. I therefore disagree with Professor Hilton where he states in his autopsy report that the time of death is 01:30 hours on 30 August 1993, noting this was the time of death proffered by Senior Constable Saunders in the Form P79Apolice in the . I am also of the opinion that the formal time of death given by Maitland Hospital staff of 04:30 hours can be considered as being the time when death was declared, and not the time death occurred.

In my opinion, Sarah's cause of death can be reasonably attributed to **SUDDEN INFANT DEATH SYNDROME, CATEGORY 2**, using the definitions of SIDS by Krous et al. I subclassify the death as Category 2 on the basis of age at death greater than 9 months, and an autopsy diagnosed abnormality of the upper airway which has the potential to obstruct the flow of air (the unusually located uvula). Additionally, Category 2 SIDS can be given where there is death of siblings while in custody of the same caregiver. It is my opinion that the two punctate abrasions below the mouth are entirely non-specific and not in any way indicative of external obstruction of respiration; for example it would be totally normal for a 10 month old infant to cause minor injuries to their face during normal day-to-day activities. Further, the presence of intra-thoracic petechiae is typical of Sudden Infant Death Syndrome, although not specific of that condition.

Laura: Laura died at 20 months of age. Pregnancy was normal, and multiple investigations were performed in the months after birth to exclude causes of sudden infant death. A sleep study in the first two weeks of life was reported as showing mild central apnoea but no evidence of obstruction or bradycardia and a second sleep study at 8 weeks was reported as showing moderate central apnoea of infancy with a mild improvement since the previous study, although an obstructive component was now documented. A third sleep study at 6

months was reported as normal. Laura had an apparent respiratory tract infection in the days leading up to her death and was being administered Demazin. She appears to have had a sudden cardiorespiratory arrest during the day of 1 March 1999, with bradycardia reported as the initial heart rhythm during resuscitation. Despite attempted resuscitation by Kathleen, paramedics and hospital personnel, Laura did not respond and she was declared dead at 12:45 hours.

At autopsy, minor, likely old, "toddler" injuries in the form of discoloured bruises were identified on the lower extremities. No injuries to the face or mouth were identified despite extensive dissection of these structures. Pathology in the form of myocarditis likely the result of a viral infection was identified on microscopic examination of the heart, and changes in the lungs, spleen and lungs were all considered consistent with a recent viral infection. Ancillary tests were negative, including specific toxicological testing of blood for chlorpheniramine. Dr Cala gave the cause of death as UNDETERMINED.

Time of death in this case appears to be at the time of cardiac standstill, which would have been during the treatment provided by paramedics and hospital personnel.

In my opinion, there is without doubt myocarditis of a severity which can readily cause sudden and unexpected death. As indicated by multiple pathologists, myocarditis can either cause death through the gradual development of congestive cardiac failure, or can result in a sudden onset lethal cardiac arrhythmia without evidence of prior illness in the patient. I have seen multiple examples of both presentations in my autopsy practice. I have also co-authored multiple papers where death due to myocarditis has been examined,[13] [14] [15] and in common with many other authors have concluded that myocarditis can be both incidental to death and causative of death.[16] [17] [18] Attempts at differentiating the two on the basis of naked

[13] Krishnamohan S, Fang B, Bao S, Duflou J, Puranik R. Significance of myocardial inflammation identified at autopsy: a 10 year retrospective review. Experimental and Clinical Cardiology 2014;20:5707-5712.
[14] Puranik R, Chow C, Duflou J, Kilborn M, McGuire M. A 10 year review of sudden death in young Australians. Heart Rhythm 2005;2:1277-1282.
[15] Bagnall R, Weintraub R, Ingles J, Duflou J, Yeates L, Lam L, Davis A, Thompson T, Connell V, Wallace J, Naylor C, Crawford J, Love D, Hallam L, White J, Lawrence C, Lynch M, Morgan N, James P, DuSart D, Puranik R, Langlois N, Vohra J, Winship I, Atherton J, McGaughran J, Skinner J, Semsarian C. Sudden cardiac death in the young: results from a prospective population-based bi-national registry. New England Journal of Medicine 2016;374:2441-2452.
[16] Busuttil A, Keeling J. 2008. Paediatric Forensic Medicine and Pathology. CRC Press, Boca Raton FL.
[17] DeSa D. Isolated myocarditis as a cause of sudden death in the first year of life. Forensic Science International 1986;30:113-117.
[18] Thiene G, Corrado D, Basso C. 2016. Sudden cardiac death in the young and athletes. Springer-Verlag, Milan.

eye and microscopic appearances of the heart has been attempted,[19] but in general both severe myocarditis can be incidental while relatively mild myocarditis can readily cause death, and grading schemes have not gained widespread acceptance.

In this case, no competing cause of death has been identified at autopsy, therefore based purely on the autopsy findings, the cause of death would be given as **MYOCARDITIS**. 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 the deceased not being obviously short of breath in the days leading up to death.

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.

3. **Have there been scientific advances since 2003 that would have changed the way the autopsies were conducted? ANSWER:** In general terms and at a superficial level, the autopsy conducted today is very similar to that of the autopsy of 10 to 20 years ago. In some ways, the standards for the autopsy examination of death in Australia have gone backwards during that time, in that there is a greater reliance on external examinations and partial autopsies, organ retention for the purposes of detailed examination of organs such as the heart and brain is now infrequently done, and greater reliance is placed on imaging modalities such as whole body post mortem CT scans. This has however generally not been the case in sudden death in infancy and the young, and if anything, the autopsies in those cases, together with additional testing have becoming significantly more comprehensive and detailed.

   Specific advances which have revolutionised the investigation of sudden death in the young, as also detailed by Professor Cordner and Dr Cala, revolve around the investigation of

[19] Casali M, Lazzaro A, Gentile G, et al. Forensic grading of myocarditis: an experimental contribution to the distinction between lethal myocarditis and incidental myocarditis. *Forensic Science International* 2012;223:78-86.

familial, genetic and metabolic causes of sudden death. This has involved the use of multidisciplinary teams, often based in cardiology and genetics units, investigating blood relatives of the deceased over multiple generations, performing a range of clinical investigations on those relatives, and where appropriate progressing to various forms of genetic testing.

In the early 2000's, very few genetic tests which detected specific sudden death mutations were available, and these were invariably prohibitively expensive, difficult to interpret, and generally of limited value. Generally, testing of a genetic nature related to specific disease conditions such as hypertrophic cardiomyopathy and various storage disease, and DNA testing was not done as a routine and was at best only available as a research tool. Today, whole exome and whole genome testing is considered the norm in the investigation of possible familial sudden death, especially in cases where the autopsy is negative. The results of genetic abnormalities on DNA testing remain difficult to interpret in a large percentage of cases, and there is the added problem that many of the cases where a genetic abnormality is strongly suspected on clinical grounds is unable to be identified despite state-of-the-art technology and clinical expertise.

Multiple scientific papers have been published on the topic of DNA testing for channelopathies and similar conditions in sudden death in babies, including one in which I am a co-author.[20] [21] [22] [23] [24] Overall, it appears likely that a genetic defect capable of causing death in infancy is present in about 10% of cases where the conventional autopsy diagnosis is SIDS. The "molecular autopsy" has become an established concept in relation to the investigation of death in the young where no cause of death is evident, and there are certainly experts in the field who suggest that such an examination should be a component of any competently conducted sudden infant death autopsy where no lethal morphologic abnormalities are detected. The paper by Sweeting and Semsarian referenced above takes a

[20] Evans A, Bagnall R, Duflou J, Semsarian C. Postmortem review and genetic analysis in sudden infant death syndrome (SIDS): An 11-year review. Human Pathology 2013;44:1730-1736.
[21] Arnestad M, Crotti L, Rognum T, et al. Prevalence of Long QT syndrome gene variants in sudden infant death syndrome. Circulation 2007;115:361-367.
[22] Tester D, Ackerman M. Postmortem long QT syndrome genetic testing for sudden unexplained death in the young. Journal of the American College of Cardiology 2007;49:240-246.
[23] Gando I, Yang H, Coetzee W. Functional significance of channelopathy gene variants in unexplained death. Forensic Science Medicine and Pathology 2018 DOI 10.1007/s12024-018-0063-y.
[24] Sweeting J, Semsarian C. Cardiac abnormalities and sudden infant death syndrome. Paediatric Respiratory Reviews 2014;15:301-316.

somewhat more measured approach to testing but does emphasise the importance of targeted screening and management of families in subsequent pregnancies where there has been a previous history of SIDS.

Additionally, a greater emphasis has been placed on standardisation of autopsies. In addition to the National SIDS Autopsy Protocol mentioned above, NSW Health has issued a number of policy directives which detail the management and investigation of cases of sudden unexpected death in infancy, including various expected clinical pathways, information required prior to conducting the autopsy, and various autopsy requirements.[25] I note that the PD2008_070 is scheduled for review on 1 April 2019, but I am not aware of any proposed modifications to the Directive at this time.

In summary, had the autopsies been done today, in all likelihood they would have been conducted in greater detail, with greater knowledge of causes of sudden death by the autopsy pathologists informing them of the tests available at this time. Very likely, "molecular autopsies" would have been conducted by the time of the second death, and a multidisciplinary team which included cardiologists, geneticists, laboratory scientists and genetics counsellors would have been involved in the detailed and ongoing management of the family.

4. **Is there any evidence that would reasonably allow a forensic pathologist to conclude that any of the children had been smothered? ANSWER:** Smothering (sometimes termed suffocation) is variably defined but in my opinion a reasonable working definition is asphyxia caused by the mechanical obstruction or occlusion of the external airways, i.e the nose and mouth. According to some classifications, it is a form of mechanical asphyxia, and some authorities will also use the term suffocation and smothering interchangeably. For the purposes of completeness, I define asphyxia as a lack of oxygen to vital parts of the body – in general, the term has little utility, in that it gives no indication of cause of death, mechanism of death, mode of death or manner of death. Sauvageau and Boghossian describe some of the difficulties in relation to the various classifications of asphyxia, noting that multiple forensic pathology text books give different classifications of asphyxia and that all of them

[25] NSW Health. PD2008_070 Death – management of sudden unexpected death in infancy. Accessed 31 January 2018 at https://www1.health.nsw.gov.au/pds/ActivePDSDocuments/PD2008_070.pdf

have inadequacies.[26] I note that Professor Cordner identifies similar difficulties with the definitions of asphyxia and in common with my views does not advocate use of the word as a cause of death. I hold similar views in relation to the phrase "catastrophic asphyxiation event" and variations on that term, which I have similarly not been able to identify as a cause of death except when used in the context of the Folbigg Trial – the term is not one which is used by the medical fraternity to describe a cause of death.

There are differing views in relation to the autopsy signs of smothering. Many authorities including DiMaio and DiMaio[27], Sauko and Knight[28] and Collins and Byard[29] are of the view that smothering often leaves no signs of violence to the body, while others such as Dolinak et al[30] are of the view that injuries to the face, nose and mouth are frequently present, and Professor Cordner in his report identified injuries to the face and/or mouth in 3 out of 5 cases of confirmed infant smothering.

I am of the opinion that a diagnosis of death due to smothering can only be made in the setting of a negative autopsy in the presence of a positive history of it having taken place (for example a verified confession, eye witness evidence, or video footage of the act), or where there is autopsy evidence of external obstruction of the airway such as the presence of facial or conjunctival petechiae, injuries to the lips, the frenula, bruising to the mouth and face, injuries to the nose, and injuries to the chin of a type which would be expected in compressive force applied to that part of the body.

In the case of the four Folbigg children, I emphasise that no such injuries of any type are seen in any of the children, and as such I am of the opinion that a diagnosis of smothering cannot be reasonably suggested by the expert in court proceedings.

5. **What definition of SIDS do you rely upon? Please detail the definition. ANSWER:** I rely on the following schema for the determination of cause of death in infants who have died

[26] Sauvageau A, Boghossian E. Classification of asphyxia: The need for standardization. Journal of Forensic Sciences 2010;55:1259-1267.
[27] DiMaio V, DiMaio D. 2001. Forensic Pathology, 2nd edition. CRC Press, Boca Raton.
[28] Saukko P & Knight B. 2004. Knight's Forensic Pathology, 3rd edition. Arnold Publishing, London.
[29] Collins K, Byard R. 2014. Forensic pathology of infancy and childhood. Springer, New York.
[30] Dolinak D, Matshes E, Lew E. 2005. Forensic pathology: Principles and practice. Elsevier Academic Press, Burlington MA.

suddenly and unexpectedly, used by NSW Health, based on work of Krous and others, and as discussed in the paper by the same author:[31]

General SIDS Definition: Sudden unexpected death of an infant under 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.

