EXHIBIT M

Forensic Analytical Science Service
Forensic Medicine Newcastle

NSW GOVERNMENT Health Pathology

Inquiry into the Convictions of Kathleen Megan Folbigg,
Level 2
Industrial Relations Commission
47 Bridge St
SYDNEY NSW 2000

26 November 2018

Dear Sir/Madam,

INQUIRY INTO THE CONVICTIONS OF KATHLEEN MEGAN FOLBIGG

I refer to the 7 page letter from Ms Deanne Tadros, NSW Ministry of Health, dated 7 November 2018 in relation to the above Inquiry. I acknowledge that I have read the *Expert Witness Code of Conduct* in Schedule 7 of the *NSW Uniform Civil Procedure Rules 2005*; and agree to be bound by the Code.

I have been asked to prepare a report for the Inquiry setting out:

1. A response to the undated report of Professor Stephen Cordner and;
2. Whether there have been any medical advances since 2003 relevant to my area of expertise (Forensic Pathology), with advice whether the views I expressed at trial in 2003 have changed or remain the same and the reasons therefore.

I have not been able to read all the material provided prior to compiling this report, given its quantity and the time frame of 19 days for a reply. This is therefore a preliminary report and should a further report be requested, that could be done in early 2019. (I am taking leave from 28 November until 24 December. I will not be available during that period.)

Introductory remarks:

It is simply remarkable and unthinkable that the four Folbigg children lived their entire lives in Australia from 1989 to 1999, and all are now deceased. Even if the family was from an exceptionally deprived economic background and had been residing in a location with no medical facilities, the casual acceptance of a 100% mortality rate in the one family from purported natural disease or diseases, without any further scrutiny or investigation is, in my view, a most unsatisfactory situation.

I will attempt to respond to Professor Cordner's report by providing a synopsis of the significant medical events in the lives of, and the circumstances surrounding the deaths of all four Folbigg children (see below).

I am greatly surprised by the vast amount of medical information supplied to Professor Cordner in comparison with anything I received. I sought out as much medical information as had been compiled and was available at the time, but this was much less than the information he received and summarised.

The current situation of Sudden Infant Death Syndrome or "SIDS":

A medical syndrome is an aggregation of symptoms and physical signs that collectively constitute a clinical entity. The term SIDS is purely a descriptive one that lacks both symptoms and physical signs. It is a "label" with no actual medical meaning. SIDS is not a disease or illness but an invented term which originated in North America years ago after the term "cot death" was abandoned. It was an attempt to better explain the sudden deaths of infants from presumed natural causes, when they were found deceased during sleep. SIDS as a term was an attempt to provide parents/caregivers with "an explanation" for the death, and to allow them to grieve with reassurance that the death was not preventable or foreseeable.

The recent sub-categorisation of SIDS into various "subgroups" has been for academic purposes as opposed to being for purposes which primarily set out to improve our understanding of this mysterious condition. I do not believe this subcategorization has been very helpful and may be just confusing. Terminology and definitions surrounding SIDS have evolved over many years to this reasonably well agreed upon current definition:

"*The sudden and unexpected death of an infant less than one year of age, with the onset of the lethal episode apparently occurring during sleep; that remains unexplained after a thorough investigation including performance of a complete autopsy, review of the circumstances of death and the clinical history*". (NSW Child Death Review Team: Annual Report. NSW Government Publication: Ombudsman 2014*)*

There were a number of "sentinel" earlier publications (1,2,3) that were the first articles which focussed on issues surrounding possible inflicted (non-accidental) trauma on children. Against this background were other atraumatic infant deaths, the "typical case" being a previously healthy infant, usually aged around 2 months up to around 4 months of age, who dies suddenly and unexpectedly during sleep. The scene of death does not reveal anything of a suspicious nature. This is usually done by police, but on occasions, a forensic pathologist may attend the death scene. No convincing cause of death is found after a thorough autopsy. A review of the medical details of the infant and family reveal nothing of significance. Ancillary testing such as toxicology, microbiology studies or neuropathology fails to shed light on the death and results are all "negative".

There are believed to be "risk factors" for the development of SIDS, such as premature birth, maternal smoking, bed-sharing and prone sleeping position. The "back to sleep" campaign (placing babies on their backs) appears to have had some reduction in SIDS rates worldwide.

Some doctors (usually paediatricians or pathologists) do not believe SIDS "runs in families" whilst others do; some question whether a diagnosis of SIDS can occur twice or even more in a family, whilst others hold this view. Unsurprisingly, there has been intense disagreement on the subjects of sudden infant death and SIDS for many decades.

SIDS has recently fallen into disuse in favour of Sudden Unexplained Death in Infancy or "SUDI". This is perhaps another "label" that may carry no actual meaning either. It may be however an improvement on the term SIDS as in my opinion it removes the "syndrome" aspect from the term but carries the notion of "unexplained". That is, questions may still be forthcoming in the future concerning a sudden infant death which the SIDS diagnosis tended not to allow for. SIDS as a diagnosis closed down investigations, as opposed to started or continued them. This may have been an unintended consequence of such terminology.

Documents received:

I have received a number of documents to assist me in this report which I set out as follows:

1. Autopsy Report of LAURA ELIZABETH FOLBIGG, dated 26th July 1999.
2. Neuropathology Report of LAURA ELIZABETH FOLBIGG, dated 7th June 1999 by Dr M Rodriguez.
3. Article, "Distinguishing Sudden Infant Death Syndrome from Child Abuse Fatalities" (RE0036), American Academy of Pediatrics, Volume 107, No 2, pages 437-441.
4. Letter from Dr Allan Cala re IL-10 Gene Theory, dated 19th March 2003.
5. Expert Certificate of Dr Allan Cala, 28th March 2003.
6. Report of Professor Roger Byard, dated 14th April 2003.
7. Transcript of evidence of Dr Allan Cala at trial, pages 705-765, 15th-16th April 2003.
8. Transcript of evidence of Professor Byard at trial, pages 1195-1259, 7th May 2003.
9. Report and Opinion in the case of Kathleen Folbigg by Professor Stephen Cordner, undated.
10. Peer Review of Professor Cordner's Report on the Folbigg Case by Professor Michael Pollanen, dated 1st June 2015.
11. Chapter from book, "SIDS – Sudden Infant and Early Child Death: The Past, the Present and the Future", edited by JR Duncan and Roger W Byard, University of Adelaide Press, 2018, Chapter 24, The Autopsy and Pathology of Sudden Infant Death Syndrome, by Roger W Byard.

CALEB GIBSON FOLBIGG:

Of note, I received the original autopsy file in relation to this infant's death for the first time ever on 7/11/2018. Prior to that date, I had not seen any of the material. Within the file is:

- P79A, Report of Death to the Coroner dated 20th February 1989 (one page),
- Interim Post Mortem Report (Dr R Cummings) dated 20th February 1989,
- Final autopsy report, three pages, dated 9th May 1989,
- One page of handwritten notes, Dr Cummings

- One page report from Division of Analytical Laboratories dated 1st May 1989,
- Identification Statement by Snr Const KJ Bryant,
- One page Certification of Death,
- Exhibit List, one page,
- Form 21 Order to Medical Practitioner to Perform a Post Mortem Examination dated 21 February 1989,
- One page Summons to a Witness dated 20 October 1999 (J Abernethy, Senior Deputy State Coroner NSW),
- Exhibit List dated 21/10/1999, and
- One page letter from Snr Const Joe Lavin to Dr Bishop, Hunter Area Pathology dated 14 October 1999.

The P79A provides the following information in relation to this child on the date of its death as follows:

"*About 1.00am on Monday, the 20 February 1989, the child was fed by his mother. He was then put to bed in adjoining bedroom. About 2.53am (20.2.89) the mother awoke and checked the child and found him to be cold and apparently dead, she found a small amount of blood and throth (sic) around the child's mouth. The father was alerted and began CPR till ambulance officers arrived but to no avail. The body was conveyed to the Newcastle Hospital where life was pronounced extinct at 4.00am by Dr Sandy Chapman. The child had been taken to a Dr Springthorpe of Watt Street Newcastle for treatment for a 'Lazy Larynx'. No medication was given.*"

Dr Cummings then performed an autopsy examination several hours later commencing at 11.45am on 20th February 1989. I quote from the autopsy report:

"*EXTERNAL EXAMINATION*

The body was that of a well-nourished and normally developed male child appearing the stated age of 19 days. Height 55cm (crown-heel), weight 3970 grams. Hair was brown, eyes were blue. Rigor mortis was present and post mortem staining was seen over the back. There were no external signs of injury and the infant appeared to have been well cared for.

INTERNAL EXAMINATION

Cardiovascular System
Pericardial sac was normal. The heart (25 grams) was of normal size and was normally developed. Valves and chambers were normal. Myocardium showed no evidence of disease. Great vessels were normal.

Respiratory System
Pleural cavities were normal. Larynx, trachea and bronchi were unremarkable. The lungs (left 34 grams, right 53 grams) were somewhat mottled on their pleural surfaces. Cut surfaces were somewhat moist but otherwise unremarkable".

The Alimentary System, Haemopoetic System, Genitourinary System, Endocrine System and Central Nervous System were all examined with no abnormalities detected. The brain weighed 465 grams and showed no unusual features on its meningeal and sectioned surfaces.

Histological examination of the major organs was conducted (heart, lungs, liver, kidney, spleen, thymus, adrenal gland and lymph nodes). The heart was said to be normal. The lungs *'are congested and in places show incomplete aeration, in other sections their alveoli contain extravasated red blood cells and a small amount of eosinophilic exudate'*.

The liver, kidney, spleen, thymus, adrenal gland and lymph nodes were all said to be normal.

A summary of the autopsy findings was "*both lungs were moderately moist. No other unusual features were seen.*"

The cause of death was given as 1(a) Direct Cause – Sudden Infant Death Syndrome ("SIDS").

The toxicology report indicated that "routine screening tests for poisons were negative." Dr Cummings finally comments "*The gross and microscopic findings are consistent with 'cot death'.*"

Professor Cordner's opinion:

Page 83:

"Caleb's death was diagnosed at the time as SIDS. Dr Cala gave evidence that he would diagnose his death as Undetermined. According to approach of Krous et al 2004, the correct diagnosis for Caleb's death would be Sudden Infant Death Syndrome Category 2. This is because of the age of death being 19 days. Had Caleb died two days later his death by contemporary standards would have been properly diagnosed as SIDS (Category 1b) because of the incomplete level of investigation/documentation according to today's standards."

Page 90. *"Caleb's death is best regarded as SIDS (Category 2)."*

My opinion:

Even by today's standards, after a thorough scene examination, full autopsy and ancillary testing, it is my view that a diagnosis of SIDS is not correct. SIDS is, after all, a diagnosis of exclusion. That is, this diagnosis **may** be made only if there are no suspicious circumstances surrounding the death, the scene examination, the subsequent autopsy and the results of ancillary tests are all subsequently "negative".

In the case of the apparent sudden death of an infant of 19 days (essentially still a "neonate" being under one month old), one would be initially searching for possible underlying infectious, congenital, metabolic or other natural causes of death as well as excluding suspicious scene findings or suspicious circumstances of death.

Infants who die suddenly and unexpectedly of apparent natural but unknown causes are typically 2-4 months of age, with 90% dying within 6 months of age (23). Ages younger than 2 months are unusual for SIDS. In my opinion, an infant that dies aged less than one month of age would not be a SIDS death on the grounds of being too young. It is not SIDS but something else. Likewise, the sudden unexpected death of an infant aged 12 months or older would be considered too old for SIDS. This is not an infant but a "toddler". The current SIDS definition excludes this age and older.

The P79A describes the presence of *"a small amount of blood and throth (sic.) around the child's mouth. The father was alerted and began CPR, till ambulance officers arrived but to no avail."*

This tells me that when the mother discovered the infant to be cold and apparently dead there was already a small amount of blood and (presumably) froth ("bubbly fluid") around the infant's mouth. CPR only occurred for the first time when the father was alerted and this was continued until ambulance officers arrived. Dr Cummings' autopsy findings make no reference to any blood or froth anywhere in the airways although there is a description of lungs being *"somewhat mottled on their pleural surfaces."* This, therefore, tells me that the finding of blood and froth must have been confined to the upper airway as there is no mention anywhere in the report of blood or frothy fluid anywhere in the respiratory system.

<u>The finding of blood and frothy fluid on or around the external airway may be highly significant.</u> Whilst I cannot draw absolute conclusions from their presence, it is of concern that both were discovered prior to any CPR being undertaken, ie, CPR could not have caused the presence of blood and froth. I see no further comments anywhere in the report of Professor Cordner in relation to this finding. It may have been interpreted as an "innocent" or insignificant finding and was therefore disregarded. It is a most unusual finding if an infant is found deceased and has not been bed-sharing. It could well be a highly significant finding that requires an explanation.

I do not know the position of the infant when he was discovered by his mother at 2.53am. There is no mention that he was "face down" with his head in a pillow or the mattress. There is no mention of any blood elsewhere about the face, on the clothing or on the bed linen. I presume that from the description, the mother found him with blood and froth around the external airway and he was therefore found face up (supine) as otherwise his face would have been concealed, at least initially until he was rolled over. If he was face down and found with blood and froth around the mouth, I would expect blood to be on the pillow or sheets nearby. That does not appear to have occurred. This raises the question then of what caused the blood and froth when less than two hours earlier, he was fed milk and had been apparently well.

Early decomposition changes can sometimes cause a small amount of blood to be present at the external airways, however, the infant was found less than two hours after a feed of milk. Decomposition could be therefore reasonably excluded given the less than two hour interval between feeding and subsequent discovery.

The infant was sleeping by himself in a separate bedroom and therefore co-sleeping can be excluded. In the article by HF Krous (4), I quote *"Oronasal blood not attributable to cardiopulmonary resuscitation occurs rarely in SIDS when the infant is sleeping supine in a safe environment. Bed sharing may place infants at risk of suffocation from overlaying. Oronasal blood observed before cardiopulmonary resuscitation is given is probably of oronasal skin or mucus membrane origin and may be a sign of accidental or inflicted suffocation. Sanguineous secretions that are mucoid or frothy are likely of remote origin, such as lung alveoli. The use of an otoscope to establish the origin of oronasal blood in cases of sudden infant death is recommended."*

Although no photographic evidence exists, the description is of blood and froth around the infant's mouth. This is different from oro-nasal secretions which are observed quite commonly in cases of sudden infant death. The finding of frank blood at the external airway (nose and mouth) is unusual and of concern. In this case, bed sharing, decomposition, congenital heart disease, pulmonary oedema and a face down position after death can be excluded from having occurred. The findings as described in the P79A are therefore of concern. This is not only **my** concern, but the medical literature (6, 8, 9,10,13,16, 22) also supports concern with respect to the possibility of deliberate or inflicted smothering with such a finding.

In my opinion, this infant's death is not satisfactorily explained by "SIDS". I am concerned an external agent may have caused the blood and froth around the mouth. The so-called "lazy larynx" (*laryngomalacia*) is unrelated to the death. It is a relatively benign abnormality of the larynx that is discovered in the neonatal period which the infant outgrows as cartilage strengthens. It generally disappears with "masterly inactivity" ie "observe for now".

PATRICK ALLAN DAVID FOLBIGG:

Patrick was born on 3 June 1990 after spontaneous onset of labour with artificial rupture of the membranes by vertex presentation at thirty-nine weeks gestation. The history of the pregnancy and the delivery have already been documented in Professor Cordner's report. Sleep studies were arranged for 14 June 1990. An ECG and serum electrolytes were normal. The sleep study was normal. A barium swallow showed no gastro oesophageal reflux. Contrast in the nose suggested inco-ordination of swallowing. Patrick attended his GP on a number of occasions for vaccinations, mild viral infections and other childhood illnesses. More detailed illnesses are apparently documented in hospital records (not seen).

On 17 October 1990 Patrick was put to bed in his cot at about 8.30pm. He was seen to be lying on his back two hours later covered with a sheet and blanket. He was heard to be coughing at 3.00am when his mother attended to him. She was then apparently alerted at 4.30am because she heard him make "gasping sounds" and noted that he was blue around

the lips, appeared lifeless, was "floppy" and making minimal respiratory effort. (This episode has been referred to as an "*Apparent Life Threatening Event*", or ALTE. A precise cause for this has never been established.)

The mother screamed and the father awoke to see the mother standing in front of the cot with the rail in the "up" position and Patrick lying on his back with the covers pushed down near his feet. Resuscitation was commenced and an ambulance was called for which arrived 20 minutes later. Patrick was revived slightly when paramedics gave oxygen. He was recorded as having respiratory difficulties and was pale around the face and listless. He showed a "tracheal tug" and intercostal recession. (These are non-specific physical signs of apparent respiratory difficulty)

He was admitted to hospital and was lethargic, cyanosed and responsive only to painful stimuli. There was no evidence of trauma or any evidence to suggest any underlying serious illness such as meningitis. Glucose was in the urine but in the absence of a high blood sugar with protein in the blood was believed to be a response to "*an acute asphyxiating event".*

A chest X-ray later reported signs of possible bronchiolitis. Viral studies were subsequently negative. By the following day, he had apparently improved to his "normal self". An EEG on 18 October 1990 was apparently normal. An ECG on the same day was essentially normal with sinus rhythm at about 160 per minute with "complexes appear normal on those leads that can be identified".

At 9.00pm that day he developed a generalised seizure and was given Diazepam (Valium). Following further fits in hospital he was started on Phenobarbitone (a barbiturate drug used to treat seizures). A lumbar puncture on 20 October 1990 was normal. A metabolic screen was collected. A CT scan of the brain showed hypodense areas in both temporal and occipital lobes. He was commenced on Phenytoin (Dilantin) and Acyclovir to cover the possible of herpes simplex encephalitis, with investigations later performed for herpes simplex virus being subsequently negative.

He was discharged on 29/10/90 with a diagnosis of "Seizure Disorder and a Respiratory Tract Infection". He had several other admissions in relation to seizures associated with a febrile illness and a respiratory tract infection. A repeat EEG showed multifocal epileptogenic foci. There was a progressive deterioration from his two previous EEGs. A CT scan on 5 November showed generalised loss of brain substance with patchy enhancement in both occipital lobes. High density areas in the pre-contrast scan was thought to be due to "*dystrophic calcification"* (calcification in areas of dead tissue, or necrosis).

These films were subsequently seen by Professor de Silva who suggested the possibility of underlying child abuse such as "*shaking injury*". Patrick was discharged on 10 November 1990 with a provisional diagnosis of a Seizure Disorder perhaps due to an Encephalopathy. A later examination showed he had lost the ability to fix on a face or to follow and was found to have a degree of cortical blindness.

A cardiac ultrasound on 16/11/90 showed no evidence of intracardiac thrombus. He developed gastroenteritis on 18/11/90 and stools collected on 20 November were positive for rotavirus. He was discharged on 22 November 1990. Over the next few months he suffered a further oculogyric crisis and was admitted to hospital but discharged on 22 December. On 12 February 1991 he had a fever during the evening with a possible seizure at that time.

He slept well overnight and played with his father early in the morning of 13 February. The father left for work at 7.30am and Patrick appeared his normal self and was eating. At 10.00am that morning the mother phoned him at work screaming *"It's happened again!"* The father drove straight home and saw ambulances arriving. Patrick was lying on his back in his cot with the inside rail in the up position. He again began resuscitation. Patrick was limp, blue around the lips and warm to the touch.

An ambulance was called at 10.03 am and on their arrival Patrick was pulseless and not breathing. He was reported to be peripherally cyanosed with warm skin. An ECG showed asystole and death was pronounced at 10.40am on 13 February 1991. The death was not reported to the Coroner at the time. A Death Certificate was issued.

Permission was given for a hospital post mortem examination which occurred less than two hours after the death. The major autopsy abnormalities were found within the brain, with old infarcts ("strokes") and gliosis (repair tissue in the brain), and old laminar necrosis (death of cortical brain tissue) most severe in the parietal (side of head) and occipital (back of head) areas. There was atrophy ("shrinkage") of neurones in some areas with a slight lymphocytic (chronic inflammatory cell) infiltrate in the leptomeninges (the thin membrane surrounding the brain). There were no features of *toxoplasmosis* or *cytomegalovirus* infection with the distribution of the lesions unusual for *herpes simplex* encephalitis (a viral infection of the brain often affecting the temporal lobes).

The appearances of the brain overall suggested that the changes were as a result of an episode of cardiorespiratory arrest that Patrick suffered at about five months of age. Numerous other tests were performed which have been summarised on page 13 of Professor Cordner's report. No abnormalities were found which would implicate any underlying metabolic or infectious diseases in the brain.

It is clear that Patrick's health deteriorated acutely on 18 October 1990, having previously been generally well. Although he underwent a number of diagnostic tests, no abnormalities were ever detected.

Patrick later died suddenly, after being found by his mother to be pale and not breathing, whilst in his cot. He underwent a hospital autopsy very soon after his death with the cause of death given as

1. (a) *Asphyxia Due to Airway Obstruction due to*

2. (b) *Epileptic fits due to*

3. (c) *Encephalopathic Disorder (underlying cause not determined on investigation).*

The underlying cause for Patrick's ALTE was never known. Following this, he developed seizure activity with many seizures occurring over the space of several months. Detailed brain examination failed to detect an underlying cause such as a viral illness but there were extensive old changes following the ALTE in October 1990.

There are many underlying causes for an "ALTE", however many ALTE's are never satisfactorily explained, as in this case.

Professor Cordner's opinion:

Professor Cordner states (p82) "*The point to be made here is that Patrick's death has been considered by all participants in the trial, it seems to me, as an event quite independent of the ALTE. However once he had suffered the ALTE, is perfectly understandable as being from the delayed effects of the ALTE. This, it seems to me, is a non-controversial, ordinary thought.*"

My opinion:

But for the ALTE, it appears Patrick was a normal baby and progressing quite well. When he was examined soon after the ALTE, he exhibited signs of respiratory difficulties. This is a most unusual finding following a seizure, when the *post-ictal* period usually results in intense drowsiness and a period of sleep. If the airway is not compromised in some way, there should be no evidence after a seizure of respiratory difficulty.

I would not give Patrick's cause of death as epilepsy. Death from epilepsy at this young age is very rare. No person saw Patrick have a seizure at the time of, or just prior to being found. He had quite a number of witnessed seizures during his lifetime but I am not aware any necessitated resuscitation afterwards.

The pathological findings in the brain are subsequent to the cardiac arrest as a result of the ALTE but the underlying cause of the ALTE is unknown. To opine epilepsy as the cause of death appears to be one largely based on the history of seizures and is highly speculative.

At the time of 1989-90, the death of a second child from natural causes following the sudden death of a first child in Australia would be considered most unusual. The situation then of two deceased children in one family where an autopsy yields no satisfying or convincing cause of death in either case is of concern. In the case of Patrick, many tests were performed both *ante mortem* and *post mortem*, with no satisfying diagnosis ever reached.