SIDS IA: Older than 21 days and younger than 9 months, normal clinical history, term pregnancy, normal growth and development, absence of similar deaths among siblings, close genetic relatives, or other infants in the custody of same caregiver, investigation of the various scenes do not explain, safe sleep environment, potentially fatal pathologic findings at autopsy, No evidence of unexplained trauma, abuse, neglect or unintentional injury. toxicologic, microbiologic, radiologic, vitreous chemistry, and metabolic screening studies negative.

SIDS IB: General definition and category IA criteria met except investigation of various scenes not performed and/or 1 of following analyses not performed: toxicology, microbiology, radiology, vitreous chemistry, or metabolic screening.

SIDS II: Age 0-21 days or greater than 270 days [9 months], similar deaths among siblings, close relatives, or other infants in custody of same caregiver not considered suspect for infanticide or recognized genetic disorders, neonatal or perinatal conditions resolved by time of death, mechanical asphyxia or suffocation caused by overlaying not determined with certainty, autopsy may show abnormal growth and development not contributing to death, inflammation or abnormalities not considered sufficient to be unequivocal causes of death may be present.

Unclassified Sudden Infant Death: Criteria for category I or II SIDS not met but alternative diagnoses are equivocal, including cases for which autopsies were not performed.

[31] Krous H. Sudden unexpected death in infancy and the dilemma of defining sudden infant death syndrome. Current Pediatric Reviews 2010;6:5-12.

Not unlike the difficulties in diagnosing death due to SIDS is the difficulty in diagnosing death due to epilepsy, especially in the setting of sudden unexpected death in epilepsy (or SUDEP). To a very large extent, these deaths are 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. Added to this, there have been many suggested links and interactions between the brain and heart in epilepsy, making assessment of cause of death in these cases very complex and an area of evolving scientific investigation with familial and many ill-defined conditions likely playing a role in death.[32] [33]

6. **In cases where SIDS is found, is there any basis for a forensic pathologist to link such a finding to a lack of oxygen? ANSWER:** The cause of SIDS is not known, but in all likelihood a major mechanism for a percentage of cases is a vulnerability of the infant which causes interruption in normal respiration/breathing. This is not based on autopsy findings, but rather based on the presence of petechiae within the chest and a known association with dangerous sleeping environments capable of obstructing airways, both of which are seen in "typical" SIDS, together with various research findings indicating various subtle (and contested) changes in the brainstem. There are however no specific pathological features to identify a lack of oxygen at autopsy.

   Considered from a different perspective, are there specific autopsy signs of asphyxia? To a very large extent this question has been considered by Professor Cordner in his report, and I agree with his comments and observations in that respect. Historical 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 expressed in publications since 1949.[34] Saukko and Knight state the following:

   > *"It cannot be emphasised too strongly that the mere finding of any of the non-specific features, such as congestion and petechiae, without firm circumstantial or preferably*

[32] Tu E, Bagnall RD, Duflou J, Semsarian C. Post-mortem review and genetic analysis of sudden unexpected death in epilepsy (SUDEP) cases. Brain Pathology 2010;21:201-208.
[33] Panayiotopoulos C. 2010. A clinical guide to epileptic syndromes and their treatment (2nd ed). Springer Healthcare.
[34] Saukko & Knight. 2004.

physical evidence of mechanical obstruction of respiration, is quite insufficient to warrant a speculative diagnosis of asphyxia. If such collateral evidence is not forthcoming, then the cause of death must be left undetermined." (page 357).

7. **Is there any evidence that would allow a forensic pathologist to conclude that any of the children died as a result of a lack of oxygen? ANSWER:** No.

8. **Are there any opinions provided by Professor Cordner in his report that you disagree with? If there is, please provide details and reasons for your disagreement. ANSWER:** In general terms, I am in agreement with the observations and conclusions made by Professor Cordner in his report. With reference to the cause of death of the four children, as detailed in my response to question 2 above, I have some minor differences of opinion in relation to the wording of the cause of death of Patrick: Professor Cordner has proffered "The consequences of an unexplained ALTE", while I have suggested "The consequences of hypoxic-ischaemic encephalopathy brought on by an acute life-threatening event (ALTE) of unascertained cause" – in my opinion, the differences in this cause of death can be attributed mainly to stylistic preferences and not to any material differences in opinion.

   With reference to Laura, Professor Cordner has given the cause of death as myocarditis, stating in his report:

   > ***"****I believe the middle of the road conclusion in relation to Laura's death is that considered alone, most forensic pathologists would be comfortable ascribing the death in similar circumstances to Laura's as being due to myocarditis. This is indeed my own view. It would have been acceptable, and I would support a pathologist who gave the cause of death as "I(a): Undetermined", but in the comments section of the report, fully canvassed the possibilities that death could be due to myocarditis, but because it was the 4th death in the particular family there could be other factors, including but not limited to homicide, at work."* (page 80).

   I fully endorse this view, and in my last two paragraphs of my response to question 2 above I believe I have expressed a largely similar opinion. In common with Professor Cordner, I have researched the issue of causative versus incidental myocarditis, and have had a scientific paper published on the topic.[35] In that research study, we examined the circumstances and

[35] Krishnamohan S, et al. Significance of myocardial inflammation… Experimental and Clinical Cardiology 2014.

pathology of cases of confirmed myocarditis diagnosed at autopsy at the Department of Forensic Medicine at Glebe over a 10 year period. There were 143 cases (out of 18,705 autopsies during that time period) with myocarditis being considered the cause of death in 87 cases (60.7%), and incidental to the cause of death in 56 cases (39.3%). In our study, we were unable to find strong markers which allowed differentiation of the two groups purely by examination of the heart, but we did find that the heart was generally heavier in cases of fatal myocarditis, and a lymphocytic infiltrate was more common in fatal myocarditis. None of the other data points reached statistical significance. This may be of some relevance in the case of Laura, where the inflammation was also predominantly lymphocytic in type, and previous research has shown that this form of myocarditis tends to have a worse prognosis than other forms of myocarditis.[36]

I believe my opinions of the cause of death of both Caleb and Sarah mirror those of Professor Cordner.

Professor Cordner and I have for many years disagreed on the utility of *pro forma* type autopsy reports. The report style of the National SIDS Autopsy Protocol is in my view an example of such an approach. The reports of the four pathologists in the Folbigg autopsies is a style which I feel more comfortable with, one which I prefer, and is a general format which is used by the overwhelming majority of pathologists in New South Wales. However, I accept that in the case of this second style of report an absence of a statement is taken as that part of the body was normal, while the first style specifically comments on the status of that part of the body. I am not aware of any research which shows which style of report is better, but I suspect that a competent pathologist will examine all organs diligently and comment on any abnormality identified. Considering the pathologists involved and the time when the reports were compiled, I am of the view that the autopsies were generally conducted to a standard which was at least adequate for the time in question – I have examined this in some detail in my response to question 1 above.

9. **What is your understanding of the term 'asphyxia' and how should it properly be used in forensic pathology? Does it have any application in these cases? ANSWER:** I have largely answered this in my response to the various questions above. In my opinion, it should not be used as a standalone cause of death. As indicated above, variations such as "catastrophic

[36] Cooper L. Myocarditis. New England Journal of Medicine, 2009;360:1526-1538.

asphyxiation event" are meaningless from a forensic pathology perspective and are potentially pejorative. This term has no place in forensic pathology practice.

10. **In the case of Laura Folbigg, very shortly before her death she was given the drug Demazin and was suffering from a cold. Noting recent findings in respect of the use of Demazin, please comment on any impact it may have had on her. Also comment on the role a viral infection may have had. ANSWER:** Demazin is an over-the-counter medication generally used for symptomatic relief of colds and flu. Depending on the formulation, Demazin may contain oxymetazoline, chlorpheniramine, pseudoephedrine, bromhexine, guaifenesin, paracetamol, phenylephrine and dextromethorphan.[37] I do not know the composition of Demazin given to Laura, but it remains possible that other active ingredients may have been present in the formulations produced in 1999.

    I note that in this case, toxicological testing of Laura's blood was reported as detecting no common drugs or poisons and that specifically no chlorpheniramine was detected. However, I do not know whether any of the other active ingredients which may have been present in the medication administered would have been detectable by the toxicology laboratory.

    Chlorpheniramine is an antihistamine medication, and in common with many of these earlier forms of the drug class, is not recommended for use in infancy. It has been known for many years that antihistamine use affects respiration and various laryngeal structures in infancy, and this class of drugs has been implicated in SIDS, apnoea of infancy and reflux.[38] [39] [40] I have also considered this topic in passing in my consideration of ALTE's at page 31 *et seq* in this report. It has also been a topic of repeated discussion at the NSW Sudden Infant Death Advisory Committee, of which I was a member, with a strong recommendation that antihistamines be totally avoided under the age of 2 years because of the risk of contributing to sudden death in infancy.

[37] Demazin. MIMS Online, accessed 30 January 2019, http://www.mimsonline.com.au
[38] Downs D, Johnson K, Goding G. The effect of antihistamines on the laryngeal reflex. Laryngoscope 1995;105:857-861.
[39] McKelvey G, Post E, Wood A, Jeffery H. Airway protection following simulated gastro-oesophageal reflux in sedated and sleeping neonatal piglets during active sleep. Clinical and Experimental Pharmacology and Physiology 2001;28:533-9.
[40] Jefferey H. 2018. Future directions in sudden unexpected death in infancy research. In J Duncan, R Byard (eds) SIDS, sudden infant and early childhood death: The past, the present and the future. University of Adelaide Press, Adelaide.

I do emphasise, however, that (1) no antihistamine medication was detected in the blood of Laura, (2) it is not clear to me which Demazin preparation was administered, (3) whether the other components of Demazin were tested for, and (4) there is of course incontrovertible evidence of myocarditis which would in my opinion be entirely capable of causing death in this case.

With reference to the viral infection Laura may have had: I am of the view that very likely the myocarditis was a manifestation of that viral condition and as such could be viewed as its cause.

11. **As you are aware, genome sequencing is being undertaken in respect of all of the children. If genome sequencing reveals no genetic abnormality, does this exclude the possibility that there may still be a genetic abnormality involved in the deaths? ANSWER:** No. It is important to note that in cases where a cardiac genetic abnormality is clinically suspected to have caused sudden death, and where current state-of-the-art technology has been used, it is our experience that a likely pathogenic genetic abnormality will be identified in only about a quarter of cases.[41] This is not surprising, given the current understanding of genetic cardiac disease, the inherent difficulties with interpreting genetic variants of uncertain significance, and the potential role of epigenetic influences on gene expression.

12. **The Inquiry is undertaking a literature review on the number of unexplained infant deaths in the one family. What is your opinion about forensic pathologists relying on such literature? Please detail your reasoning including a description of 'Meadow's Law' and its role in forensic pathology. ANSWER:** I am not a medical statistician and claim no specific expertise in that field. With reference to the so-called "Meadow's Law", also called "DiMaio's Law", I understand it is generally stated as follows: *"One is a tragedy, two is suspicious and three is murder unless there is proof to the contrary."* DiMaio and DiMaio state it as follows:

    "It is the general policy of the authors to ascribe the first death in a family presenting as SIDS to SIDS. The second death by the same mother is labeled undetermined, and a more intensive investigation of the circumstances surrounding the death is conducted. The police are usually asked to interview the family, though in a discreet fashion. A third death with the same mother is thought by the authors to be homicide until proven

[41] Bagnall R, et al. New England Journal of Medicine 2016;374:2441-2452.

otherwise. Infanticide by smothering and repetitive episodes of apnea and cyanosis caused by smothering, presenting as near-miss SIDS cases, are discussed elsewhere in the book. It is the authors' opinion that, while a second SIDS death with one mother is improbable, it is remotely possible and she should be given the benefit of the doubt under most, but not all, circumstances. A third case, in our opinion, is not possible and is a case of homicide. The second case is labeled "undetermined" rather than SIDS to flag the case, that is, to make it stand out for future reference."[42]

I am very much familiar with the various expressions of "Meadow's Law", which I recall being taught as a forensic pathology registrar in the mid-1980s, and which was certainly used as a guide for forensic pathologists and other investigators well into the 21st century. The Sally Clark case and the subsequent hearings by the GMC in the United Kingdom showed that Professor Meadow had misused statistics to inform the jury in that case, giving a much more remote chance of multiple deaths due to SIDS in families than is actually the case. I understand that Dr Meadow was struck off, but was subsequently allowed to practice on appeal.[43]

Current teaching in forensic pathology, as I understand it, is that Meadow's Law is not valid, and that multiple deaths of infants can and do occur in families as a result of a variety of natural causes. I agree with that view. Repeat SIDS deaths are rare, but I have investigated a number of these personally over the years as a forensic pathologist, and have concluded in those cases that SIDS did occur on more than one occasion in each family. Of course, it is always important for the forensic pathologist to have a high level of vigilance in the death of any infant, and even more so where there are multiple deaths, but that vigilance should include a considered search for natural disease processes as well as a search for any evidence of injury on the body to which death can be reasonably attributed to.[44]

In the Folbigg case, there is of course one death (of Laura) where the cause is able to be entirely explained, and arguably the deaths of Patrick (consequences of an ALTE), Caleb (possible laryngomalacia with upper airway obstruction) and Sarah (a structurally abnormal larynx) have also been explained, depending on the importance these conditions are placed

[42] Di Maio V, DiMaio D. 2001.
[43] Meadow and General Medical Council [2006] EWHC 146 (Admin).
[44] Jenny C, Isaac R. The relation between child death and child maltreatment. Archives of Diseases of Childhood. 2006;91:265-269.

on causing the death of those children. I am therefore of the view, that even if Meadow's Law was valid, it would not be applicable in this case given the clinical and pathological findings in the children. Added to that, there is no indication of any injury whatsoever in any of the cases which indicate that any form of physical force has been applied.