Although I cannot definitely prove smothering has occurred, I am concerned that smothering could explain Patrick's ALTE and his sudden death. In my opinion, that is a not

unreasonable view to express. A segment from the American Academy of Pediatrics article (13) is worthy of quotation:

"*It is impossible to distinguish at autopsy between SIDS and accidental or deliberate asphyxiation with a soft object. However, certain circumstances should indicate the possibility of intentional suffocation, including:*

1. *Previous recurrent cyanosis, apnoea or ALTE while in the care of the same person;*
2. *Age at death older than 6 months*
3. *Previous unexpected or unexplained deaths of 1 or more siblings;*
4. *Simultaneous or near simultaneous death of twins;*
5. *Previous death of infants under the care of the same unrelated person;*
6. *Discovery of blood on the infant's nose or mouth in association with ALTEs".*

Although since revised in *Pediatrics* Vol. 118 No. 1 July 1, 2006, this paragraph is not substantially different from 2003 except it includes "evidence of previous pulmonary haemorrhage (such as marked siderophages in the lung).

SARAH FOLBIGG:

Sarah was born on 14 October 1992 after spontaneous onset of labour at 39 weeks' gestation. The pregnancy was complicated by early vaginal bleeding on 21 February but this subsided spontaneously. She was breast fed and subsequent neonatal examination was normal apart from mild plethora ("redness"). Training in CPR was given to both parents and Sarah went home with an apnoea alarm on 19 October. She was then bottle fed and slept in a cot by her parents' bed. She apparently used to snore when she was asleep.

She slept with an apnoea blanket under the mattress, however, both parents apparently found the apnoea mattress quite difficult to cope with. At 16 days of age she was well above her birth weight and her general and neurological examinations were normal. A sleep study on 5 November showed very few sleep apnoeas and some periodic breathing. A paediatrician suggested theophylline may have been of assistance in view of some quite long episodes of hypoventilation. A urine metabolic screen showed dicarboxylicaciduria without significant ketosis. Further investigations were indicated if the child was not on MCT containing feeds.

Her progress over the next few weeks was apparently satisfactory and her growth and development were also normal. A test for "MCAD" deficiency (Medium Chain Acyl-CoA Dehydrogenase Deficiency, a metabolic abnormality) was said to be normal. A further sleep study and urine metabolic screen were arranged. She was seen 5 times by a GP and was given the usual childhood vaccinations and treatment for a virus infection and a fungal skin rash.

She was prescribed Flucloxacillin for a cold-like illness on 18 August but this was discontinued on 26 August because of difficulty in administration and on that day she was seen for a "croupy cough". On 29 August Sarah ate normally and was put into bed in a single bed in her parents' bedroom at 9.00pm. The apnoea monitor had been discontinued for about a week. At 9.30-10.00pm she was observed to be snoring and her mother apparently heard her turn over in her

sleep at midnight or 12.30am. The mother got up to go to the toilet at 1.30am but apparently could not hear Sarah breathing. She turned on the bedroom light and saw Sarah was blue and there was a discharge from her nose. She roused her father who commenced CPR and called an ambulance.

When they arrived, the father was giving CPR to Sarah on the floor. She was fully clothed, was blue around the mouth and had mucus and vomit in her mouth but she was not breathing. Full resuscitation was unsuccessful and there was no electrical activity in her heart. The ambulance crew at the time had indicated to the parents that she was deceased. The ambulance report indicated her temperature as both normal and cold. The body was conveyed to Maitland Hospital where life was pronounced extinct at 4.30am.

An autopsy was consequently conducted by Professor John Hilton in Sydney on 30 August 1993. The child appeared well-nourished and there was apparent minor abrading and drying of the lips. A 1.5cm scratch was detected on the anterolateral aspect of the right upper arm. The frenulum of the lips were normal. There were also 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.

Subsequent internal examination showed an essentially normal brain with clear cerebrospinal fluid. The uvula was of normal size but appeared congested on its anterior surface. The epiglottis was normal. Stomach contents were in the trachea and major bronchi. The lungs showed focal areas of collapse with a "geographic pattern". There were occasional petechial haemorrhages and minor congestion and minimal oedema.

The heart was normal. There were occasional petechial haemorrhages on the surface of the thymus and within the substance of the thymus but it was normal in size, shape and location. There was a moderate amount of curdled milk in the stomach. The liver, pancreas, spleen, bone marrow, adrenal glands, kidneys, bladder and genital organs were normal.

Subsequent microscopic examination showed apparent "marked vascular congestion of the pharyngeal aspect of the uvula". This is a most unusual autopsy finding. There was a light lymphocytic inflammatory infiltrate deep to the respiratory epithelium in the larynx (a normal finding given her age). There were no viral inclusions in the salivary gland. A section of diaphragm showed two foci of individual muscle degeneration. The lungs showed congestion and oedema and in one section there was a light interstitial acute inflammatory infiltrate around occasional bronchioles. A further section of lung showed multiple neutrophils within the lymphoid deposits and again, some interstitial infiltration (age related findings). Examination of the brain was essentially normal. Additional post mortem studies for bacteria and viruses were normal. Vitreous biochemistry was normal. Comprehensive screening tests for drugs and other common poisons were negative with no alcohol detected.

The cause of death was given as

1. (a) Sudden Infant Death Syndrome (SIDS).

Professor Cordner agrees with this diagnosis.

My opinion:

I would not give the cause of death of Sarah Folbigg as SIDS. The previous two deaths alone would make me extremely cautious about this death being due to "SIDS'. I would give the cause of death as "Undetermined".

As well, the apparent abnormalities of the uvula are non-specific findings but of some concern. Unfortunately, there is no photographic record of this. I am not aware of any medical literature which describes elongation of the uvula with fatal consequences to the airway.

Although I question the apparent "minor abrading and drying of the lips", I accept that no firm conclusions are able to be drawn from these findings. They may be CPR related and without a photograph, I cannot comment further.

LAURA ELIZABETH FOLBIGG:

Laura was not an unwell child generally but had suffered from a number of childhood illnesses during her life. She was seen by her general practitioner on several occasions over seven months. She underwent a sleep study on 2/10/97 and this showed mild improvement from an earlier sleep study which demonstrated "mild central apnoea and no obstructive apnoea." She also previously underwent full biochemical, blood and metabolic investigations, all of which were normal. There was no evidence of any metabolic abnormality. Following the sleep study on 19/8/97, she was discharged with a home monitoring device designed to record and download breathing and heart information. Both parents were instructed on CPR technique and how to operate the monitor. Laura was monitored on the machine for approximately twelve months without complications. A subsequent sleep study on 2/10/97 showed mild improvement from the first one. The mild central apnoea had improved and was later totally normal.

On the day of her death, Laura was woken at about 6.30am and was seen to have a "runny nose" and "congestion of her chest". She was fed as normal and her father and she then watched television for a period of time. The mother got out of bed at 7.00am and the father began to get ready for work. Laura was in a bad mood but did not appear to be seriously ill during the morning. Laura apparently became upset and the mother and father then had an argument with the mother becoming very agitated. The father drove to work but at 8.15am he received a telephone call from the mother when she apologised and the father apparently "agreed to try harder". Laura was taken to the gymnasium and at around 11am went to visit her father at his place of work. Laura played in his office for about half an hour. The father told the mother that he would be home for lunch as usual.

At around lunchtime he received a phone call to go to the hospital because there was "something wrong with Laura". The mother was sitting in a waiting room and Laura was lying on her back in an adjoining room being attended to by hospital staff. The mother indicated that Laura fell asleep on the way home and she was placed into bed. The mother then went to

feed the dog and had a play with the dog. She heard Laura coughing on the monitor but did not check on her immediately. About ten minutes later she checked on her and found her "lying on her back and pale". She carried her to the breakfast bar in the kitchen where she rang for an ambulance and commenced CPR.

An ambulance attended at 12.14pm. Ambulance officers found Laura's mother giving CPR on the breakfast bar. She indicated that Laura had been heard coughing and when checked on approximately ten minutes later was found to be not breathing. Laura was warm to the touch, was blue in colour, but there was no blood, vomit or foreign object in the child's mouth. An ECG monitor found asystole or a bradycardia (very slow heart rate). Resuscitation was continued and Laura was transferred to hospital at 12.35pm where she was found to be cyanosed with no heartbeat, respiration but with fixed dilated pupils. Death was declared at 12.45pm. No bruise, mark or other abnormality in physical appearance was found on examination at the time. Police then attended the scene. Their examination showed some small dark stains on the pillow of Laura's bed with a screening test positive for blood.

The finding of blood on a pillow is of concern, although I accept there are a range of possible explanations for how any blood was deposited on such a pillow. It is unknown as to whose blood this belongs to, when it was deposited, and under what circumstances. I did not know of this at the time of the autopsy, but it is an issue that remains of concern. Pillows may have areas of staining from nasal mucus, or "drool" from the mouth, but it is most uncommon in my experience for police to discover either frank blood or what appears to be blood on a pillow or elsewhere on bed linen after an infant or young child is found apparently lifeless or deceased.

I performed an autopsy examination at 9.00pm on 1 March 1999. This post mortem was performed in the presence of Professor John Hilton, Director of the then NSW Institute of Forensic Medicine and a Crime Scene Officer from NSW Police, Clint Nicol. At the time of the autopsy, I knew very little in relation to the details surrounding the earlier Folbigg children deaths, but was aware there were now no living children following the death of Laura.

The autopsy revealed a number of small old bruises on both legs but no other bruises or injuries were detected. A facial dissection failed to reveal any injuries around the mouth or about the face.

Internal examination showed no injuries although an 8mm diameter of haemorrhage was on the posterior surface of the left atrium of the heart, which was presumed to be from CPR but may not be. The airways were normal with the finding of focal haemorrhage and collapse on cut section of both lungs. These are quite non-specific changes. There were petechial haemorrhages on the anterior aspect of the thymus above the level of the sternum. This is likewise is a non-specific finding and no conclusion can be drawn from this however it is unusual.

There were prominent lymph nodes in the mesentery of the abdomen, which is a common finding in deceased children following viral infection at an earlier time. The heart was of normal weight and showed no anatomical abnormalities. Cut section of the left ventricle was normal. There was, however, upon microscopic examination of the heart, a patchy and light but

occasionally denser infiltrate of lymphocytes in the heart, with degeneration of muscle cells. The changes were indicative of myocarditis, presumed to be related to a viral illness.

It is noteworthy to point out that Laura apparently had a cold or 'flu type illness within days prior to her death and the presence of myocarditis may reflect a generalised viral infection. Professor Cordner believes myocarditis to be the cause of death however I disagree with this opinion. The finding of "an abnormality" at autopsy is not automatically "upgraded" to be **the** cause of death unless it satisfies other criteria such as according with the clinical history, or after laboratory tests are performed. Part of Byard's article (11) is worthy of quoting:

"*...care should be exercised in proposing a cause of death even when obvious significant disease is present....While death is often confidently ascribed to lesions found during post mortem assessment, the nature of autopsy material makes it difficult to precisely correlate histological lesions with functional impairment. For example, it is often impossible to determine why apparently identical lesions result in death in one individual and yet cause minimal disturbance in another. This reflects the complex and idiosyncratic nature of pathophysiological processes and the limitations of autopsy pathology. It also underlines the importance of obtaining good clinical and death scene information so that the final decision on the cause of death can be guided by clinicopathological correlation rather than based on pathological findings in isolation."*

When opining a cause of death, especially in a young child, the circumstances surrounding the death must be considered in detail, as well as the autopsy findings. In this case I was aware of a recent cold or 'flu type illness around the time of death but I had no knowledge or information that indicated Laura had been unwell with this or showed any symptoms of an underlying more serious illness prior to her death. Although on occasions, persons with myocarditis can die suddenly and unexpectedly, in my opinion I did not consider this to be likely as Laura had not been unwell on the day of her death and the amount of inflammation in the heart was quite light and patchy. I have seen fatal cases of myocarditis in this age group and in my view this case was not typical of sudden death from myocarditis.