13. **In your opinion, are there cases that would fall into the category of 'unusual' or even 'extraordinary' that are not reported in the scientific literature? ANSWER:** I am an assistant editor of one peer-reviewed journal (Addiction), a member of the editorial board of another (Journal of Forensic Sciences) and have peer reviewed articles in multiple journals, including The Lancet, Aviation Space & Environmental Medicine, Medical Journal of Australia, Forensic Science Medicine & Pathology, Medico-legal Update, Pathology, Research & Reports in Forensic Medical Science, and Forensic Science, Medicine & Pathology, and have had many scientific papers published over the years. I am therefore aware in some detail of the processes involved. The medical literature can be viewed as being broadly composed of a combination of research studies, presentation of educational and unusual cases as case reports, and reviews of evidence. With reference to case studies, the process usually involves a practitioner identifying an unusual or educational case as part of their medical practice, then "writing up" the case for publication in a peer-reviewed medical journal. The editors and peer reviewers of that journal then consider the scientific merit of the report, whether it should be published in the journal it has been submitted to, and what modifications should be made. A detailed examination of the process for case reports can be seen in the paper by Gopikrishna, which although relating to dentistry is certainly applicable to medicine generally and forensic pathology specifically.[45] That paper also shows that the majority of case reports submitted to that journal are not accepted for publication (75 out of 86 rejected in 2010), and it is my experience as well that most submitted case report manuscripts are rejected.

    There are many reasons for submitted manuscripts being rejected, but likely the major one in my experience is that there are large numbers of submitted manuscripts and only a limited amount of space in the journal for such articles.[46] This of course assumes that the article has got to the stage of rejection. It is however an unfortunate fact that many medical practitioners, especially those practising in more esoteric areas of medicine such as arguably

[45] Gopikrishna V. A report on case reports. Journal of Conservative Dentistry 2010;13:265-271.
[46] Lee K, Boyd E, Holroyd-Leduc J, et al. Predictors of publication: characteristics of submitted manuscripts associated with acceptance at major biomedical journals. Medical Journal of Australia 2006;184:621-626.

forensic pathology, see many unusual, extraordinary and at times astounding cases, but due to many reasons those cases are never reported. This has certainly been the case with me over the years, and despite having authored over 120 peer reviewed articles to my name, of which a significant proportion are case reports, I have seen many cases in my professional career which are in all likelihood publishable as case reports but which for a variety of reasons have not been submitted or even written.

14. **We have provided you with some literature about the link between opioid peptides (b-casemorphin-7) found in cow's milk and baby formula, and respiratory events and SIDS. Please provide an opinion about the role that opioid peptides may have played in the deaths. ANSWER:** The provided papers is a selection of research articles examining the effect of a substance in cow's milk (β-casomorphin) on infants, and suggest that this opioid may be a factor in SIDS and episodes of apnoea.[47] [48] [49] [50] [51] β-casomorphin is an opioid like substance which has actions similar to morphine in the brainstem, and which would typically include respiratory depression. I emphasise that I have no specific experience in this field of research, but there are certainly aspects of this research which are appealing in relation to SIDS causation. It is known that from an epidemiological perspective, SIDS is more likely in bottle fed than breast fed infants, and breast feeding is strongly recommended in part for that reason. Further, there is extensive research which shows that at least a proportion of SIDS and infantile apnoea cases are the result of immaturity or other deficiencies of the respiratory control centres in the brainstem, noting that some of that research involved brain tissue from SIDS cases retrieved at the Department of Forensic Medicine in Glebe.[52]

    One of the central theories of SIDS is the so-called triple risk model of SIDS, where it is hypothesised that a SIDS event requires the simultaneous occurrence of three factors:[53]

[47] Hedner J, Hedner T. β-casomorphins induce apnea and irregular breathing in adult rats and newborn rabbits. Life Sciences 1987;41:2303-2312.
[48] Wasilewska J, Kaczmarski M, Kostyra E, Iwan M. Cow's-milk–induced infant apnoea with increased serum content of bovine b-casomorphin-5. Journal of Pediatric Gastroenterology and Nutrition 2011;52:772-775.
[49] Sahin O, Boztepe S, Aytekin I. A1 and A2 bovine milk, the risk of Beta-casomorphin-7 and its possible effects on human health. Selcuk Journal of Agriculture and Food Sciences 2018;32:640-645.
[50] Sun Z, Zhang Z, Wang Z, et al. Relation of β-casomorphin to apnea in sudden infant death syndrome. Peptides 2003;24:937-943.
[51] Wasillewska J, Sienkiewicz-Szlapka E, Kuzbida E, et al. The exogenous opioid peptides and DPPIV serum activity in infants with apnoea expressed as apparent life threatening events (ALTE). Neuropeptides 2011;45:189-195.
[52] Machaalani R, Waters K. Neurochemical abnormalities in the brainstem of the sudden infant death syndrome. Paediatric Respiratory Reviews 2014;15:293-300.
[53] Collins K, Byard R. 2014.

1. An infant sleeping during a critical stage of development.
2. An underlying abnormality such as an abnormal medullary (brainstem) serotonergic system.
3. The presence of an exogenous homeostatic stressor, including as examples a soft mattress, the face down sleeping position, parental smoking, minor respiratory tract infection, to name a few.

Using that schema and knowing some of the effects of β-casomorphin, there is certainly an argument for this substance to be viewed as a potential exogenous homeostatic stressor. Given it is present in many forms of cow's milk, and all four children were fed with cow's milk based formula with both Caleb and Sarah having evidence of milk ingestion in the time immediately preceding death, it raises the intriguing possibility that the deaths could in some way be related to consumption of that cow's milk, causing respiratory depression and resultant demise.

15. **Accepting that there is some guidance provided to expert witnesses in the UCPR, are there any other guidelines provided by the Royal College of Pathologists of Australasia or other relevant body? If yes, please provide them. ANSWER:** The RCPA has issued a document on expert evidence titled "Nomination of Pathologists for Expert Panels,"[54] noting that it largely uses the "Guidelines for Expert Witnesses in Proceedings in the Federal Court of Australia." The College has also produced a guideline on provision of second opinions in forensic pathology.[55]

NSW Health has a Policy Directive (PD2012_017 Forensic Pathology – Code of Practice and Performance Standards in NSW)[56] – I understand that this policy directive was withdrawn from use in around 2017, but it remains an active document on the NSW Health website which states that compliance with the policy directive is mandatory. As indicated in that document, the policy directive is based on a document issued jointly by the United Kingdom Home Office and Royal College of Pathologists (of the UK) in 2004, which in turn was used by

[54] Nomination of Pathologists for Expert Panels. Accessed 31 January 2019 at https://www.rcpa.edu.au/getattachment/a1d715fe-c61e-4466-97ad-ae3c21eb5743/Nominations-of-Pathologists-for-Expert-Panels.aspx
[55] Provision of second opinions in forensic pathology. Accessed 31 January 2019 at https://www.rcpa.edu.au/getattachment/5675fc33-1618-453c-b054-8cc017f11431/Provisions-of-Second-Opinions-in-Forensic-Patholog.aspx
[56] PD2012_017 Forensic Pathology – Code of Practice and Performance Standards in NSW. Accessed 31 January 2019 at https://www1.health.nsw.gov.au/pds/ActivePDSDocuments/PD2012_049.pdf

a working party of forensic pathologists of the RCPA as the basis for a document on which the NSW Health department document was based – I was a member of that working party and also was involved in the production of the NSW Health version of the document.

The Australia and New Zealand Forensic Science Society also provides guidance in the form of a code of professional practice for members of the ANZFSS.[57] Please note that I am not currently a member of the ANZFSS.

To my knowledge none of these documents or any previous versions of such documents were published at the time of the death of the Folbigg children or at the time of the Trial.

16. **Are there ethical limits placed on forensic pathologists when they provide reports or give evidence in court? Of particular concern is the obligation to research an issue prior to providing an opinion, and to provide the literature relevant to that opinion. ANSWER:** I am not aware of any specific ethical requirements in relation to forensic pathologists generating reports or giving evidence in court. There are of course the general ethical requirements for all medical practitioners, and it would be hoped that all medical practitioners adhere to those standards. The Medical Board of Australia has produced a document entitled "Good Medical Practice: A Code of Conduct for Doctors in Australia,"[58] which I believe can be used as guidance. I note specifically that document has a brief section on expert evidence titled "Medical reports, certificates, expert evidence", which I reproduce in full as follows:

> *"The community places a great deal of trust in doctors. Consequently, doctors have been given the authority to sign a variety of documents, such as death certificates and sickness certificates, on the assumption that they will only sign statements that they know, or reasonably believe, to be true. Good medical practice involves:*
>
> - *Being honest and not misleading when writing reports and certificates, and only signing documents you believe to be accurate.*
> - *Taking reasonable steps to verify the content before you sign a report or certificate, and not omitting relevant information deliberately.*

[57] Code of professional practice for members of the ANZFSS. Accessed 31 January 2019 at http://anzfss.org/wp-content/uploads/2012/05/ANZFSS-Code-of-Professional-Practice-Final.pdf
[58] Good Medical Practice: A Code of Conduct for Doctors in Australia. Accessed 31 January 2019 at https://www.medicalboard.gov.au/codes-guidelines-policies/code-of-conduct.aspx

- *Preparing or signing documents and reports if you have agreed to do so, within a reasonable and justifiable timeframe.*
- *Making clear the limits of your knowledge and not giving opinion beyond those limits when providing evidence."*

In my opinion these are entirely reasonable requirements.

With reference to the use of research to inform opinions, and the use of medical literature references relevant to the opinion, my general experience is that it is the exception rather than the rule that references are quoted in autopsy reports, and it is certainly not my practice to routinely include medical article reference in my autopsy reports, with some limited exceptions. Examples of such exceptions would typically include particularly complex cases, or pathological processes which might be easily misunderstood by non-pathologist readers of the report. On the other hand, it is almost invariably the case that in my reports where I provide a detailed analysis of a case that I will include a selection of relevant references germane to the issues discussed.

17. **What role does a forensic pathologist have in providing an opinion about possible causes of death? ANSWER:** One of the primary purposes of forensic pathologists in the Australian medicolegal (coronial) death investigation system is to provide an opinion as to cause of death. The pathologist will perform an investigation of the death, which frequently includes an autopsy to a greater or lesser extent possibly with a range of ancillary investigations, which will eventually result in production of a report to the coroner which will include an opinion as to cause of death. In the majority of cases, the coroner will accept the cause of death as proffered and an inquest will be dispensed with (see S.27 of the *Coroners Act 2009 No 41*). A consequence of this, and a view that in my opinion is held by the majority of forensic pathologists in Australia is that there is a degree of pressure on the pathologist to provide a cause of death if at all possible.

There is of course the added consideration that the coronial jurisdiction, in general, has a "balance of probabilities" standard of proof, while it is of course much higher in criminal matters. A consequence of this is that a cause of death can be given for the coroner's investigation which would be entirely reasonable, "on balance", but which might be misleading in a criminal matter if the pathologist does not expressly state the level of

certainty in that written opinion document, or it is not explicitly expressed at later time during proceedings.

Most lay people, and I suspect most doctors, view the cause of death as expressed in a death certificate as a positive and incontrovertible finding. Unfortunately, the intellectual process used in determining cause of death is very far from this. There are relatively few deaths where the cause can be stated without doubt. Examples would include a ruptured heart due to a coronary occlusion, a ruptured aneurysm in the brain, a high velocity gunshot wound to the head, and exsanguinating blood loss from a severed limb. Even then, it would not be uncommon to find competing potential causes of death in those case, such as the presence of a potentially fatal illicit drug level, or terminal cancer in the person who shot himself, or very advanced coronary artery disease – these are daily occurrences which have to be considered in a forensic pathologist's professional life and in those cases they would be easy to dismiss as a cause of death. More difficult are those where there is potentially lethal heart disease as well as a potentially lethal drug level, or a case where there is restraint of a person during say a police operation and the deceased may have a combination of heart disease, drug intoxication and possibly the controversial condition excited delirium syndrome. Even in relatively straightforward cases such as an elderly person dying with advanced coronary artery disease of such severity to be capable of causing death at any time but without the presence of any acute cardiac lesions, the cause of death comes down to an opinion by the pathologist, and in the main the absence of an obvious competing other cause of death. SIDS cases are a further category of cases, where the diagnosis of SIDS is based on the absence of any other cause of death – this might appear a unique situation at first sight, but it is in fact not an uncommon situation. For example, a similar type of reasoning is used in drug overdose deaths where the level of many drugs cannot be used to determine whether death was due to the drug or not but rather the absence of any other cause of death is used as an indication that death was the result of a drug overdose.

In the current cases, there is obviously a desire by all concerned to determine the cause of death and this should be on the basis of a combination of the histories provided and the objective findings in each case. I do not believe that an unequivocal cause of death can be given in any of the cases, emphasising this is more often the case in coronial autopsy practice than not. It is on that basis that I have given the various causes of death in my response to question 2.

18. **The Supreme Court of Victoria has issued Practice Note No. 2 'Expert Evidence in Criminal Trials.' This includes Item 1 'Forensic Pathology: Standard Questions to Address the Cause of Death in an Alleged Homicide.' What utility do these guidelines have in this case?**
ANSWER: I cannot commend this document highly enough for assessment of complex deaths and subsequent presentation of evidence in court proceedings. To a large extent the document appears to have been written with infant deaths in mind, and I understand the document was produced with the involvement of Professor Cordner subsequent to his input into the Goudge Inquiry.

I will not attempt to specifically examine the four Folbigg deaths through the prism of the series of questions, but in my opinion the conclusions reached by any objective pathologists would be very different to those expressed by a number of expert witnesses in this matter.

I have provided expert reports in multiple cases in Victoria, and many of those have involved child deaths where a person has been charged with inflicting their death. The schema certainly adds to the level of complexity and length of any report which may be required, and some of the questions may be entirely irrelevant to the issues at hand, but there is no doubt in my mind that writing a report in response to those questions provides a more rigorous analysis of the cases, and importantly should allow direct comparison of views expressed by various experts.

I have found two major problems with the schema:

1. Question 9: "Have the findings of injury in this case ever been described in the literature as being caused accidentally, or by self-infliction or not due to intentional harm by another?" The problem with this question is the sheer volume of the medical literature published, and a certainty that not all research on a topic will necessarily be found using even advanced medical and scientific database searches. For example, a simple search performed using the University of Sydney library website returns 38,022 results for the entire phrase "Sudden Infant Death Syndrome," of which 22,910 are articles, 18,911 of those are peer-reviewed, and there are 377 text resources (generally books). It goes without saying that it is physically impossible to critically review all these sources, many of which would involve attending various libraries in person and the cost of purchasing of a large number of articles.

2. It is my experience that the pathologist who wrote the original autopsy report does not explicitly respond to the questions asked. It could be argued that at least some of the questions would be answered *en passant* in any properly compiled autopsy report, and this is true, but of course such an approach is arguably not an adequate response to the requirements of the Practice Note. Again, a detailed and comprehensive response to each of the question asked would in all likelihood place an impossible workload on the medicolegal death investigation system, and at least some of the questions asked in that schema are probably not able to be answered as fully as might be expected at first sight.

Nevertheless, I reiterate my view that I highly recommend this document as a schema for any thoughtful assessment of an infant homicide case, and that use of such a document has the potential for the presentation of better, more impartial and more considered expert evidence by forensic pathologists.

19. **We understand that you provided a brief report to the NSW Health Department at their request some years ago. Please attach that report and provide any relevant explanatory comments. ANSWER:** I attach a copy of that Briefing Note, which I emailed to Ms Catherine Foster-Curry, Director of Forensic Medicine, for action on around 16 June 2015 (**ATTACHMENT 3**). Professor Cordner had provided me with a copy of his report on 15 June 2015 as a courtesy and after briefly reading the report, I spoke to Ms Foster-Curry and emailed a copy of the report to her. Ms Foster-Curry in turn asked me to write an urgent briefing note to inform her and other personnel in NSW Health on the major issues relating to the case, and to provide advice relating to the implications of and possible institutional responses to that report. I wrote the draft briefing note as a matter of urgency commencing on 15 June 2015, and finished writing it the following day, at which time I sent it to Ms Foster-Curry.

I was not informed of any further actions by Ms Foster-Curry subsequent to this, but I believed that the document I provided to her was for the purposes of her informing either senior management in NSW Health Pathology or in NSW Health by way of a formal Briefing Note or similar. I may or may not have had further conversations in relation to this document with Ms Foster-Curry, but have no recollection of such conversations, if they indeed did take place. To my knowledge, my strong recommendation (last dot point of the document) that all four autopsies be reviewed in detail was not acted on. I resigned from my

position of Clinical Director about a month after I wrote the note and had no further involvement in the case.

I stand by the views expressed in that draft Briefing Note.

Johan Duflou MBChB MMedPath(Forens) FRCPA FFFLM DAvMed

Consulting Forensic Pathologist

13 February 2019

CURRICULUM VITAE

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QUALIFICATIONS AND EDUCATION:

1976-1981: Bachelor of Medicine and Bachelor of Surgery (MBChB), University of Cape Town.

1983-1987: Master of Medicine in Forensic Pathology (MMedPath(Forens)), University of Cape Town. Awarded the Nikon Prize.

1991: Fellow of the Royal College of Pathologists of Australasia (FRCPA), in Anatomical Pathology, Forensic Slant.

1994: Postgraduate research fellow in the Department of Cardiovascular Pathology at the Armed Forces Institute of Pathology (AFIP), Washington DC.

1993-1996: Diploma in Aviation Medicine (DAvMed), University of Otago - Title of dissertation: Heat-induced Coronary Artery Artefacts: Pitfalls in the Diagnosis of Ischaemic Heart Disease in Aviation Autopsies.

1999: Visiting Professor, Office of the Medical Investigator, University of New Mexico.

2005: Foundation Fellow of the Faculty of Forensic & Legal Medicine, Royal College of Physicians (UK).

2005: Certificate in Early Management of Severe Trauma (EMST), Royal Australasian College of Surgeons.

2014: Executive Clinical Leadership Programme, Clinical Excellence Commission, NSW Health.

PRESENT APPOINTMENTS:

- Consultant forensic pathologist in private practise.
  - Consultancy work in various aspects of Forensic Medicine and Forensic Science. Opinions have been provided to all parties in disputes, including defence and prosecution counsel. Evidence has been given as a retained consultant in criminal, coronial, family law, medical negligence and civil cases and judicial inquiries in numerous jurisdictions, including most Australian States and Territories, New Zealand, Papua New Guinea, USA, Indonesia, Thailand, Samoa and Singapore.
  - Registered as expert witness with:
    New South Global, University of New South Wales
    Expert Opinion Australia
    Expert Experts
    National Expert Witness Service
    Crime Management Faculty, NSW Police Service.
- Part-time specialist forensic pathologist, Forensic Medicine Centre, Canberra.
- Clinical Professor, Sydney Medical School, University of Sydney.
- Conjoint Associate Professor, National Drug and Alcohol Research Centre, University of New South Wales.
- Current research interests:
  - Sudden cardiac death in young persons, in collaboration with Trans-Tasman Response Against Death in the Young (TRAGADY) and the Department of Cardiology, Royal Prince Alfred Hospital.
  - Injury mechanism, including mechanism of injury in childhood, and mechanism of death in asphyxial death cases.
  - Aviation pathology, investigating various pathological artefacts caused by air crashes and examining the accuracy of aircrew medical examinations.
  - Illicit drug use deaths, in collaboration with the National Drug and Alcohol Research Centre of the University of NSW.
- Medical and scientific journal editorial and peer review roles:
  - Assistant Editor, Addiction.
  - Member of Editorial Board – Journal of Forensic Sciences.
  - Editorial Review Panel member of various medical journals, including the Lancet, Aviation Space & Environmental Medicine, Medical Journal of Australia, Journal of Forensic Sciences, Forensic Science Medicine & Pathology, Medico-legal Update, Addiction, Pathology, Research & Reports in Forensic Medical Science, and Forensic Science, Medicine & Pathology.
- Wing Commander, Royal Australian Air Force Standby Reserve.

PAST APPOINTMENTS:

2015-2017: Longterm locum Forensic Pathologist, National Forensic Pathology Service, Southern Hub in Christchurch, New Zealand.

1988-2015: Specialist Forensic Pathologist, Senior Forensic Pathologist, Chief Forensic Pathologist and Clinical Director, Department of Forensic Medicine, Sydney. Duties included death investigations for the coroner, extensive attendance at various courts as an expert witness, administrative duties as head of department, teaching of medical and other undergraduate and postgraduate students at the University of Sydney, University of NSW, Notre Dame University and University of Technology

Sydney (UTS), and training and supervision of pathology registrars for admission to Fellowship of ther Royal College of Pathologists of Australasia.

1988: Consultant State Pathologist - Tripartite appointment between the Department of Health, Groote Schuur Hospital and the University of Cape Town, South Africa.

1984-1988: Lecturer in the Department of Forensic Medicine and Toxicology at the University of Cape Town - whole class lectures, practical demonstrations and tutorials to Medical (third and fourth year), Law (fifth year) and Anatomical Science (second and third year) students.

1984-1987: Registrar in Forensic Medicine at the University of Cape Town and Groote Schuur Hospital, South Africa.

1983: Medical Officer in Forensic Medicine at the State Regional Laboratories, Cape Town, South Africa.

1982: Intern at Uitenhage Provincial Hospital, South Africa.

COMMITTEE MEMBERSHIP:

- Member of Clinical Council, NSW Health Pathology (2014-2015).
- Member of the Forensic Pathology Advisory Committee, Royal College of Pathologists of Australasia (2002-2014).
- Member of Overseas Trained Specialist Assessment Committee, Royal College of Pathologists of Australasia (current).
- Member of the Australian Cardiac Genetics Testing Network Steering Committee (current).
- Member of Trauma Committees of Royal North Shore Hospital, St Vincent's Hospital and St George Hospital (1994-2015).
- Member of the NSW Sudden Infant Death Advisory Committee (1992-2015).

OTHER ACTIVITIES:

- Retained by AusAID in 1996 to:
  - Provide technical advice for Papua New Guinea on forensic medicine matters including the setting up of a national forensic pathology service appropriate to the needs of a developing country.
  - Set up an autopsy consultancy service to Papua New Guinea medical and air crash investigation personnel.
  - Conduct undergraduate and postgraduate teaching at the University of Papua New Guinea in all areas of forensic medicine.
  - Transfer of skills knowledge to local medical staff in PNG to minimise dependence on long-term Australian aid.
- Seconded to Australian Federal Police to assist in investigation of Bali Bombings of 1 October 2005. Duties included performing autopsies on Australian victims and "suicide" bombers for purposes of identification and trace evidence collection.
- Medical member of QANTAS Immediate Response Team (IRT).
- Frequent involvement with the media (print, radio and television) and public in numerous roles, including interviewed expert (e.g. A Case for the Coroner and Outback Coroner reality

television series, ABC Radio and Television current affairs programs, Today Tonight, A Current Affair, National Geographic, many newspapers articles in the Sydney Morning Herald, Sunday Telegraph, Canberra Times, and magazines including Australian Geographic and Cosmos), background technical advisor (e.g. White Collar Blue, Dateline, Blackjack, The Strip, Top of the Lake), plot and technical advisor to print fiction authors (e.g. Jon Cleary, Kathryn Fox, Narelle Day, Gabrielle Lord), and public interest lectures (e.g. Death and the sculptures of Ron Mueck at the Museum of Contemporary Art).

COLLEGE & SOCIETY MEMBERSHIPS:

Fellow of the Royal College of Pathologists of Australasia.
Fellow of the Faculty of Forensic and Legal Medicine, Royal College of Physicians (UK).
Fellow of the American Academy of Forensic Sciences.
Member of the Australasian Society of Aerospace Medicine.
Member of the Australian Society of Air Safety Investigators.
Member of the International Society of Air Safety Investigators.
Member of the Australian Academy of Forensic Sciences.

MILITARY SERVICE:

Wing Commander, Royal Australian Air Force Standby Reserve.
Duties include military air crash investigation, including autopsies on personnel and examination of aircraft wreckage, provision of medicolegal reports and expert testimony at various military courts and Boards of Inquiry, and training of ADF medical officers in various areas of forensic pathology, death investigation and air crash investigation.