From a clinical perspective, children with myocarditis have a range of symptoms from asymptomatic or non-specific generalised illness to cardiogenic shock and sudden death (25). "*Infants and young children more often have a fulminant presentation with fever, respiratory distress, tachycardia (fast heart rate), hypotension (low blood pressure), gallop rhythm and cardiac murmur. Associated findings may include a rash, or evidence of end organ involvement such as hepatitis, or aseptic meningitis".*

The diagnosis clinically can be very difficult to make as most children recover fully and no tissue diagnosis is ever made (it would require a biopsy of the heart which is clearly a medical procedure that is not undertaken for casual reasons).

The autopsy findings have been documented in Professor Cordner's report, pages 22-23 with the cause of death ultimately given by me as 1(a) Undetermined.

Even allowing for the passage of nearly 20 years since the autopsy, I remain of the view that myocarditis does not adequately explain this child's death. Other organs examined at autopsy and which were subsequently microscopically examined also showed features consistent with what I believe is an incidental viral infection around the time of death.

2. Whether there have been any medical advances since 2003 relevant to my area of expertise (Forensic Pathology), with advice whether the views I expressed at trial in 2003 have changed or remain the same and the reasons therefore.

There have been many medical "advances" since 2003 relating to the investigation of sudden death in both infants and adults. The main advances relate to the genetic determination of a number of diseases which affect the cardiac conduction system and which may predispose affected individuals to cardiac rhythm disturbances and the risk of sudden death. Other conditions which can now be genetically tested for include arrhythmogenic right ventricular cardiomyopathy, Brugada syndrome, cathecholaminergic polymorphic ventricular tachycardia, dilated cardiomyopathy, hypertrophic cardiomyopathy, familial thoracic aneurysm and short QT syndrome.

Cardiac conduction disorders are however usually associated with a morphologically normal appearing heart and it is only based on subsequent genetic testing of families affected that these abnormalities are diagnosed. The most common cardiac conduction disorder with an inherited basis is the so called "long QT Syndrome" which refers to an abnormality in an ECG of an affected individual where the "QT" interval is abnormally prolonged. This abnormality relates to sodium and potassium channels within the heart which may affect individuals, by causing the spontaneous onset of cardiac rhythm abnormalities.

At least 13 genes are now implicated in this condition. 75-80% of cases have an identifiable mutation based on current testing. The inheritance pattern for these abnormalities varies. In some cases there is a family history of fainting, fitting or "funny turns" and/or sudden death in the maternal or paternal lineage, or other "red flags" such as exercise intolerance, drowning, unexplained single motor vehicle accidents, enlarged heart, "SIDS" deafness and syndactyly. In some families, there is no history and the diagnosis is only made after genetic testing of the affected individual.

In former times, an ECG tracing of the heart would have been performed if the condition was suspected, however now that these genes can be looked for in accredited laboratories, the diagnosis has been refined and made more accurate.

I understand that Patrick, Sarah and Laura Folbigg had ECG traces at least once each during their lifetimes. I also understand that on each occasion, no abnormality was ever found. That indicates that a significant cardiac rhythm abnormality such as long QT syndrome is unlikely but is worth considering and screening for, now that genetic testing can be performed.

It is at least theoretically possible to extract DNA from the paraffin wax blocks of tissue taken from the autopsy and make very thin ribbons of tissue to test for the presence of these

abnormal genes. All four Folbigg children had autopsies, whether coronial or hospital, and that tissue from each child should still be in existence.

Other medical advances have occurred since 2003 with respect to the diagnosis of sleep apnoea in both adults and children. The more accurate elucidation of infectious diseases particularly rapid PCR (Polymerase Chain Reaction) testing for a variety of bacteria and viruses has also since occurred. Our knowledge in relation to mitochondrial diseases has also vastly increased. These are a bewildering array of diseases, all due to abnormalities in the mitochondria, the "power house" of cells. Fatal cases do not usually occur suddenly and unexpectedly, and there are often clinical and laboratory abnormalities identified soon after birth which are clues to these diseases. For further expert information, I would seek assistance and guidance from accredited specialists in these fields.

There have also been advances in the area of metabolic disease testing with many more diseases identified which are able to be tested for particularly in the newborn screening test. In forensic pathology practice, it has become much more commonplace to also perform a "genetic autopsy" if the pathologist considers there is a possibility that a genetic disease may have contributed to or caused the death of one individual, including children and infants. Although the autopsy *per se* has not changed over the years, the procurement of more tissue and blood for DNA testing would be regarded as "routine" particularly in cases of a sudden infant death where no cause is found.

That is not to say that blood is routinely sent to any DNA laboratory for testing of "all known genetic diseases", however a detailed family history in certain cases would be obtained and selective testing for a number of diseases may be performed, in conjunction with consultation with a genetic counsellor and/or genetic specialist.

Had any of the Folbigg children died suddenly in 2018, it is almost certainly the case that further testing of this nature would have been performed, especially after the death of Patrick. In theory, therefore, DNA testing is theoretically possible as tissue exists, and that tissue is now able to be examined which in former times it was not.

Comments on "deliberate smothering" in infants and young children, and quotes from relevant literature:

"Blockage of the nose and mouth causes death by asphyxia due to inability to breathe. This may be effected in a variety of ways, such as by gagging, holding a pillow over the face of a victim, or shutting the airway with one's hands" (26).

This is an area where much has been published but little is actually known. Witnesses are very few and perpetrators rarely confess. It is believed to be a rare event but we simply do not know the full story in these cases. It remains a difficult area of study for obvious reasons. Nevertheless it has been the subject of published articles (5, 8,10, 13, 14, 16, 19, 21-23) and commentaries in many texts and journals over decades.

Smothering (the occlusion of the outer airways ie the nostrils and mouth) may be the most common method of deliberate asphyxiation in children. As the surface area of the outer airways of an infant is very small, around 5-6 square centimetres maximum, a small object could be used to quite easily cover an infant's outer airway and cause death. Smothering may leave no physical trace. An adult hand or even another child's hand, if large enough, could be used to smother an infant or child. Many household objects such as soft toys, pillows, or bed linen could be employed also.

In some cases, there may be pressure on the chest to further restrict breathing but this may leave physical signs such as petechial haemorrhages on the face (due to impairment of venous return in the head and neck region) and cause rupture of tiny blood vessels in relatively unsupported areas such as the upper or lower eyelids or areas of the face, especially the cheeks or forehead.

Any physical signs left after smothering may be much more difficult to interpret when extensive cardio-pulmonary resuscitation (CPR) has occurred after discovery of the body. CPR is almost universally performed in infants and children but it is known to cause a variety of injuries to the face, mouth, and throat regions. Differentiating true inflicted injuries therefore from CPR induced injuries can be very difficult.

Physical evidence found after fatal smothering has occurred could be anything from nil to "some", depending on the age of the child, the amount of "struggle" that occurred, whether the child has teeth, whether the child is already debilitated and weakened physically, or robust and otherwise healthy, or has been given a sedative drug, or even alcohol. Injuries described include bruises and abrasions around the outer airways, compression marks from teeth on the lips, or blood in the nose and mouth from local trauma (22, 26, 27). By their very nature, these injuries are often small in size, and can be overlooked or explained by other mechanisms, especially as CPR "injuries" when the outer airway is manipulated for airway insertion.

Pathologists are generally unable to indicate the true extent of smothering (whether accidental or deliberate) in cases of sudden infant death as physical signs can be limited, and any physical signs which are present could be interpreted in a number of ways. "*When there are other infants or children in the family who experience such episodes, abuse, metabolic defect, or respiratory malfunction must be excluded. It is, at times, most difficult to distinguish homicidal smothering from SIDS…. Repeated episodes of SIDS within a family warrant further investigation to rule out child abuse and homicide, metabolic disease, or congenital hypoventilation syndrome (Ondine's curse).*" (27)

Professor Cordner is a co-author of the book "*Ethics, Legal Medicine and Forensic Pathology*" (21). In the paragraph on "The Autopsy" in the chapter of sudden death in childhood, (p.204), I quote the following:

"Although the forensic pathologist has to rely initially on the history provided by the coroner, he or she should always attempt to elicit the following details: date and place of death, gestational age at birth, previous health of the infant and the family, feeding and immunisation history, when the child was last seen by a doctor; time the child was last seen alive; circumstances in

which the infant was found, including clothing and evidence of social conditions; details of siblings and whether there had been any previous perinatal or infant deaths in the family….

The state of the infant should be noted, with details such as the quantity of clothing and presence of froth round nose and mouth. ……External features should be noted and recorded including state of nutrition, minor anomalies, and evidence of trauma and treatment: particular attention should be paid to the airways….

Autopsy Findings

(p205) "The forensic pathologist is usually confronted with difficulties in arriving at his or her diagnosis in such deaths. The signs, if any, of suffocation or other physical mistreatment may be absent or only slight. An examination which is biased towards the internal findings may miss small bruises to the inside of the lips or cheeks, or subcutaneous bruises on the chest, arms or back, for example. The importance of negative findings is built into the definition of the sudden infant death syndrome and failure to inspect these areas may render the conclusion invalid. An acceptable cause of death after complete autopsy is only found in about 5 per cent of deaths. The more common of such causes are bronchiolitis, pneumonia, gastroenteritis, meningitis and myocarditis."

Multiple "Cot Deaths" in Siblings

"Extreme care must be taken when performing an autopsy on a presumed second SIDS death in the same family. An extremely careful history, a very thorough external examination of the infant, and a complete set of body X-ray pictures must be taken before commencing the autopsy…… In 1986 Emery reported that a survey of twelve families in Sheffield, England, in which two or more cot deaths had occurred showed that the causes of death were completely unexplained in two families; the babies had a probable familial developmental disorder in three families; the carer of the infants were seriously at fault in two families; and in five families filicide was probable. Three of the mothers in this last group were psychologically ill. Although the possibility of infanticide or child abuse must be considered in all unexpected child deaths, the suspicion and care with the post mortem examination must be greatly heightened when more than one 'cot death' occurs in the same family."

I agree with these remarks.

"Homicidal Pediatric Asphyxia (22)

*In most homicidal pediatric asphyxia deaths, the victims are very young, the child is smothered, and autopsy findings are minimal or absent. The smothering agent may be a pillow, blanket, hand, sheet of plastic wrap, or innumerable other items. The incidence of actual infanticide cases mistakenly designated as SIDS or similar terminology is presumed to be quite low, but estimates vary (*13).

Many cases come to light only after a confession or when multiple children die in the care of the same caregiver. In the investigation of a homicidal asphyxiation of a child, if it is even recognized, three questions often loom:

How long does it take to suffocate an infant?
Are the autopsy findings consistent with the confession?
Do infants and small children "put up a fight"?.....

Is the Autopsy Consistent with the Confession?
*As with many accidental asphyxiations, the autopsy alone may offer few (if any) clues as to the cause of death. Petechiae (*tiny "dot-like" haemorrhages*) are uncommon. Meadow (*19*) reviewed the cases of 27 children (18 living, 9 dead) from 27 different families who had been suffocated. Five of the children had facial petechiae and two had bruises on the neck, while at least 14 had no external findings related to suffocation. In these 27 families, 18 of the 33 siblings born before the index case was identified had died. Thirteen of these 18 had been certified as SIDS or similar terminology.*

*Southall (*16*) reported 39 cases referred to a children's hospital after exhaustive medical evaluations for apparent life-threatening events. Using constantly monitored CVS, the authors documented abuse in 33 of the 39 cases. In 30 of the 39 cases, the abuse was intentional suffocation. Only 4 of the 30 suffocated children had petechiae. Nasal/oral bleeding was seen in only 11 of the abused children (compared to zero instances of nasal/oral bleeding among 46 normal controls). Of 41 siblings born before the index cases, 12 had died suddenly and unexpectedly. After CVS, the parents confessed to having suffocated 8 of the 12. A number of valuable lessons emerge from these studies regarding the care and diagnostic evaluation of living children. For the pathologist charged with the post mortem examination of a dead infant, one lesson is clear: more often than not, even in a child known to have been smothered, the autopsy will not demonstrate specific physical injuries referable to intentional asphyxia.....*

Conclusion:

In accidental asphyxiation of the small child:
Many forms of pediatric asphyxia, particularly infant deaths from wedging, overlaying, and smothering, will, more often than not, have negative autopsies or autopsies with nonspecific findings.
In most cases, the autopsy findings in a suffocation death of an infant (accidental or intentional) will not differ from the findings in "SIDS". Therefore, the death investigation (scene, medical history, witnesses) is of paramount importance.
Death investigators, law enforcement officials, coroners, medical examiners and other physicians reviewing childhood deaths should not assume that a negative autopsy alone is sufficient for a determination of "SIDS".
The likelihood of petechiae is highly dependent on the particular type of asphyxia involved. In general, external petechiae (facial, conjunctival) are more rare in the very young child than the older child or adult.....