AWARDS:

Australian Defence Medal.
Australian Federal Police Operations Medal - Operation Alliance.

RESEARCH AND PUBLICATIONS:

A. RESEARCH FUNDING (Total $3,741,232):

1. Cordner S, Campbell P, Lee K, Herdson P, Naylor C, Bourne T, Brain T, Hilton J, Duflou J, Ellis P, Cooke C. **National SIDS Autopsy Protocol and Database.** National SIDS Council of Australia (1992-94) $348,550.

2. Waite P, Duflou J, Brown J, Griffiths M. **A multidisciplinary study of motor vehicle accidents and fatal head injury in children: implications for Australian road safety standards.** Australian Research Council Collaborative Grant, with the University of NSW and the NSW Roads and Traffic Authority (1996-99) $306,000.

3. Duflou J, Waite P. **Spinal cord injury in children involved in fatal child abuse and road trauma.** NSW Department of Community Services Research Grant (1997-99) $18,000.

4. Waite P, Duflou J, Moran K. **Spinal cord injury in fatal child abuse - an MRI-autopsy correlation.** Sydney Childrens Hospital Foundation (2000) $12,000.

5. Waite P, Gorrie C, Duflou J, Rodriguez M. **Fatalities in elderly drivers and pedestrians: Neuropathological evaluation of sub-clinical dementia.** NSW Motor Accidents Authority (2000-03) $240,000.

6. Taylor R, Dudley M, Carter G, Duflou J, Morrell S. **Case control study of suicide and attempted suicide.** National Health & Medical Research Council Grant (2004-2007) $801,933.

7. Darke S, Kaye S, Duflou J, McKetin R. **Cardiac and other harms of psycho-stimulant use.** Commonwealth Department of Health and Aged Care (2005-2007) $149,229.

8. Semsarian C, Twigg S, Duflou J, Langlois N. **Genetic predisposition to sudden cardiac death in young people with type 1 diabetes.** National Heart Foundation of Australia (2007-2008) $124,000.

9. Taylor R, Dudley M, Carter G, Duflou J. **Case control study of suicide and attempted suicide in young adults in NSW.** Department of Health & Aged Care (2008-2010) $240,020.

10. Darke S, Kaye S, McKetin R, Duflou J. **The comparative health of methamphetamine and opioid users.** Commonwealth Department of Health and Aged Care (2008-2010) $150,000.

11. Semsarian C, Weintraub R, Duflou J, Puranik R, Skinner J. **Investigation of sudden cardiac death in the young.** National Health & Medical Research Project Grant (2010-2012) $658,500.

12. Curtis K, Holland A, Mitchell R, Foster K, Burns B, Duflou J, Wilson K. **Evidence for policy change and better outcomes for severely injured children.** Thyne Reid Foundation & NSW Agency for Clinical Innovation (2014-2017) $192,000.

13. Curtis K, Holland A, Mitchell R, Foster K, Burns B, Duflou J, Wilson K. **Evidence to change policy and improve outcomes in children suffering major injury.** National Health & Medical Research Partnership Grant (2014-2019) $501,694.

B. BOOK CHAPTERS, MONOGRAPHS, ETC:

1. Duflou J, Haikal N, Evans W, Hilton J. **HIV autopsies in Sydney.** In Advances in Forensic Sciences, volume 1. Jacob B, Bonte W (eds). Berlin: Verlag Dr Köster, 1995,1-3.

2. Chan W, Duflou J, Hilton J. **A quick screening test for HIV-1 antibody prior to autopsies.** In Advances in Forensic Sciences, volume 1. Jacob B, Bonte W (eds). Berlin: Verlag Dr Köster, 1995,4-5.

3. Duflou J, Hilton J. **Forensic autopsies - an audit.** In Advances in Forensic Sciences, volume 1. Jacob B, Bonte W (eds). Berlin: Verlag Dr Köster, 1995,35-37.

4. Kencian J, Duflou J, Hilton J. **Medicolegal postmortems in rural Australia - a survey of 100 postmortems performed by non-forensic pathologists.** In Advances in Forensic Sciences, volume 1. Jacob B, Bonte W (eds). Berlin: Verlag Dr Köster, 1995,47-50.

5. Duflou J, Marriott D. **The role of the autopsy in HIV infection.** In Managing HIV. Stewart G (ed). Sydney: Australasian Medical Publishing Co, 1997, 123-124.

6. Duflou J. **Classification of opioid deaths - a forensic pathologist's perspective.** In Proceedings of the International Opioid Overdose Symposium. Hall W (ed), 1997;77-82. National Drug and Alcohol Research Centre Monograph No. 35. Sydney: National Drug and Alcohol Research Centre.

7. Gorrie C, Duflou J, Mark A, Waite P. **Neuropathological injury in child pedestrians.** In Proceedings of the Conference on Pedestrian Safety. Smith KP, Aitken BG, Grzebieta RH (eds). 1998, 45-52.

8. Darke S, Hall W, Kaye S, Ross J, Duflou J. **Hair morphine concentrations of fatal heroin overdose cases and living heroin users.** National Drug and Alcohol Research Centre Technical Report No. 90. 2000.

9. Darke S, Kaye S, Duflou J. **Cocaine-related fatalities in New South Wales, Australia, 1993-2002.** National Drug and Alcohol Research Centre Technical Report No. 171. 2003.

10. Gorrie C, Waite P, Sachdev P, Rodriguez M, Duflou J, Brown J. **Fatalities in older drivers and pedestrians: Neuropathological evaluation of pre-clinical dementia.** Commissioned by the Motor Accidents Authority NSW, November 2004.

11. Darke S, Kaye S, McKetin R, Duflou J. **Physical and psychological harms of psychostimulant use.** National Drug and Alcohol Research Centre Technical Report No. 286. 2007.

12. Darke S, Torok M, Kaye S, Duflou J. **Cardiovascular disease risk factors and symptoms amongst regular psychostimulant users.** National Drug and Alcohol Research Centre Technical Report No. 303. 2009.

13. Tu E, Bagnell R, Duflou J, Semsarian C. **SUDEP: heart and brain link**, In Chapman D, Panelli R, Hanna J, Jeffs T (eds), 2011, Sudden unexpected death in epilepsy: continuing the global conversation, Epilepsy Australia, Camberwell, Australia.

14. Duflou J. **Air crash investigation.** In Byard R & Payne-James J (eds), 2015, Encyclopedia of Forensic and Legal Medicine, 2nd edition, Elsevier.

15. Duflou J, Ong B, Baber J, Burke M, Ranson D, Cordner S. **Forensic pathology.** In Freckleton I & Selby H (eds), 2017, Expert Evidence. Thompson Reuters.

C. SCIENTIFIC JOURNAL PUBLICATIONS:

1. Duflou J, Odes R. **Idiopathic uterine rupture in the mid-trimester of pregnancy.** South African Medical Journal 1984:65;221-222.

2. Wormald P, Milton R, Abrahamson M, Duflou J, Millar R. **A peptide fragment from the GnRH precursor stimulates gonadotrophin release from cultured human pituitary cells.** South African Journal of Science 1985:81;579.

3. Duflou J. **A study of 93 firearm associated deaths during security force action in the greater Cape Town area, 1985.** South African Medical Journal 1986:70;88-90.

4. Duflou J, Mausling G. **Anti-epileptic drug monitoring (letter).** South African Medical Journal 1986:70;636.

5. Duflou J. **Fatherly advise to spouses of housemen (letter).** South African Medical Journal 1987:71;400.

6. Duflou J. **Combined phenylketonuria and pyruvate kinase deficiency - statistical fallacies? (letter).** South African Medical Journal 1987:72;363.

7. Lamont D, Duflou J. **Dagga smoker's hand - a new physical sign?** South African Medical Journal 1988:73;356-358.

8. Lamont D, Duflou J. **Copper sulfate - not a harmless chemical.** American Journal of Forensic Medicine and Pathology 1988:9;226-227.

9. Duflou J, Lamont D, Knobel G. **Homicide in Cape Town, South Africa.** American Journal of Forensic Medicine and Pathology 1988:9;290-294.

10. Kennedy M, Lawrence C, Duflou J. **Drug related cardiac death in sport (Abstract).** Clinical and Experimental Pharmacology and Physiology 1992; Suppl 21: 35.

11. Cunningham A, Mathijs JM, Kannourakis G, Tiong T, Evans WA, Bilous AM, Miliken J, Duflou J. **Identification of IL-4 and other cytokine RNA in HIV-infected human brain in proximity to multinucleated giant-cells (Abstract).** Journal of Cellular Biochemistry 1993;17E:44.

12. Hill DA, West RH, Duflou J. **Value of the prospective "before and after" study as a methodology to evaluate outcome in a trauma centre.** Australian and New Zealand Journal of Surgery 1993:63;940-945.

13. Katelaris A, Kencian J, Duflou J, Hilton J. **Brains at necropsy: To fix or not to fix?** Journal of Clinical Pathology 1994:47;718-720.

14. Kencian J, Duflou J, Hilton J. **Medicolegal post mortems in rural New South Wales - another rural health crisis.** Medical Journal of Australia 1994:161;366-368.

15. Foroudi F, Cheung K, Duflou J. **A comparison of the needle biopsy post mortem with the conventional autopsy.** Pathology 1995:27;79-82.

16. Kedziora A, Duflou J. **Attempted suicide by intravenous injection of mercury: a case report.** American Journal of Forensic Medicine and Pathology 1995:16;172-175.

17. Duflou J, Virmani R, Rabin I, Burke A, Farb A, Smialek J. **Sudden death as a result of heart disease in morbid obesity.** American Heart Journal 1995:130;306-313.

18. Dore G, Marriott D, Duflou J. **Clinico-pathological study of cytomegalovirus (CMV) in AIDS autopsies: under-recognition of CMV pneumonitis and CMV adrenalitis.** Australian and New Zealand Journal of Medicine 1995:25;503-506.

19. Cala A, Lawrence C, Duflou J. **When is post mortem lividity fixed? (Abstract)** Pathology 1995;27:A8. doi:10.1016/S0031-3025(16)35368-5.

20. Duflou J, Marriott D. **The role of the autopsy in HIV infection.** Medical Journal of Australia. 1996;164:616-617.

21. Hill D, Duflou J, Delaney L. **Blunt traumatic rupture of the thoracic aorta: an epidemiological perspective.** Journal of the Royal College of Surgeons of Edinburgh 1996;41:84-87.

22. Hill D, Delaney L, Duflou J. **A population-based study of outcome after injury to car occupants and to pedestrians.** Journal of Trauma: Injury, Infection and Critical Care 1996;40:351-355.

23. Waite P, Gorrie C, Duflou J, Mark A, Brown J. **A multidisciplinary study of traffic crashes and fatal head injury in children.** Child's Nervous System 1998;14:414-415.

24. Duflou J. **Autopsies and next-of-kin (letter).** Noah's Arc (Journal of the Australian Society for HIV Medicine) 1998;9:1.

25. Gorrie C, Duflou J, Brown J, Waite P. **Fatal head injury in children: a new approach to scoring axonal and vascular damage.** Child's Nervous System 1999;15:322-328.

26. Mark A, Duflou J, Waite PME. **Changes in the human nucleus basalis following traumatic brain injury (Abstract).** Journal of Neurotrauma 1999;16:1003.

27. Griffiths C, Duflou J. **Recovery of Australian Service personnel MIA from WWII: The ADF Forensic Recovery Team.** Journal of the Australian Defence Health Service 2000;1:47-53.

28. Duflou J, Mark A. **Aortic dissection after ingestion of 'Ecstasy' (MDMA).** American Journal of Forensic Medicine and Pathology 2000;21:261-263.

29. Waite PME, Gorrie CA, Benetatos E, Brown J, Duflou J, Gibson T. **Neuropathology in children following fatal road trauma (Abstract).** European Journal of Neuroscience 2000;12 Suppl:U223.

30. Cheung B, Duflou J. **Pre-existing pulmonary thromboembolic disease in passengers with the 'economy class syndrome'.** Aviation, Space and Environmental Medicine 2001;72:747-749.

31. Gorrie C, Duflou J, Brown J, Gibson T and Waite PME. **Extent and distribution of vascular brain injury in pediatric road fatalities.** Journal of Neurotrauma 2001;18:849-860.

32. Darke S, Hall W, Kaye S, Ross J, Duflou J. **Hair morphine concentrations of fatal heroin overdose cases and living heroin users.** Addiction 2002;97:977-984.

33. Cheung B, Duflou J. **Air travel and thrombo-embolic disease.** Phlebology Digest 2002;15:13-15.

34. Gorrie C, Oakes S, Duflou J, Blumbergs P, Waite PME. **Axonal injury in children after motor vehicle crashes: extent, distribution, and size of axonal swellings using ß-APP immunohistochemistry.** Journal of Neurotrauma 2002;19:1171-82.