In homicidal asphyxiation of the small child:

Smothering of infants is the most common type of homicidal asphyxia in children, but other types do occur.
In most cases, the autopsy findings in the intentional suffocation death of an infant will not differ from the findings in SIDS, overlaying, wedgings, and other non homicidal infant deaths. Significant nasal or oral bleeding may suggest inflicted airway obstruction but would be expected only in a minority of cases. The intentionally smothered infant may show no signs at all to even experienced forensic pathologists or clinicians .
Physiological data and covert video surveillance from MSBP studies may help to answer some of the relevant questions in known fatal infant smotherings."

I agree with these remarks.

"***Gentle" Homicides and the Lethal Variant Munchausen's Syndrome by Proxy*** (20)**.**

Probably the most commonly missed method of homicide in infants and young children is smothering. Based on the authors' experiences, smothering is, after impulse homicides, the second most common type of homicide in infants. In infants, smothering is very easily accomplished. One closes off the child's nose with two fingers, at the same time pushing up on the lower jaw with the palm to occlude the airway. Other methods have involved placing a pillow or towel over the child's face and pressing down; pushing the face down into bed clothing, or just covering the nose and mouth with one's hand. These descriptions are based on either confessions or witnessed homicides. In a few cases, attempted homicides have been videotaped.

The true number of smotherings in infants is not known and can never be known until there is some scientific test to determine whether an individual has been smothered. The amount of force necessary to produce smothering is so minor in this age group that there is virtually never evidence of trauma. Autopsy findings are essentially unremarkable, the same as those found in SIDS deaths. Undoubtedly, a small percentage of SIDS deaths are due to smothering. This has been estimated as high as 10%.....

The amount of time necessary to smother an infant, such that their electroencephalogram (EEG) is flat and there is no spontaneous respiration, has been documented at 70-90 seconds. Deaths caused by smothering tend to be missed in large urban communities with high crime rates, where both the medical examiner's office and police agencies are overworked and cannot afford the time on detailed investigations of all cases.....

While most homicidal suffocation of infants is sporadic, a small number of individuals, virtually all of whom are mothers, practice a lethal form of Munchausen's syndrome by proxy, a form of child abuse in which children are brought to physicians and hospitals for induced signs and symptoms of illnesses in conjunction with a fictitious history. The child is usually subjected to multiple hospital admissions and extensive medical evaluations, treatments and procedures for these non-existent medical conditions"

p 354- ***SIDS and the Munchausen Syndrome by Proxy***

*Over the years, a number of causes have been proposed for SIDS. One suggestion was that the episodes of prolonged apnoea (*cessation of breathing*) seen in premature infants are a "form frustre" of SIDS. This concept was proposed by Steinschneider in 1972.* (16) *He studied five infants, three of whom were referred to him about one month of age because of cyanotic ("blue") episodes of undetermined etiology (cause). Two subsequently died of what was called the sudden infant death syndrome; three survived. One of the two was a 29 day old female seen because of recurrent cyanotic episodes. The first occurred at the 8th to 9th days of life; the second five days later. She was admitted to the hospital at the time and discharged at the age of 25 days without a diagnosis. She was then re-admitted following another severe cyanotic episode. This pattern of admission, discharge and readmission occurred until her death at home at 79 days of age. During her work-up by Steinschneider, the child allegedly suffered multiple episodes of prolonged apnoea.*

The second child, a male, was studied age 5 days to 33 days. The morning following discharge, he had an alleged episode of prolonged apnoea and cyanosis. A similar alleged episode occurred 15 to 20 minutes later. The child was hospitalized for 34 days and then discharged. He was re-admitted the following day for a period of 6 days because of apparent aspiration during feeding. He was discharged and, on the morning following, had an apnoeic episode, became cyanotic and died.

....In addition to these two deaths, mention is made in the article that three other children in the family had also died. The first male developed recurrent cyanotic spells and died suddenly at 102 days of age; the second, a female, turned blue and died aged 48 days of age. Neither of the two children was autopsied. The third cried out and died suddenly at 28 months of age. An autopsy was negative, except for the fact that the adrenal glands were "considered to be of small size....

The Steinschneider article became one of the most quoted articles in the field of SIDS. It led to the introduction of apnoea monitors to "prevent" SIDS. In addition, the article indicated there was hereditary basis for SIDS. Following its publication, a number of articles appeared in the medical literature describing multiple cases of SIDS in families, thus appearing to confirm that SIDS was indeed hereditary.

Forensic pathologists were immediately sceptical about the article, strongly suspecting serial homicide. In the first edition of this book, the authors stated that the deaths in the article were homicide and that Steinschneider's article was describing a lethal variant of Munchausen syndrome by proxy. In March 1994, the mother of the two dead children in Steinschneider's article confessed to smothering all five children because she couldn't stand their crying. She was subsequently tried and convicted of homicide". Firstman and Talan (14) subsequently published the Steinschneider cases in 1997.

The Steinschneider commentary by di Maio has been included "for completeness" as it is important to give some history and context to how the notion of infant "apnoeas" began, which then created an "industry" of SIDS pneumogram monitoring and the notion of being able to

"prevent SIDS". In 1997, Dr Jerold F.Lucey, then Editor of *Pediatrics,* wrote (12) after the 25th anniversary of the publication of the Steinschneider article: "*We should never have published this article. My only excuse is that peer reviewers are not detectives. If you're really crooked, reviewers can be easily duped. They didn't know the details of the study, which were only revealed in the Hoyt trial two decades later. The patients studied were murdered. They were not SIDS patients. This seriously flawed study led to the birth of the SIDS monitoring pneumogram industry…."*

Professor Cordner states on p 90, Chapter XI, para 2: "*If the convictions in this case are to stand, I want to clearly state there is no pathological or medical basis for concluding homicide".* Through the narrow prism of the autopsy, this may be so. We know that an autopsy by itself can only take a matter so far and there are often limits to what an autopsy is able to conclude. If, for example, a child is found deceased in a locked cupboard, or has been deliberately drowned, or asphyxiates from lack of oxygen if a plastic bag is kept forcibly over its head (but the bag is removed prior to autopsy), one may be able to say the same thing- *viz. "there is no pathological or medical basis for concluding homicide."*

A similar conclusion could be reached in cases of fatal torture (28) where victims have been sleep deprived for days and then die, or have been made to stand indefinitely, or have been forced to drink copious amounts of sea water, or have been "water-boarded", or have been buried alive, or placed in a "punishment cell" for days on end, or have died from deliberate exposure. It would be greatly misleading and incorrect to opine that in these cases there was no medical or pathological basis for concluding homicide merely because of the absence of injuries.

"The findings cannot rule out smothering in one or more of the cases (I agree)*, but especially in the case of Laura, not only is there an acceptable natural cause of death easily visible microscopically, it is important that no signs of compression of the face are present."* I do not agree there is an acceptable natural disease. Whilst there no marks to the face, and I am unable to prove smothering, it remains a possible explanation for her sudden death.

Professor Cordner says p 91: "*It is worth pointing out that at no point have any of the pathologists or physicians put the homicide hypothesis as "one or more of the Folbigg children were smothered. It was always a binary proposition: all four or none. However, it seems logical, leaving aside the legal imperatives, that with the combination of the existence of SIDS, the newly characterised arrhythmic diseases, the pathological explanations for Laura's and Patrick's deaths and the knowledge that smothering can occur without leaving signs, one must necessarily agree that it is possible, for example, that one child was smothered and the others died naturally. Of course, as soon as that is agreed, it is not possible to say which one was smothered, and at that point it has to be accepted that they could all well have died from natural causes."* He appears to have accepted the notion however that smothering could have occurred but left no trace. I agree with this proposition.

Comments on references cited by Professor Cordner:

There are 65 references cited by Professor Cordner. Of these, I count 33 (51%) that relate to abnormalities in cardiac rhythm disorders and their genetic identification. There are at most 4 (7%) references to facial injuries in children. Two of these (Matshes EW and Lew EO; Kaplan JA and Fossum RM) relate to resuscitation related injuries in children. Another (Hall K and Zalman B) is on the "*Evaluation and management of apparent life threatening events in children*" and is not obviously trauma related.

There is one quote from "Bernard Knight-Forensic Pathology" in relation to possible smothering in children, which is a paragraph taken from p358. The more notable sentences are included here: "*Deliberate homicide is seen usually in the old, the debilitated and in infants. It is extremely difficult to prove homicide from objective findings. ……In relation to infants, the matter will be further discussed in the chapter on sudden infant death syndrome, but it is essential to appreciate that the smothering of babies, whether intentional or accidental, is both rare and difficult to prove.*"

There is one other reference to neck injuries (Vanezis P, p 44). The other 28 references (42%) relate to sudden infant death issues such as safe sleeping environments.

So, despite 65 references, very few refer to the pathological findings in cases of suspected smothering of infants and young children and the difficulty in making this diagnosis.

For completeness and "balance", I therefore list publications and texts (4-10, 13, 14, 16, 17, 19-23) which are concerned with inflicted trauma including smothering.

Concluding remarks:

As the pathologist charged with the investigation of Laura's death, I could not disregard the long shadow of the other three deceased children and sought to investigate firstly whether they were all linked by one single natural disease (such as a genetic or metabolic abnormality) in some way.

Further medical information which came to light around the same time revealed that Patrick, Sarah and Laura Folbigg all underwent very intensive medical testing and investigation during their short lifetimes to search for some possible disease to explain both the sudden death of Caleb and to answer the concern there might exist some condition or disease which could place these living children at substantial risk for sudden death. No condition or disease was ever found. Now that technology exists to submit physical evidence (in the form of paraffin blocks from autopsies) for DNA testing, I urge that testing occur to exclude or confirm the possibility there is an underlying, "unifying" disease which could explain these sudden deaths.

Despite autopsies, there are no convincing pathological findings which could singly or collectively explain the deaths of these children. I have read Professor Cordner's opinion which expresses words to the effect that there is no evidence from the autopsies to propose

homicide as an explanation for the deaths. I have heard this viewpoint on another similar occasion (R v Matthey, Victoria, 2006) but regard it as being artificially narrow and unrealistic.

I know of no forensic pathologist anywhere who would ever seriously offer an opinion about any case based purely on the autopsy findings, (let alone explain multiple infant deaths in the one family), without placing the autopsy findings in the context of the preceding events and the known circumstances surrounding the death.

A diagnosis of "SIDS" essentially stops any further investigation of an infant death. It may incorrectly confer a diagnosis of sudden death from natural causes when that diagnosis may not be correct. To then make such a diagnosis twice in a family where another intervening child was also known to have died suddenly, having also had a prior ALTE, is both astonishing but very troubling. Further, when a fourth child in the same family dies suddenly, and a pathological finding from the autopsy becomes elevated to be **the** cause of death without further question, I must express my deepest concern.