35. Darke S, Kaye S, Duflou J. **Cocaine-related fatalities in New South Wales, Australia 1993-2002.** Drug and Alcohol Dependence 2004;77:107-114.

36. Gorrie CA, Rodriguez ML, Duflou J, Sachdev P, Waite PME. **Older pedestrian fatalities: Assessment of dementia related neuropathology (Abstract).** Journal of Neurotrauma 2004;21:1314.

37. Gilchrist E, Duflou J. **Incidental Myocardial Infarction in Ehlers-Danlos Syndrome Type IV?** Journal of Forensic Science 2005;50:461-464.

38. Puranik R, Chow C, Duflou J, Kilborn M, McGuire M. **A 10 year review of sudden death in young Australians.** Heart Rhythm 2005;2:1277-1282.

39. Puranik R, Chow C, Duflou J, Kilborn M, McGuire M. **A 10 year review of sudden death in the young (Abstract).** Circulation 2005;112 Suppl:U1183.

40. Tseung J, Duflou J. **Diffuse pulmonary ossification: an uncommon incidental autopsy finding.** Pathology 2006;38:45-48.

41. Gorrie C, Rodriguez M, Sachdev P, Duflou J, Waite P. **Increased neurofibrillary tangles in the brains of older pedestrians killed in traffic accidents.** Dementia & Geriatric Cognitive Diseases 2006;22:20-26.

42. Duflou J, Nickols G, Waite P, Griffiths R, Sage M. **Artefactual contraction band necrosis of the myocardium in fatal air crashes.** Aviation, Space & Environmental Medicine 2006;77:944-949.

43. Darke S, Kaye S, Duflou J. **Systemic disease among cases of fatal opioid toxicity.** Addiction 2006;101:1299-1305.

44. Kastowsky T, Stevenson M, Duflou J. **Sudden death from saccular laryngeal cyst.** Journal of Forensic Sciences, 2006;51:1144-1146.

45. Darke S, Kaye S, Duflou J. **Comparative cardiac pathology among deaths due to cocaine toxicity, opioid toxicity and non-drug related causes.** Addiction, 2006:101;1771-1777.

46. Kaye S, McKetin R, Duflou J, Darke S. **Methamphetamine and cardiovascular pathology: A review of the evidence.** Addiction 2007;102:1204-1211.

47. Darke S, Duflou J, Kaye S. **Comparative toxicology of fatal heroin overdose cases and morphine positive homicide victims.** Addiction 2007;102:1793-1797.

48. Gorrie C, Rodriguez M, Sachdev P, Duflou J, Waite P. **Mild neuritic changes are increased in the brains of fatally injured older motor vehicle drivers.** Accident Analysis and Prevention 2007;39:1114-1120.

49. Donlon D, Blenkin M, Duflou J, Lain R, Lowe T, Parsons J. **Identification of the unknown sailor from HMAS Sydney (Abstract).** Homo - Journal of Comparative Human Biology 2007;58:245.

50. Chase J, Donaldson L, Duflou J, Gorrie C. **Safety restraint injuries in fatal motor vehicle collisions.** Forensic Science, Medicine & Pathology 2007;3:258-263.

51. Lim C, Duflou J. **Hypothermia fatalities in a temperate climate: Sydney, Australia.** Pathology 2008;40:46-51.

52. Darke S, Duflou J. **Toxicology and circumstances of death of homicide victims in New South Wales, Australia 1996-2005.** Journal of Forensic Sciences 2008;53:447-451.

53. Chan P, Duflou J. **Suicidal electrocution in Sydney, Australia – a 10 year review.** Journal of Forensic Sciences 2008;53:455-459.

54. Darke S, Kaye S, McKetin R, Duflou J. **The major physical and psychological harms of methamphetamine use.** Drug and Alcohol Review 2008;27:253-262.

55. Tu E, Twigg S, Duflou J, Semsarian C. **Causes of death in young Australians with type 1 diabetes: A review of coronial post mortem cases.** Medical Journal of Australia 2008;188:699-702.

56. Puranik R, Fox O, Duflou J, Bao S. **Premature coronary artery disease in Australian men: an autopsy study (Abstract).** Heart, Lung and Circulation 2008;17(S3):S105.

57. Tu E, Twigg S, Duflou J, Semsarian C. **Sudden unexplained death in young Australians with type 1 diabetes mellitus: Clinical and genetic analysis of "dead in bed" cases (Abstract).** Heart, Lung and Circulation 2008;17(S3):S133.

58. Skinner J, Duflou J, Semsarian C. **Reducing sudden death in the young in Australia and New Zealand: the TRAGADY initiative (Editorial).** Medical Journal of Australia 2008;189:539-540.

59. May A, Fulde G, Duflou J, Mengersen K, Read-Allsopp C. **External injury documentation is inadequate: Grounds for routine photography in the emergency department.** Emergency Medicine Australasia 2008;20:500-507.

60. Kaye S, Darke S, Duflou J, McKetin R. **Methamphetamine-related fatalities in Australia: Demographics, circumstances, toxicology and major organ pathology.** Addiction 2008;103:1353-1360.

61. Kocovski L, Duflou J. **Can renal acute tubular necrosis be differentiated from autolysis at autopsy?** Journal of Forensic Sciences 2009;54:439-442.

62. Darke S, Duflou J, Torok M. **Toxicology and circumstances of completed suicide by means other than overdose.** Journal of Forensic Sciences 2009;54:490-494.

63. Darke S, Duflou J, Torok M. **Drugs and violent death: Comparative toxicology of homicide and violent suicide victims.** Addiction 2009;104:1000-1004.

64. Duflou J, Darke S, Easson J. **Morphine concentrations in stomach contents in intravenous opioid overdose deaths.** Journal of Forensic Sciences 2009;54:1181-1184.

65. Kaye S, Darke S, Duflou J. **Methylenedioxymethamphetamine (MDMA)-related fatalities in Australia: Demographics, circumstances, toxicology and major organ pathology.** Drug & Alcohol Dependence 2009;104:254-261.

66. Tu E, Bagnall R, Duflou J, Lynch M, Twigg S, Semsarian C. **Post mortem pathological and genetic studies in "Dead in bed syndrome" cases in type 1 diabetes.** Human Pathology 2010;41:392-400.

67. Darke S, Duflou J. Torok M. **The comparative toxicology and major organ pathology of fatal methadone and heroin toxicity cases.** Drug & Alcohol Dependence 2010;106:1-6.

68. Duflou J, Brouwer I, Darke S. **Levamisole as an adulterant in a cocaine overdose fatality (letter).** Medical Journal of Australia 2010;192:724.

69. Darke S, Torok M, Kaye S, Duflou J. **Cardiovascular disease risk factors and symptoms amongst regular psychostimulant users.** Drug and Alcohol Review 2010;29:371-377.

70. Darke S, Duflou J, Kaye S. **Comparative toxicology of intentional and accidental heroin overdose.** Journal of Forensic Sciences 2010;55:1015-1018.

71. Zreiqat H, Yeng V, James B, Duflou J, Lowe HC. **Probable endothelisation of bare metal stent struts extending from the left main coronary into the aorta.** Journal of Thrombosis and Thrombolysis 2010;30:500-1.

72. Puranik R, Fox O, Sullivan D, Duflou J, Bao S. **Inflammatory characteristics of premature coronary artery disease.** International Journal of Cardiology 2010;145:288-290.

73. Tu E, Bagnall RD, Duflou J, Semsarian C. **Post-mortem review and genetic analysis of sudden unexpected death in epilepsy (SUDEP) cases.** Brain Pathology 2010;21:201-208.

74. Bailey K, Puranik R, Duflou J. **16-year retrospective of fatal aortic dissection: a review of coronial post-mortems (Abstract).** Heart, Lung and Circulation. 2010;19:S10-S10.

75. Najib E, Puranik R, Duflou J, Xia Q, Bao S. **Age related inflammatory characteristics of coronary artery disease (Abstract).** Heart, Lung and Circulation. 2010;19:S21-S22.

76. Krishnamohan S, Duflou J, Puranik R. **10-year retrospective review of myocarditis: an autopsy study (Abstract).** Heart, Lung and Circulation. 2010;19:S38-S38.

77. Tu E, Bagnall R, Duflou J, Semsarian C. **Post-mortem review and genetic analysis of sudden unexpected death in epilepsy (SUDEP) cases (Abstract).** Heart, Lung and Circulation. 2010;19:S231-S231.

78. Darke S, Duflou J, Torok M. **A reduction in blood morphine concentrations amongst heroin overdose fatalities associated with a sustained reduction in street heroin purity.** Forensic Science International 2010;198:118-120.

79. Changsirivathanathamrong D, Wang Y, Rajbhandari D, Maghzal G, Mak W, Woolfe C, Duflou J, Gebski V, dos Remedios C, Celermayer D, Stocker R. **Tryptophan metabolism to kynurenine is a potential novel contributor to hypotension in human sepsis.** Critical Care Medicine 2011;39:2678-2683.

80. Orde M, Puranik R, Morrow P, Duflou J. **Myocardial pathology in pulmonary thromboembolism.** Heart 2011;97:1695-1700.

81. Darke S, Duflou J, Torok M. **Toxicology and characteristics of fatal oxycodone toxicity cases in New South Wales, Australia 1999-2008.** Journal of Forensic Sciences 2011;56:690-693.

82. Tu E, Waterhouse L, Duflou J, Bagnall R, Semsarian C. **Genetic analysis of hyperpolarisation-activated cyclic nucleotide-gated cation (HCN) channels in sudden unexpected death in epilepsy (SUDEP) cases.** Brain Pathology 2011;21:692-698.

83. Najib E, Puranik R, Duflou J, Xia Q, Bao S. **Age related inflammatory characteristics of coronary artery disease.** International Journal of Cardiology 2012;154:65-70.

84. Darke S, Deady M, Duflou J. **Toxicology and characteristics of deaths involving zolpidem in New South Wales, Australia 2001-2010.** Journal of Forensic Sciences 2012;57:1259-1262.

85. Bailey K, Duflou J, Puranik R. **Fatal cases of aortic dissection: an autopsy study.** International Journal of Cardiology 2012;158:148-149.

86. Choudhary P, Canniffe C, Klimis H, Duflou J, Walsh K, Celermajer D. **Aortopathy in Turner's syndrome – a potentially lethal complication (Abstract).** Circulation 2012;126 Supplement, 20 November 2012.

87. Duflou J. **Response to: Tormey WP. Tobacco toxicity ignored as a cause of death. Why? J Forens Legal Med 2012;19:239.** Journal of Forensic and Legal Medicine 2013;20:179.

88. Darke S, Torok M, Duflou J. **Contributory and incidental blood concentrations in deaths involving citalopram.** Journal of Forensic Sciences 2013;58:432-435.

89. O'Meagher S, Choudhary P, Duflou J, Celermajer D, Puranik R. **Causes of death in Tetralogy of Fallot in adults – An autopsy study.** International Journal of Cardiology 2013;168:1547-1548.

90. Darke S, Duflou J, Torok M, Prolov T. **Characteristics, circumstances and toxicology of sudden or unnatural deaths involving very high range alcohol concentrations.** Addiction 2013;108:1411-1417.

91. Evans A, Bagnall R, Duflou J, Semsarian C. **Postmortem review and genetic analysis in Sudden Infant Death Syndrome (SIDS): An 11-year review.** Human Pathology 2013;44:1730-1736.

92. Fang B, Dai A, Duflou J, Zhang X, Puranik R, Bao S. **Age related inflammatory mediators in coronary artery disease (II).** International Journal of Cardiology 2013;168:4839-41.

93. Sweeting J, Duflou J, Semsarian C. **Postmortem analysis of cardiovascular deaths in schizophrenia: a 10 year review.** Schizophrenia Research 2013;150:398-403.

94. Darke S, Duflou J, Torok M, Prolov T. **Toxicology, circumstances and pathology of deaths from acute alcohol toxicity.** Journal of Forensic and Legal Medicine, 2013;20:1122-1125.

95. Duflou J, McNamara B, Cluney R. **A safer method for body restoration following autopsy.** Journal of Forensic Sciences 2014;59:224-225.

96. Bagnall R, Das J, Duflou J, Semsarian C. **Exome analysis based molecular autopsy in cases of sudden unexplained death in the young.** Heart Rhythm 2014;11:655-662.

97. Duflou J. **Right ventricular pathology in pulmonary thromboembolism and athletes (abstract).** Pathology 2014;46:S19-S20.

98. Mahasneh T, Harrington Z, Williamson J, Duflou J, Shin J. **Invasive pulmonary aspergillosis with intravascular large B-cell lymphoma: a rare presentation.** Respirology Case Reports 2014 doi: 10.1002/rcr2.51.

99. Darke S, Torok M, Duflou J. **Sudden or unnatural deaths involving anabolic-androgenic steroids.** Journal of Forensic Sciences 2014;59:1025-1028.

100. Page A, Morrell S, Hobbs C, Carter G, Dudley M, Duflou J, Taylor R. **Suicide in young adults: psychiatric and socio-economic factors from a case-control study.** BMC Psychiatry 2014;14:68 doi:10.1186/1471-244X-14-68.

101. Darke S, Torok M, Duflou J. **Circumstances and toxicology of sudden or unnatural deaths involving alprazolam.** Drug and Alcohol Dependence 2014;138:61-66.

102. Milner A, Page A, Morrell S, Hobbs C, Carter G, Dudley M, Duflou J, Taylor R. **The effects of involuntary job loss on suicide among young adults: evidence from a case-control study.** Australian and New Zealand Journal of Psychiatry 2014;48:333-340.

103. Puranik R, Gray B, Lackey H, Yeates L, Parker G, Duflou J, Semsarian C. **Comparison of conventional autopsy and magnetic resonance imaging in determining the cause of sudden death in the young.** Journal of Cardiovascular Magnetic Resonance 2014;16:44 doi:10.1186/1532-429X-16-44.

104. Krishnamohan S, Fang B, Bao S, Duflou J, Puranik R. **Significance of myocardial inflammation identified at autopsy: a 10 year retrospective review.** Experimental and Clinical Cardiology 2014;20:5707-5712.

105. Rashid I, Klimis H, Duflou J, Sullivan D, Puranik R. **Utility of post-mortem lipid levels in fatal premature coronary artery disease: an autopsy study.** International Journal of Cardiology 2014;174:212-214.

106. Morgan M, Schwartz L, Duflou J. **Hypothermia deaths secondary to glioblastoma multiforme.** Journal of Forensic Sciences 2015;60:511-513 doi:10.1111/1556-4029.12699.

107. Cawsey T, Duflou J, Shannon-Weikert C, Gorrie C. **Nestin positive ependymal cells are increased in the human spinal cord after traumatic injury.** Journal of Neurotrauma 2015;32:1393-1402 doi:10.1089/neu.2014.3575.

108. Darke S, Duflou J, Torok M. **The health consequences of injecting tablet preparations: foreign body pulmonary embolisation and pulmonary hypertension amongst deceased injecting drug users.** Addiction 2015;110:1144-1151 doi: 10.1111/add.12930.

109. Lawson J, Glenn W, Tran D, Ngan C, Duflou J, Whitaker N. **Human papilloma virus and atheromatous coronary artery disease.** Frontiers in Cardiovascular Medicine 2015 May 20, doi: 10.3389/fcvm.2015.00017.

110. Milner A, Page A, Morrell S, Hobbs C, Carter G, Dudley M, Duflou J, Taylor R. **Social connections and suicidal behaviour in young Australian adults: Evidence from a case-control study of persons aged 18 to 34 years in NSW, Australia.** SSM – Population Health 2015;1:1-7 doi:10.1016/j.ssmph.2015.09.001.

111. Bagnall R, Crompton D, Petrovski S, Lam L, Cutmore C, Garry S, Sadleir L, Howell K, Dibbens L, Cairns A, Newton M, Afawi Z, Regan B, Duflou J, Berkovic S, Scheffer I, Semsarian C. **Exome-based analysis of cardiac arrhythmia, respiratory control and epilepsy genes in sudden unexpected death in epilepsy (SUDEP).** Annals of Neurology 2016;79:522-534.

112. Meredith L, Baldock M, Fitzharris M, Duflou J, Dal Nevo R, Griffiths M, Brown J. **A motorcycle fuel tank syndrome: the need for attention to the protection of the pelvis of motorcycle riders.** Traffic Injury Prevention 2016;17:644-649.

113. Haymet A, Edelman J, Seco M, Duflou J, Vallely M, Ng B, Ng M, Wilson M. **Aortic perforation following transcatheter aortic valve implantation (TAVI).** International Journal of Cardiology 2016;207:384-386.

114. Darke S, Duflou J. **The toxicology of heroin-related death: estimating survival times.** Addiction 2016;111:1607-1613.

115. Orchard JW, Puranik R, Semsarian C, Orchard JJ, Samra D, Duflou J, Groenstein P, Young M, Brukner P, Kountouris A. **The prevention of sudden cardiac death in cricketers (Invited review).** Journal of Postgraduate Medicine Education and Research 2016;50:49-58.

116. Bagnall R, Weintraub R, Ingles J, Duflou J, Yeates L, Lam L, Davis A, Thompson T, Connell V, Wallace J, Naylor C, Crawford J, Love D, Hallam L, White J, Lawrence C, Lynch M, Morgan N, James P, DuSart D, Puranik R, Langlois N, Vohra J, Winship I, Atherton J, McGaughran J, Skinner J, Semsarian C. **Sudden cardiac death in the young: results from a prospective population-based bi-national registry.** New England Journal of Medicine 2016;374:2441-2452.

117. Darke S, Duflou J. **Response to Ruan and colleagues concerning "The toxicology of heroin-related death: estimating survival times."** Addiction 2016 doi: 10.1111/add.13587.

118. Darke S, Duflou J. **Characteristics, circumstances and pathology of sudden or unnatural deaths of pathological hoarders.** Journal of Forensic and Legal Medicine 2017;45:36-40 doi:10.1016/j.jflm.2016.11.004.

119. Darke S, Kaye S, Duflou J. **Rates, characteristics and circumstances of methamphetamine-related death: A national 7 year study.** Addiction 2017;112:2191-2201.

120. Darke S, Kaye S, Duflou J. **Commentary: Methamphetamine-related death is an under-addressed public health problem.** Addiction 2017;112:2204-2205.

121. Darke J, Duflou J, Kaye S. **Prevalence and nature of cardiovascular disease in methamphetamine-related death: A national study.** Drug and Alcohol Dependence 2017;179:174-179.

122. Darke S, Lappin J, Kaye S, Duflou J. **Clinical characteristics of fatal methamphetamine-related stroke: a national study.** Journal of Forensic Sciences 2018;63:735-739.

123. Darke S, Duflou J, Lappin J, Kaye S. **Clinical and autopsy characteristics of fatal methamphetamine toxicity in Australia.** Journal of Forensic Sciences 2018;63:1466-1471.

124. Darke S, Kaye S, Duflou J, Lappin J. **Completed suicide amongst methamphetamine users: a national study.** Suicide and Life-Threatening Behavior 2018 doi: 10.1111/sltb.12442.

125. Konecny P, Weatherall C, Ashikari S, Choo L, Patil M, Duflou J, McWhinney B, Pretorius C. **Subcutaneous ivermectin pharmacology in severe Strongyloides hyperinfection: case report with post-mortem analysis.** American Journal of Tropical Medicine and Hygiene 2018 doi: 10.4269/ajtmh.18-0387.

126. Lappin J, Darke S, Duflou J, Kaye S, Farrell M. **Fatal stroke in pregnancy and the puerperium: a case series.** Accepted by Stroke.

127. Ingles J, Bagnall R, Yeates L, McGrady M, Berman Y, Whalley D, Duflou J, Semsarian C. **Concealed arrhythmogenic right ventricular cardiomyopathy in sudden unexplained cardiac death events.** Accepted by Circulation: Genomic and Precision Medicine.

128. Darke S, Duflou J, Kaye S, Farrell M, Lappin J. **Psychostimulant use and fatal stroke in young adults: a national study.** Submitted to Journal of Neurology, Neurosurgery, and Psychiatry.

129. Darke S, Duflou J, Kaye S, Farrell M, Lappin J. **Body mass index and fatal stroke in young adults: a national study.** Submitted to Journal of Neurology.

D. EXAMPLES OF RECENT PODIUM PRESENTATIONS & POSTER PRESENTATIONS:

1. Duflou J, Williams J, Howson K. **Autopsy findings in the morbidly obese.** Presented at the 64th Annual Meeting of the American Academy of Forensic Sciences in Atlanta in February 2012.

2. Duflou J, Howson K, Williams J. **Normal isn't what it used to be - the spectrum of liver abnormalities in an Australian autopsy population.** Presented at the 64th Annual Meeting of the American Academy of Forensic Sciences in Atlanta in February 2012.

3. O'Meagher S, Choudhary P, Duflou J, Celermajer D, Puranik R. **Causes of death in repaired Tetralogy of Fallot in adults.** Presented at the Annual Scientific Meeting of the Cardiac Society of Australia and New Zealand in Brisbane in August 2012.

4. Evans A, Bagnall R, Duflou J, Semsarian C. **Post-mortem review and genetic analysis of Sudden Infant Death Syndrome (SIDS) cases.** Presented at the Annual Scientific Meeting of the Cardiac Society of Australia and New Zealand in Brisbane in August 2012.

5. Wheatley J, Wilson K, Cass D, Manglick P, Duflou J, Bilston L, Brown J. **Risk factors for serious injury to young child occupants in motor vehicle accidents.** Presented at the Australian College of Road Safety Conference in Sydney in August 2012.

6. Duflou J. **Australian experience of sudden cardiac death.** Presented at the 29th International Congress of the International Academy of Pathology in Cape Town in October 2012.

7. Cawsey T, Duflou J, Shannon-Weickert C, Gorrie C. **Response of human spinal cord ependymal cells to traumatic injury.** Presented at the Australian Neuroscience Society Conference in Melbourne in February 2013.

8. Wang R, Sy J, Duflou J. **Intravascular large B-cell lymphoma complicated by invasive aspergillosis – a case report.** Presented at the International Academy of Pathology, Australasian Division, Meeting, in Sydney in May 2013.

9. Fang B, Duflou J, Puranik R, Bao B. **IL-27 and IL-29 in coronary artery disease in premature cardiac death.** Presented at the Annual Scientific Meeting of the Cardiac Society of Australia and New Zealand in Gold Coast in August 2013.

10. Sweeting J, Duflou J, Semsarian C. **Cardiovascular deaths in schizophrenia: a post mortem analysis.** Presented at the Annual Scientific Meeting of the Cardiac Society of Australia and New Zealand in Gold Coast in August 2013.

11. Rashid I, Klimis H, Duflou J, Sullivan D, Puranik R. **Utility of post-mortem lipid levels in fatal premature coronary artery disease: an autopsy study.** Presented at the Annual Scientific Meeting of the Cardiac Society of Australia and New Zealand in Gold Coast in August 2013.

12. Konecny P, Adhikari S, Choo L, Patil M, Duflou J, McWhinney B, Pretorius C, Weatherall C. **Parenteral ivermectin pharmacology: first report with post-mortem analysis.** Presented at Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC 2013) in Denver, CO, in September 2013.

13. Duflou J. **Right ventricular pathology in pulmonary thromboembolism and athletes.** Presented at Pathology Update 2014 of the Royal College of Pathologists of Australasia, Melbourne in February 2014.

14. Das J, Bagnall R, Duflou J, Semsarian C. **Whole exome analysis of sudden unexplained death in the young.** Presented at Heart Rhythm 2014, in San Francisco CA, in May 2014.

15. Duflou J. **What the autopsy contributes to sudden cardiac death evaluation.** Presented at International Clinical Cardiovascular Genetics Conference 2014 in Melbourne, in August 2014.

16. Duflou J. **Is there a common mechanism for right ventricular pathology in pulmonary thromboembolism, endurance athletes and arrhythmogenic right ventricular cardiomyopathy?** Presented at the 6th Biennial Meeting of the Association for European Cardiovascular Pathology in Paris France, in October 2014.

17. Duflou J. **Alcohol and the pathologist.** Presented at the New Zealand Annual Scientific Meeting of the Royal College of Pathologists of Australasia in Hamilton NZ, in October 2014.

18. Duflou J. **Death by restraint.** Presented at the New Zealand Annual Scientific Meeting of the Royal College of Pathologists of Australasia in Hamilton NZ, in October 2014.

19. Brown J, DeRome L, Meredith L, Fitzsharris M, Baldock M, Savino G, Ferndez R, Griffiths M, Dal Nevo R, Chee D, Oomens M, Duflou J. **A qualitative analysis of motorcycle crash and injury causation factors identified during multidisciplinary review of in-depth crash investigation data.** Presented at Australasian Road Safety Research, Policing and Education Conference 2014 in Melbourne, in November 2014.

20. Duflou J. **Anabolic-androgenic steroids in medico-legal death investigation cases.** Presented at the 7th European Academy of Forensic Science Conference in Prague, Czech Republic, in September 2015.

21. Duflou J, Darke S, Torok M. **Can cannabis consumption cause sudden death?** Presented at the 7th European Academy of Forensic Science Conference in Prague, Czech Republic, in September 2015.