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. I do not consider this to be an unreasonable opinion, based on the facts of each case, when one considers the exhaustive medical testing performed on Patrick, Sarah and Laura Folbigg, together with a thoughtful consideration of the individual and collective circumstances of each death, and the autopsy findings. It is a view that I have to say is shared by many of my forensic pathology colleagues both in Australia and overseas.

The only other suggestion I wish to make is to request an experienced paediatrician be invited for comment on these cases, perhaps after the results of any DNA testing becomes available.

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10. Forensic Pathology Principles and Practice. Dolinak, Matshes and Lew. Elsevier Academic Press, 2005. p 206
11. Significant coincidental findings at autopsy in accidental childhood death. Byard RW Med. Sci. Law (1997) Vol. 37, No.3.p259-262.
12. Editorial, Lucey J. *Pediatrics* October 1997
13. Distinguishing Sudden Infant Death Syndrome from Child Abuse Fatalities (RE0036). American Academy of Pediatrics et al. Pediatrics. 2006; 118(1):421-7
14. The Death of Innocents. Firstman R., Talan J. Bantam Books, New York 1997
15. Contribution of long Q-T syndrome genes to Sudden Infant Death Syndrome. Berul C., Perry J. Circulation 2007; 115:294-296
16. Covert video recordings of life threatening child abuse; lessons for child protection. Southall DP et al. Pediatrics. 1997; 100:735-760.
17. Estimates of fatal child abuse and neglect, United States, 1979 through 1988. Pediatrics. McClain PW et al. 1993; 91:338-343.
18. Prolonged apnea and the sudden infant death syndrome: clinical and laboratory observations. Steinschneider A. Pediatrics. 1972; 50:646-654.
19. Suffocation, recurrent apnea, sudden infant death. Meadow R. J Pediatr. 1990; 117:351-357.
20. Meadow. Br Med J. 1989; 298(6687):1572-3
21. Ethics, Legal Medicine and Forensic Pathology. Plueckhahn and Cordner, Melbourne University Press 1991 2nd Edition p204
22. Forensic Pathology of Infancy and Childhood", Kim Collins and Roger W.Byard (Editors), Springer Reference, 2014 Volume 1, Chapter 9, (Dr Andrew Baker)
23. Forensic Pathology, 2nd Edition, Vincent and Dominick Di Maio, CRC Press, 2001, p349
24. Clinical implications of sudden infant death syndrome epidemiology. Peterson DR. *Pediatrician.* 1988; 15:198-203.
25. Nelson Textbook of Pediatrics. 19th Ed. Elsevier Press. p1635.
26. Spitz and Fisher's Medicolegal Investigation of Death. 3rd Edition. p.479.
27. *ibid.* p.725-6.
28. The Gulag Archipelago. Chapter -"*Interrogation*". Alexander Solzhenitsyn. 1974. Collins Publishers. pp 93-144.

Dr Allan D.Cala
FRCPA

ALLAN DAVID CALA

CURRICULUM VITAE

FULL NAME: Allan David CALA

DATE AND PLACE OF BIRTH: 4 March 1959, Sydney, NSW.

CITIZENSHIP: Australian

PROFESSIONAL ADDRESS:

Newcastle Department of Forensic Medicine,
Forensic Analytical and Scientific Services (FAASS)
John Hunter Hospital,
New Lambton Heights,
Newcastle,
N.S.W. 2305
AUSTRALIA

Phone: 02-4935-9700
Fax: 02-4922-3735
email: Allan.Cala@health.nsw.gov.au

SCHOOL ATTENDED:

Homebush Boys' High School, Sydney

UNIVERSITY ATTENDED:

University of New South Wales (1977-81)
St. Vincent's Hospital Clinical School, Darlinghurst (1978-80)

DEGREES AND FELLOWSHIPS:

1. Bachelor of Medicine, Bachelor of Surgery (M.B., B.S.) University of NSW, awarded January 1982.

2. Diploma of Royal Australian College of Obstetricians and Gynaecologists (Dip.R.A.C.O.G.)-1985. Examination and *Viva*.

3. Fellow of the Royal College of Pathologists of Australasia (F.R.C.P.A.)-1994, with Part II Examination slanted to Forensic Pathology. Fellowship awarded in Adelaide on 2 October 1994.

OTHER RELATED ORGANISATIONAL MEMBERSHIPS:

1. Australian Medical Association (AMA)
2. British Association of Forensic Medicine (BAFM)
3. Australian Military Medicine Association (AMMA)
4. International Academy of Pathology (Australasian Division)
5. International Society of Forensic Radiology and Imaging (ISFRI)
6. Australian & New Zealand History of Medicine Society
7. NSW Forensic Medicine & Science Group, University of Sydney.
8. Australian Defence Force Forensic Group

MEDICAL REGISTRATION:

1. MEDICAL BOARD OF AUSTRALIA (AHPRA) MEMBERSHIP No. MED0001064509 (GENERAL AND SPECIALIST MEDICAL REGISTRATION)-REGISTERED UNTIL 30.09.18

2. GENERAL MEDICAL COUNCIL (U.K) -1995 OVERSEAS LIST

CAREER SUMMARY:

2008-Current
Senior Staff Specialist Forensic Pathologist
Newcastle Department of Forensic Medicine,
NSW Forensic Analytical and Scientific Services

2003-7
Forensic Pathologist, Forensic Science South Australia

1996-2002
Forensic Pathologist, (then) NSW Institute of Forensic Medicine, Sydney

1995
Forensic Pathology Registrar, NSW Institute of Forensic Medicine (January-April)
Universities of Glasgow and Edinburgh, Scotland, UK. (April-December)
Registered with General Medical Council, Great Portland St, London.

1994
Forensic Pathology Registrar, NSW Institute of Forensic Medicine, Sydney.

1993
Anatomical Pathology Registrar,
St Vincent's Hospital, Sydney.

1989 - 1992
Anatomical Pathology Registrar,
Canberra Hospital, A.C.T.

1988
Haematology Registrar (Laboratory)
Canberra Hospital, A.C.T. (Previously known as Woden Valley Hospital)

July –December 1987
Registrar, Accident and Emergency Department,
Royal Canberra Hospital, A.C.T.

1986 – July 1987
Medical Officer
Royal Flying Doctor Service,
Port Augusta, South Australia.

1985
Resident Medical Officer
St Margaret's Hospital, Sydney.
Obstetrics & Gynaecology (6 months) and Neonatal ICU (6 months).

1982 - 1984
Intern and Resident Medical Officer,
Concord Repatriation Hospital, Sydney.

PROFESSIONAL DUTIES AND RESPONSIBILITIES:

1. Perform legally authorised post mortem (autopsy) examinations on deceased persons whose deaths have been reported to the Coroner; and prepare timely, relevant and accurate reports.

2. Testify competently as an expert witness at inquests, committal hearings, criminal trials and any other relevant courts where expert testimony is required.

3. Supervise and assist in training of registrars in Forensic and Anatomical Pathology.

4. Conduct lectures, tutorials and other learning methods in forensic pathology for medical students, registrars and other groups.

5. Provide an unbiased, objective and professional service to external agencies.

6. Be actively involved in on-going learning, training, research and education.

7. Participate in internal and external quality assurance programmes, RCPA-directed Continuous Professional Development Programmes.

PUBLICATIONS:

1. Spinal cord injury after forceps rotation: the role of glioneuronal heterotopias. R.Pamphlett, A.Cala -Aust N Z J Obstet Gynaecol, 33 (1):91-3 1993 Feb

2. An unusual foreign body in the small intestine. A.Cala, E.Sugo. - American Journal of Forensic Medicine and Pathology, 21 (1):53-5 March 2000

3. Murder not most foul. C.H.Lawrence, A.D.Cala, MJA Christmas Edition, 2001.

4. Written and oral submission (with Dr Ella Sugo) to the NSW Legislative Council Inquiry into mental health services in New South Wales. (Final Report December 2002) **www.parliament.nsw.gov.au**

5. Letter, RCPath Bulletin, submitted 30 July 2008-*Histology in Coroner's cases.*

6. Unusual presentation of pulmonary tumour thrombotic microangiopathy with no detectable primary tumour. Seppala N, Cala A, Klebe S. JPostgrad Med Jan 2009 Vol 55 Issue1

7. Letter to MJA. Accidental asphyxiation and bicycle helmets. Byard R et al. Jan 2011 Vol Issue 1

8. Elevated *post mortem* vitreous sodium and chloride levels distinguish salt water drowning (SWD) from immersion deaths not related to drowning but recovered from saltwater (DNRD). A Cala, R.Tse, R.Vilain. Am J Forensic Med Pathol. 2013 Jun;34 (2):133-8.

9. Takotsubo cardiomyopathy: Not always innocent or predictable a unique post mortem insight. Elsokkari, A Cala, Khan and Hill. (Letter to the Editor), Int J Cardiol 167 (2013) e46-e48

10. Use of radiology in Disaster Victim Identification. Position statement of members of the DVI working group of the International Society of Forensic Radiology and Imaging; Journal of Radiology and Forensic Imaging May 2013 and 2014. Principle author Guy N.Rutty.

11. A fatal case of isolated methiopropamine toxicity: a case report. R. Tse, A. Cala –accepted for publication in *Am J For Med Path,* 31 March 2015

12. A decline in 2 consecutive post-mortem serum tryptase levels in an anaphylactic death. S.Anne, R.Tse and A. Cala. *Am J For Med Path*; accepted 7 June 2015

13. Immersion of bovine eyeballs after one hour in sea water does not result in elevation of post mortem vitreous sodium and chloride levels. S.Anne, R.Tse, C.Oldmeadow, J.Attia, AD Cala *Am J Forensic Pathol.* April 2016

14. Elevation of post mortem vitreous humor sodium and chloride levels can be used as a reliable test in cases of suspected salt water drowning when immersion times are less than one hour. J.Garland, R.Tse, C.Oldmeadow, J.Attia, S.Anne, A.Cala. *For Sci Int* (In Press, accepted for publication June 2016)

15. *Neisseria meningitidis* isolated in *post mortem* vitreous humor in a death due to meningococcal sepsis. J.Garland, R.Tse, A.Cala. *Am J Forensic Pathol* 2016-in print.

16. Post mortem tryptase cut-off level for anaphylactic death. Rexson Tse et al, *For Sci Int* 2017, accepted November 2017.