22. Duflou J. **The role of the autopsy in sudden cardiac death.** Presented at the 8th Asia Pacific Heart Rhythm Society meeting in Melbourne in November 2015.

23. Duflou J. **Pathological findings in arrhythmogenic right ventricular cardiomyopathy (ARVC).** Presented at the International Clinical Cardiovascular Genetics Conference 2016 in Brisbane in May 2016.

24. Duflou J. **Death certification.** Presented at the Annual Conference of the Veterans Review Board in Sydney in June 2017.

25. Duflou J. **Plenary speaker - Forensic pathologist perspective.** Presented at the 15th International Conference for Emergency Nurses (ICEN 2017) in Sydney in October 2017.

26. Ackerman M, Haan E, Duflou J, Haqqani H, Skinner J, Macciocca I. **Panel discussion - Arrhythmias and sudden cardiac death.** Presented at the International Clinical Cardiovascular Genetics Conference 2018, in Brisbane in May 2018.

27. Duflou J. **Key role of the forensic pathologist in investigating arrhythmogenic cardiomyopathies and sudden death.** Presented at the International Clinical Cardiovascular Genetics Conference 2018, in Brisbane in May 2018.

28. Ingles J, Bagnall RD, Yeates L, McGrady M, Berman Y, Whalley D, Duflou J, Semsarian C. ***PKP2* loss-of-function variants in probands with sudden unexplained cardiac death events.** To be presented at the annual conference of the American Heart Association (AHA 2018) in Chicago in November 2018.

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24 January 2019

Professor Johan Duflou
PO Box 331
MANLY NSW 1655

By email: jduflou@forensicmedicine.com.au

Dear Professor,

Re: Kathleen Megan Folbigg

We advise that we act on behalf of Ms Kathleen Megan Folbigg in relation to the Inquiry ordered into her convictions by the NSW Governor on 22 August 2018.

Background

Please find **enclosed** a copy of the Petition to the Governor of New South Wales containing relevant background information.

Report

We have been instructed to seek your expert opinion in relation to the cause of death of Caleb Gibson Folbigg, Patrick Allen Folbigg, Sarah Kathleen Folbigg and Laura Elizabeth Folbigg.

I draw your attention to the following Court Rules, copies attached:

1. UCPR 31.23 Code of Conduct;
2. UCPR 31.27 Experts' reports; and
3. UCPR Schedule 7 – Expert witness code of conduct.

Your report should be addressed to me at Cardillo Gray Partners.

In order for the report to be of value in the current proceedings, your report must state:-

1. Your name and address.
2. That you comply with Regulation 31.27,
3. That as an expert, you have specialised knowledge based upon your training, study or experience set out in the report,
4. Sets out the opinion that you hold as an expert, and which is wholly or substantially based upon that specialised knowledge, and
5. Set out your reasons for your opinion, and to the extent that you have relied on any scientific study or other literature, refer to that literature by footnote or bibliography.
6. Attach a copy of this letter, the letter of instruction and its attachments to your report;
7. Complete and attach the Certificate - Expert Report;
8. Complete and attach the Expert Certificate, s 177 Evidence Act.

Attached Documents

We enclose for your information the following documents:

1. Autopsy reports for Caleb, Patrick, Sarah and Laura Folbigg;
2. Black Book - list of transcripts (incorporating material tendered by Counsel Assisting to be found at https://www.folbigginquiry.justice.nsw.gov.au/Pages/documents.aspx);
3. Blue Book - list of exhibits and MFIs (Marked for Identification) from the original trial;
4. Report of Professor Stephen Cordner;
5. Peer review of Professor Stephen Cordner's report by Professor Michael Pollanen;
6. Report of Professor Carola Vinuesa;
7. Report of Dr Cala dated 26.11.18;
8. Material provided by the Crown Solicitors Office relating to Cardiology, Forensic Pathology, Genetics, SUDI and other documentation; and
9. Schedule of SIDS literature.

Questions

In preparing your report we would be pleased if you would address the following questions:

1. Provide an opinion on the standard of the autopsies conducted in each case bearing in mind best practice at the time of each autopsy.
2. Provide your opinions about the causes of death in each case. In providing these opinions, also list the factual matters you rely upon and the assumptions that you make.
3. Have there been scientific advances since 2003 that would have changed the way the autopsies were conducted?
4. Is there any evidence that would reasonably allow a forensic pathologist to conclude that any of the children had been smothered?
5. What definition of SIDS do you rely upon? Please detail the definition.

6. In cases where SIDS is found, is there any basis for a forensic pathologist to link such a finding to a lack of oxygen?

7. Is there any evidence that would allow a forensic pathologist to conclude that any of the children died as a result of a lack of oxygen?

8. Are there any opinions provided by Professor Cordner in his report that you disagree with? If there is, please provide details and reasons for your disagreement.

9. What is your understanding of the term 'asphyxia' and how should it properly be used in forensic pathology? Does it have any application in these cases?

10. In the case of Laura Folbigg, very shortly before her death she was given the drug 'Demazin' and was suffering from a cold. Noting recent findings in respect of the use of Demazin, please comment on any impact it may have had on her. Also comment on the role a viral infection may have had.

11. As you are aware, genome sequencing is being undertaken in respect all of the children. If genome sequencing reveals no genetic abnormality, does this exclude the possibility that there may still be a genetic abnormality involved in the deaths?

12. The Inquiry is undertaking a literature review on the number of unexplained infant deaths in the one family. What is your opinion about forensic pathologist's relying on such literature? Please detail your reasoning including a description of 'Meadows Law' and its role in forensic pathology.

13. In your opinion, are there cases that would fall into the category of 'unusual' or even 'extraordinary' that are not reported in the scientific literature?

14. We have provided you some literature about the link between opioid peptides (b-casomorphin-7) found in cows milk and baby formula, and respiratory events and SIDS. Please provide an opinion about the role that opioid peptides may have played in the deaths.

15. Accepting that there is some guidance provided to expert witnesses in the UCPR, are there any other guidelines provided by the Royal College of Pathologists of Australasia or other relevant body? If yes, please provide them.

16. Are there ethical limits placed on forensic pathologists when they provide reports or give evidence in court? Of particular concern is the obligation to research an issue prior to providing an opinion, and to provide the literature relevant to that opinion.

17. What role does a forensic pathologist have in providing an opinion about possible causes of death?

18. The Supreme Court of Victoria has issued Practice Note No 2 'Expert Evidence in Criminal Trials.' This includes Item 1 'Forensic Pathology: Standard Questions to Address the Cause of Death in An Alleged Homicide.' What utility do these guidelines have in these cases?

19. We understand that you provided a brief report to the NSW Health Department at their request some years ago. Please attach that report and provide any relevant explanatory comments.

If any question that we have posed does not make sense to you from the perspective of your specialty, kindly advise us of the issue that it presents and we will attempt to clarify it.

As you are aware genetic testing is being carried out on available samples for all the children. As soon as the test results are made available we will provide them to you so that you can amend or expand on your report.

We undertake to be responsible for your professional fee and look forward to conferring with you shortly.

Would you please address your tax invoice as follows:

Kathleen Megan Folbigg
CO/ Cardillo Gray Partners
PO Box 409
Newcastle NSW 2300

Should you have any questions or wish to discuss this matter please do not hesitate to contact us on (02) 4910 0677.

Yours Faithfully
CARDILLO GRAY PARTNERS

Stuart Gray
Partner

Encl.:

- The report by Professor Stephen Cordner was produced at the request of the University of Newcastle Legal Centre.

- The University of Newcastle Legal Centre applied for access to documentation and materials held by DOFM in December 2013, and Professor Cordner attended DOFM Sydney in July 2014 to review the microscopy slides of three of the four fatalities (Patrick, Laura and Sarah).

- Select slides were sent to VIFM at the request of Professor Cordner in December 2014 for further examination, and were subsequently returned.

- Professor Cordner's report is largely based on review of select material, including the autopsy reports, verbal evidence of Dr Cala, and review of microscopy slides, particularly of Laura. A full list of material available to Professor Cordner is found at pages 4 and 5 of his report. Please note that Professor Cordner did not avail himself to any material available at DOFM with the exception of the microscopy slides.

- **Major relevant conclusions of Professor Cordner:** Professor Cordner's report considers multiple areas of the case against Kathleen Folbigg, and in summary comes to the following major conclusions:
  - Much of the evidence adduced by the Crown on the topic of asphyxia and infant deaths is misconceived, flawed and faulty.
  - There is no forensic pathology support for the contention that any or all of the children were victims of homicide, let alone having been smothered.
  - All deaths are compatible with being the result of natural processes.
  - Smothering cannot be ruled out in any cases.
  - The term used by the Crown, "acute catastrophic asphyxiating event" has no meaning in forensic pathology.
  - Contrary to the evidence of Dr Cala at the Trial:
    - There are frequently signs of smothering in infants and children who have been smothered.
    - The extent of myocarditis in Laura is extensive and likely the cause of death.
  - Professor Cordner's opinion as to the cause of death of the four children compared to the various autopsy pathologists' views is as follows:

Deceased	COD Cordner	Original COD
Caleb	Sudden Infant Death Syndrome, Category II	Sudden Infant Death Syndrome
Patrick	The consequences of an unexplained ALTE	Asphyxia due to airways obstruction due to epileptic fits due to encephalopathic disorder (underlying cause not determined on investigation)
Sarah	Sudden Infant Death Syndrome, Category II	Sudden Infant Death Syndrome
Laura	Myocarditis	Undetermined

  Comment: Effective, therefore, the only case where there is a significant difference of opinion between the original pathologist and Professor Cordner as to the cause of death is that of Laura Folbigg.
  - None of the autopsies were conducted to an adequate standard, operative at the time. Professor Cordner provides a list "of the procedural and data elements which are required today at VIFM which are apparently missing from all four death investigation." However, this is not correct. However, many of the listed missing

elements were in fact performed on the two cases examined in Sydney, for example photography of the body and radiography, and others may reasonably have been considered but not performed as a result of specific circumstances in the case.

- **Limited critique of Professor Cordner's report:** There are a number of criticisms which can be made of Professor Cordner's report:
  - Professor Cordner's analysis and report are written with the benefit of medical knowledge having progressed over the last 15 to 25 years, and in the last 12 years since the Trial. Despite his attempts at applying standards operative at the time of the various autopsies, this is not the case in many parts of his report.
  - Cause of death determination is always complex and open to opinion. Indeed, Professor Cordner indicates that a not unreasonable cause of death in the case of Laura would have been "Undetermined" (page 80). I note that Professor Cordner has written on the topic of the difficulty in making an accurate determination of cause of death – I agree with his observations.
  - To buttress his opinion as to the presence of lethal myocarditis, Professor Cordner showed photomicrographs of heart tissue of Laura to pathologists at VIFM. Such photographs would not be expected to be representative, but would typically illustrate the more dramatic and severe manifestations of the pathology. It is therefore to be expected that there would have been significant bias by those pathologists to give a diagnosis of myocarditis.
  - Despite lengthy discussion of cardiac channelopathies and their role in sudden death in infancy, there appears to be no suggestion that the deceased's retained tissues should be examined for the presence of such genetic abnormalities, nor is there suggestion that family members should be examined clinically. NOTE: I am not certain about this, but I recall molecular investigations for channelopathies having taken place at VIFM some considerable time back in relation to these fatalities.

- **Possible implications for Dr Cala:**
  - The report by Professor Cordner implies strongly that Dr Cala has not been impartial in his assessment of Laura's death. In my opinion, there is definite myocarditis present, and this could reasonable have been the cause of death. Given the preceding events in this family, however, I am of the view that the cause of death was reasonably attributed by Dr Cala as "Undetermined". However, I share Professor Cordner's opinion that Dr Cala's view on the likelihood of death not being due myocarditis is one that would not be shared by most forensic pathologists.
  - In 2014, I co-authored a paper on the topic of incidental vs. causative myocarditis (see attached). This paper identified similar problems to those discussed by Professor Cordner, and emphasises the problem of not being able to determine, on the basis of autopsy cardiac pathology alone, whether a person has died of myocarditis or died of another cause. Although this paper was published well after the Folbigg trial, I believe this type of information was known to forensic pathologists, at least on an empirical basis. I therefore agree with the implicit views of Professor Cordner that there may have been bias on the part of Dr Cala.
  - [redacted]
  - **Possible implications for DOFM:**
    - Professor Cordner has without a doubt a solid and very highly regarded reputation in the field of forensic pathology. This is a well-deserved reputation, and consequently it can be expected that much weight will be given to his report.

- In my opinion there is a strong likelihood of adverse comment being made in the media and elsewhere against DOFM when the contents of this report is released.
- It is strongly recommended that all four autopsies be reviewed in detail, in anticipation of this being required at some time in the future. This would have significant resource implications, given the large amount of material involved in the case.