17. Elevated post mortem vitreous sodium chloride (PMVSC) level in a salt water drowning death during self-contained underwater breathing apparatus (SCUBA) diving with diving mask in place-case report. Am J Forensic Pathol 2018

CONFERENCE PAPERS PRESENTED:

1. SIDS Annual Conference, Melbourne, 1997. "SIDS and overlaying-was Solomon right?"

2. RCPA Meeting, Melbourne 1998 "Sudden death at the Star City Casino, Sydney"

3. RCPA Meeting, Melbourne, 1998 "An unusual overdose...or was it?"

4. RCPA Meeting, 11 November 2000, Canberra "Military Forensic Pathology"

5. RCPA Update, Sydney, March 2007 "Restraint asphyxia"

6. IAP Meeting, Sydney 31 May 2008 "Military Dermatopathology"

7. All India Institute of Medical Science (AIIMS), New Delhi, 26 November 2009 "Australian Forensic Pathology"

8. East-West Medico-legal Conference, Istanbul Turkey-14 October 2012 "Crime Scenes-the facts from the fiction".

9. Pan-European Medicolegal Conference, Paris 2013-"Excited delirium" and "The Gonzales Family Murders, July 2001"

10. North American Pacific Legal Conference October 2015 "Uses of CT scans in the Coronial Setting"

POSTERS:

1994:

1. A.Cala "**WHEN DOES POST MORTEM LIVIDITY BECOME FIXED?"**-RCPA Meeting, Adelaide, 1994

2. A. Cala "**INACCURACIES IN BLOOD LOSS ESTIMATION AT DEATH SCENES"**-RCPA Meeting, Adelaide, 1994

2002:

3. A.Cala **"PLASTIC BAG ASPHYXIA"**-ANZFSS Meeting, Canberra, May 2002.

2006:

4. A.Cala **"FATAL NEONATAL ENTEROVIRAL MYOCARDITIS"**-ANZFSS Meeting, Perth 2006

5. A.Cala, DA Eitzen **"SCRUB PYTHON ATTACK"**- ANZFSS Meeting, Perth 2006.

2011:

6. A. Cala, K.Lee **"MASSIVE GASTRIC DILATATION AND DEATH"** –RCPA Meeting, Melbourne March 2011.

7. A.Cala, C.Duthoit **"GALLSTONES AND DNA IDENTIFICATION-A CASE REPORT"**-RCPA Meeting, Melbourne March 2011.

8. A.Cala **"ALL THAT VOMITS IS NOT GASTROENTERITIS**"-RCPA Meeting, Melbourne, March 2011.

9. A.Cala **"VITREOUS SODIUM AND CHLORIDE LEVELS-A TEST FOR SALT WATER DROWNING?"**-RCPA Meeting, Melbourne March 2011. Awarded Best Forensic Poster, Roche Diagnostics

2012:

10. A.Cala **"BLOOD CARBOXYHAEMOGLOBIN SATURATION LEVELS IN A VARIETY OF FORENSIC SETTINGS".** RCPA Meeting, March 2012

11. A.Cala **"UNDIAGNOSED ADULT HIRSCHPRUNG'S DISEASE".** RCPA Meeting, March 2012

2014:

12. R.Gahan, A.Cala "**"AN UNUSUAL CASE OF SUBPHRENIC ABSCESS",** RCPA Meeting, March 2014

13. R.Tse, A.Cala **"ALCOHOL INTOXICATION AND SUICIDAL HANGING DEATHS-A 5 YEAR REVIEW",** RCPA meeting, March 2014 (Best Poster Award)

2016:

14. S.Anne, J.Attia, C.Oldmeadow, A.Cala. "**No elevation in vitreous sodium and chloride levels in bovine eyeballs immersed in sea water for 1 hour."**

DISASTER VICTIM IDENTIFICATION (DVI) AUTOPSIES:

1. 1997-Thredbo landslide (19 deceased)
2. 2001-Albury level crossing, (5 male deceased)
3. 2002-October 12, Bali Bombing, (Darwin locum)
4. 2004- Asian Tsunami
5. 2006-Gladstone (South Australia) explosion (3 male deceased)
6. 2007-Rail disaster, Kerang, Victoria (8 deceased)-observer.
7. 2008-Bathurst plane crash, NSW, (4 deceased).
8. 2009- 7 February Victorian Bushfires (VIFM).

CONSULTANT ACTIVITIES:

I have assisted local, interstate police, and other agencies in the investigation of recent, older criminal and non-criminal cases:

1. Murder of Johanne Hatty, Sydney, 1983-(Trial R-v-Fleming, April 2007)
2. Presumed murder of Ross Warren, Sydney, 1989 (Inquest, Sydney, 2 April 2003).
3. Presumed murder of John Russell, Sydney 1989 (Inquest, Sydney, 2 April 2003).
4. Presumed murder of Revel Balmain, Sydney 1994 (Inquest, Sydney, January 1999).
5. Murders of the Gilham family, Sydney, 1993.
6. Accidental death of Nathan Chaina, Kangaroo Valley, NSW, 1997. (Inquest, Westmead, 20 April 2000).
7. Police –v- Starr, (Self inflicted wounding), testified 16 June 2000, Industrial

Relations Court, 50 Phillip St, Sydney (Walton J.)

8. Deaths of Jacob, Chloe, Joshua and Shania Matthey, Geelong, 1998-2003. Testified Melbourne 17.03.06.

9. Death of Cody Gannan, Sydney 2001 (Trial Sydney 2004).

10. Death of Paul Anthony Keating, Sydney, 2002.

11. Murder of Janelle Patton, Norfolk Island, March 2002. (R-v-McNeill, testified 13-15 February 2007, Norfolk Island.)

12. Murder of Lyndsay van Blancken, Sydney 2003 (Strikeforce "Amato").

13. Death of Justin Larking, Sydney, 2003. (Trial R-v-Clare, June 2006, Sydney).

14. Death of Prithivan Simplee, Villawood Detention Centre, Sydney, Inquest Westmead, 13 March 2003.

15. Death of Anoke Vaillala, trial (R-v- Mohena), Sydney March 2005.

16. Opinion for SA DPP, Adelaide, R-v-Nguyen, Victim Mai (Grievous Bodily Harm)-Trial aborted, Adelaide, 26 June 2006.

17. Opinion for SA DPP, Adelaide , R-v-Dwiar, September 2007

18. Opinion for NSW DPP, R-v-Farrer, testified 24 December 2008, Armidale, NSW. (Malicious wounding)

19. Opinion for NSW DPP, R-v-Wells, Assault, 2009.

20. Opinion and testimony for NSW Crown Solicitor, in relation to death of Christopher Howlett (Inquest Lismore), 14 August 2009, 9 August 2010 and Glebe, July 2011.

21. Opinion for NSW Police, R-v-Peat (wounding) 23 September 2009

22. Opinion for NSW Coroner, death of Alex Wildman, 12 October 2009

23. Death of Mr John Frederick Baker, Thailand, December 2010, review for Christine Warren (daughter).

24. Deaths of James Dalton-Brown and Eliza Wannan, Inquest Orange May 2011

25. Death of Krystal Turk (NSW State Coroner, 2011).

26. Death of Alistair Harris (Parkes, December 2011)

27. Death of John May (Parkes, December 2011)

28. Death of Eric Melhuish (Parkes, December 2011)

29. Death of Wayne Wakefield (Parkes, December 2011)

30. Death of Isabella Manners (NSW State Coroner, April 2012)

31. Death of Leanna Nixon (Parkes Coroner, April 2012)

32. Death of Bailey Constable (Warren, 2011)

33. Death of Luke Ogilvie (Lismore, 2010)

34. Death of Scott Johnson, December 1988 (Sydney 2014 and 2016)

35. Wounding of Ben Rhodes (NSW Police) 2016.

CONFERENCE AND COURSE ATTENDANCE (Excluding conferences where papers were presented):

1. Annual RCPA Update Meeting. (1994-2017).

2. Neuropathology Course, University of Sydney, January 1994 and 2009

3. Summer meeting, British Association in Forensic Medicine, Edinburgh, July 1995.

4. Annual Australasian International Academy of Pathology, June, Sydney. (1994-2010)

5. Orlando, Florida, November 1997, "Pediatric Forensic Issues:Pathology, diagnosis, imaging and investigation."

6. San Diego, November 1998, "Pediatric Forensic Issues:Pathology, diagnosis, imaging and investigation."

7. Dade County Medical Examiner's Office, Miami, Florida: "Police/Medical Investigation of Death" Course, December 2000

8. Forensic Symposium, Perth, WA, 1-2 June 2001.

9. British Association in Forensic Medicine Winter Meeting, Belfast November 2001.

10. British Association of Human Identification Course (BAHID), St George's Hospital, London, 15-17 November 2002

11. American Academy of Forensic Sciences Meetings, February (Dallas 2004, Seattle 2006, Seattle 2010, Seattle 2018).

12. Australasian Coroner's Meeting, Perth WA, 15-19 November 2005

13. Basic "Virtopsy" Course, Bern, Switzerland, 4-6 September 2007

14. Australasian Coroner's Society Meeting, Adelaide SA, 18-21November 2008

15. Faculty of Forensic and Legal Medicine, Royal College of Physicians, London 8-9 May 2009

16. Australia-Pacific Coroner's Society Meeting, Canberra ACT, 11-13 November 2009

17. Mayo Surgical Pathology Course, Lisbon, 2-6 May 2011.

18. Pan-European Medicolegal Conference, 3-7 January 2012, 2014, 2015 Cortina, Italy

19. Australian Association of Forensic Physicians meeting, Brisbane, 5 May 2012

20. East-west Medicolegal Conference, Istanbul, October 2012

21. Pan-European Medicolegal Conference, Paris, April 2013

22. International Society of Forensic Radiology and Imaging (ISFRI), Zurich, May 2013 and Leicester UK May 2015

23. South Pacific Underwater Medicine Society Meeting, Fiji, May 2016

24. Australia-Pacific Coroner's Society Meeting, Perth WA, November 2016

25. International Association of Forensic Science, Toronto Canada August 2017

26. Human Identification Course, Charterhouse Square, London UK, March 2018

TEACHING/EXAMINING ACTIVITIES:

1. Examiner in Forensic Pathology, RCPA and in Pathology through RANZCR (College of Radiologists).

2. Postgraduate forensic pathology supervisor and examiner.

3. Lectures to University of Newcastle medical students/interns/RMO's/police.

4. Lecturer and workshop supervisor at "Short Course in Forensic Pathology", Royal Hobart Hospital, 2003-5 inclusive.

5. Medical student (clinical medicine and pathology) examiner, University of Adelaide 2003-5.

CONTINUING PROFESSIONAL DEVELOPMENT:

1. Daily case reviews with peers.
2. Annual RCPA participation-Continuous Professional Development Programme.
3. RCPA Quality Assurance testing programme.
4. College of American Pathologists QAP.
5. Regular (online) journal perusal-eg Lancet, British Medical Journal, Medical Journal of Australia (eMJA).
6. Attend other pathology/medical departments for teaching/training.

MILITARY SERVICE:

1999-2002

Australian Army Medical Corps; Captain.

2002-18

Royal Australian Air Force -Special Reserve, Forensic Group; Squadron Leader.

MILITARY COURSES/OPERATIONS ATTENDED:

1. Specialist Service Officer (GRES) Course (Basic) 27 November-12 December 1999, Duntroon, A.C.T.
2. Chemical, Biological and Radiological Course, Fire Brigade Headquarters, 1-3 March 2000, Alexandria, NSW.
3. Health Services Wing, Regimental Medical Officer Introduction Course, Army Logistic Training Centre, 20-24 March 2000, Bonegilla, Victoria.
4. RAAF-Specialist Reserve Basic Officer's Entry Course, 23-27 September 2002, Point Cook, Victoria
5. Ex-Miner's Axe Course, Murray Bridge, SA, October 2006
6. Pre-deployment courses, Op Catalyst and Slipper (Iraq and Afghanistan), Randwick Barracks, 4-8 December 2006
7. Operation "Kovi Moses", New Britain, Papua New Guinea from 1-14 October 2008 (recovery of remains from A16-126 Hudson Bomber crash, February 1942).
8. Holsworthy Army Base DVI course 6-10 July 2015

OVERSEAS EXPERIENCE:

UNITED KINGDOM:

April-December 1995, Universities of Glasgow and Edinburgh.
(Forensic Fellow)

Case work supervised by Professor P. Vanezis (Glasgow) and Professor A. Busuttil (Edinburgh).
Also worked with Drs MT Cassidy, JH Macfarlane, MA Black (Glasgow) and Drs B. Purdue and P. Fineron (Edinburgh).

EAST TIMOR:
19 January-13 February 2000

-Employed as a consultant forensic pathologist by the United Nations in East Timor to assist in excavation of remains from multiple grave sites in enclave of Oecussi province.

Approximately 45 bodies exhumed over one week period (see above).
Assisted by INTERFET soldiers from Australian Army and RAAF.

CHRISTMAS ISLAND:
September 2001

-Employed by the Australian Defence Force on Christmas Island as a forensic pathologist, in attempting to excavate the remains of the Unknown Sailor from the HMAS Sydney, which was lost at sea in the Indian Ocean during a naval battle with the German Cruiser "Kormoran" on 19 November 1941.

Both ships located in Indian Ocean 16-17 March 2008.

THAILAND:
10-27 February 2005

Assisted the Australian Federal Police in Disaster Victim Identification (DVI) at Phang Nga, near Phuket, Thailand, following the 2004 Boxing Day Tsunami.
Approximately 400 bodies examined externally and 20 full autopsy examinations during period of deployment.

PAPUA NEW GUINEA:
October 2008:

Operation "Kovi Moses", New Britain, Papua New Guinea from 1-14 October 2008 (recovery of remains from A16-126 Hudson Bomber crash, February 1942).

AWARDS or MEDALS:

Australian Federal Police Medal, following Bali Bombing, October 2002

AD Cala

AD Cala
26 November 2018

Sensitive: Legal

Inquiry into the convictions of Kathleen Megan Folbigg

7 November 2018

Dr Allan Cala
Senior Staff Specialist
Newcastle Department of Forensic Medicine
John Hunter Hospital
Lookout Road
NEW LAMBTON NSW 2305

C/O
Deanne Tadros
Senior Legal Officer
NSW Ministry of Health
73 Miller Street
NORTH SYDNEY NSW 2060

by email: Deanne.tadros@health.nsw.gov.au

copied to: Ian.fraser@fjc.net.au; Blaise.lyons@health.nsw.gov.au

Dear Dr Cala

Letter of engagement

Summary

On 22 August 2018 the Governor of New South Wales directed that an inquiry be 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 ("the Inquiry"). The Crown Solicitor is the Solicitor Assisting the Honourable Reginald Oliver Blanch AM QC ("the Judicial Officer") with the Inquiry.

As discussed, you are engaged to prepare an expert report for the Inquiry which:

1. sets out your response, if any, to the undated report of Professor Stephen Cordner provided with the petition prepared on Ms Folbigg's behalf; and

2. in light of any medical advances since 2003 relevant to your area of expertise, advises whether the views you expressed at trial have changed or remain the same, and the reasons therefore.

As discussed, we would be grateful if you could provide your report to Amber Richards, Senior Solicitor for the Crown Solicitor by **Friday 26 November 2018**. Please get in touch if you encounter difficulties in meeting this date.

You may also be required to give oral evidence at the public hearings of the Inquiry. It is expected the hearings will take place at the Industrial Relations Commission Building located at 47 Bridge Street, Sydney in **late February or early March 2019**. We will contact you once the hearing dates for the Inquiry have been listed.

Background

Trial

Between 1 April 2003 and 21 May 2003, Ms Folbigg stood trial in the Supreme Court before Barr J and a jury, upon an indictment containing five counts in respect of the deaths of her four infant children. Those counts were as follows:

Count 1: the murder, on 20 February 1989, of Caleb Gibson Folbigg, 19 days old.

Count 2: maliciously inflict, on 18 October 1990, grievous bodily harm upon Patrick Allen Folbigg with intent to do grievous bodily harm, 4 months old.

Count 3: the murder, on 13 February 1991, of Patrick Allen Folbigg, 8 months old.

Count 4: the murder, on 30 August 1993, of Sarah Kathleen Folbigg, 10 months old.

Count 5: the murder, on 1 March 1999, of Laura Elizabeth Folbigg, 19 months old.

The essential issue at trial was whether the Crown could establish that each child's death and Patrick's apparent life threatening event ("ALTE") was a result of a deliberate act of suffocation and not the result of natural causes. The Crown case was that the deaths and the ALTE; the circumstances in which they occurred (including by way of coincidence reasoning); evidence of Ms Folbigg's relationships with, and attitude towards, each child; entries made by Ms Folbigg in her diaries (some of which were said to amount to admissions); and the medical evidence, established that the only rational inference open was that Ms Folbigg unlawfully killed each child.

On 21 May 2003, the jury found Ms Folbigg guilty of three counts of murder in respect of Patrick, Sarah and Laura; one count of manslaughter in respect of Caleb; and one count of maliciously inflict grievous bodily harm in respect of Patrick in respect of the ALTE.

Ms Folbigg was sentenced on 24 October 2003 to an overall sentence of imprisonment of 40 years with a non-parole period of 30 years. This was later reduced on appeal to 30 years' imprisonment with a non-parole period of 25 years and she remains incarcerated at Silverwater Women's Correctional Centre. Ms Folbigg brought appeals against conviction on 17 February 2005, 2 September 2005 and 21 December 2007. These appeals were unsuccessful.

Petition for review of conviction

After having exhausted her right of appeal, on 10 June 2015, pursuant to s. 76 *Crimes (Appeal and Review) Act 2001* ("the CAR Act") a petition on behalf of Ms Folbigg was presented to the Governor of New South Wales seeking an inquiry into her convictions. Accompanying Ms Folbigg's petition were several expert reports, including:

- an undated report by Professor Stephen Cordner (forensic pathologist); and
- a report dated 1 June 2015 by Dr Michael Pollanen (forensic pathologist).

Direction
On 22 August 2018, the Governor of New South Wales issued a Direction pursuant s. 77(1)(a) of the *CAR Act* that an inquiry be conducted into the convictions of Ms Folbigg. The primary focus of the Inquiry is expected to be on expert medical evidence, including:

- Any new research or advances in medical science relevant to the causes of death of each child and the cause of the apparent or acute life threatening event in respect of Patrick.
- Expert medical opinion as to the causes of death of each child and the cause of the apparent or acute life threatening event in respect of Patrick in light of any relevant new research or advances in medical science.
- Any new research or literature concerning the incidence of reported deaths of three or more infants in the same family attributed to unidentified natural causes.
- Any other related expert medical evidence.

Preparation of your report

I understand that you performed the post-mortem examination of Laura Folbigg, prepared a report of your findings and gave evidence at Ms Folbigg's trial as to the causes of death of the children.

The Inquiry would be assisted if you could prepare a report which includes the following, having regard to the material with which you are briefed:

1. your response, if any, to the undated report of Professor Stephen Cordner (provided at **Tab 9** of your brief); and
2. in light of any medical advances since 2003 relevant to your area of expertise, a discussion as to whether the views you expressed at trial have changed or remain the same, and the reasons therefore (your report is at **Tab 1** and the transcript of your evidence at **Tab 7**).

The report should only offer opinions on these matters to the extent that they are based upon your knowledge, training and fields of specialist expertise.

In preparing your report, please:

i. identify (and reference as appropriate) any facts and assumptions from materials upon which you rely;

ii. show how those facts and assumptions relate to your opinions;

iii. provide an explanation of your reasons for each of your opinions; and

iv. if necessary, set out any qualification or reservations you have about the opinions expressed in your report (for instance, because of reservations you hold about a fact, or if further research or information is required, or for any other reason).

Documents with which you are briefed

For the purpose of your engagement, you are briefed with the documents set out in **Annexure A** to this letter.

If you believe you would be assisted by any further documents, please advise us of the same.

Giving of evidence before the Inquiry

You may be required to give evidence orally before the Inquiry at the public hearings. It is expected the hearings will take place at the Industrial Relations Commission Building located on 47 Bridge Street, Sydney in **late February or early March 2019**. We will contact you once the hearing dates for the Inquiry have been listed. Please also keep us informed of any planned periods of leave.

Expert Code of Conduct and Curriculum Vitae

At **Annexure B** to this letter I set out the Expert Witness Code of Conduct and ask that you read it carefully. In the report you should acknowledge that you have read the Code and agree to be bound by it. I suggest the following form of words be included in the body of your report:

> "I, Dr Allan Cala, acknowledge for the purpose of Rule 31.23 of the Uniform Civil Procedure Rules 2005 that I have read the Expert Witness Code of Conduct in Schedule 7 to the said rules and agree to be bound by it."

I also request that you please attach a copy of your curriculum vitae to your report.

Confidentiality

Please ensure you keep your engagement, the documents with which you are briefed, and your report **confidential**.

Conclusion

Please do not hesitate to contact Amber Richards on (02) 9258 0832 or amber.richards@cso.nsw.gov.au if you have any queries or require anything further to assist in the preparation of your report.

Kind regards

A.B. Richards

Amber Richards
Senior Solicitor
for Crown Solicitor
Encl.

Sensitive: Legal

ANNEXURE A

Index to briefing documents of Dr Allan Cala

Tab	Document	Date
1.	Autopsy Report of Laura Elizabeth Folbigg by Dr Allan Cala	26 July 1999
2.	MFI 27: Autopsy Report of Laura Elizabeth Folbigg by Dr Allan Cala; and Neuropathology Report of Laura Elizabeth Folbigg by Dr M Rodriguez	 26 July 1999 7 June 1999
3.	MFI 24: Article, Distinguishing Sudden Infant Death Syndrome From Child Abuse Fatalities (RE0036) (American Academy of Pediatrics, Volume 107, Number 2, pages 437-441) by Dr Allan Cala	February 2001
4.	Letter from Dr Allan Cala re IL-10 gene theory	19 March 2003
5.	Expert Certificate / Statement of Dr Allan Cala	28 March 2003
6.	Report of Professor Roger Byard	14 April 2003
7.	Transcript of evidence of Dr Allan Cala at trial (pages 705-765)	15 April 2003 – 16 April 2003
8.	Transcript of evidence of Professor Roger Byard at trial (pages 1195-1259)	7 May 2003
9.	Report and Opinion in the case of Kathleen Folbigg by Professor Stephen Cordner	Undated
10.	Peer Review of Professor Cordner's Report on the Folbigg case by Michael Pollanen	1 June 2015
11.	Chapter from Book, SIDS - Sudden infant and early childhood death: The past, the present and the future edited by Jhodie R. Duncan and Roger W. Byard (University Adelaide Press 2018): Chapter 24: The Autopsy and Pathology of Sudden Infant Death Syndrome by Roger W. Byard	N/A

Sensitive: Legal

ANNEXURE B

Uniform Civil Procedure Rules 2005, Sch 7: Expert Witness Code of Conduct

1 Application of code

This code of conduct applies to any expert witness engaged or appointed:

(a) to provide an expert's report for use as evidence in proceedings or proposed proceedings, or

(b) to give opinion evidence in proceedings or proposed proceedings.

2 General duties to the Court

An expert witness is not an advocate for a party and has a paramount duty, overriding any duty to the party to the proceedings or other person retaining the expert witness, to assist the court impartially on matters relevant to the area of expertise of the witness.

3 Content of report

Every report prepared by an expert witness for use in court must clearly state the opinion or opinions of the expert and must state, specify or provide:

(a) the name and address of the expert, and

(b) an acknowledgement that the expert has read this code and agrees to be bound by it, and

(c) the qualifications of the expert to prepare the report, and

(d) the assumptions and material facts on which each opinion expressed in the report is based (a letter of instructions may be annexed), and

(e) the reasons for and any literature or other materials utilised in support of each such opinion, and

(f) (if applicable) that a particular question, issue or matter falls outside the expert's field of expertise, and

(g) any examinations, tests or other investigations on which the expert has relied, identifying the person who carried them out and that person's qualifications, and

(h) the extent to which any opinion which the expert has expressed involves the acceptance of another person's opinion, the identification of that other person and the opinion expressed by that other person, and

(i) a declaration that the expert has made all the inquiries which the expert believes are desirable and appropriate (save for any matters identified explicitly in the report), and that no matters of significance which the expert regards as relevant have, to the knowledge of the expert, been withheld from the court, and

(j) any qualification of an opinion expressed in the report without which the report is or may be incomplete or inaccurate, and

(k) whether any opinion expressed in the report is not a concluded opinion because of insufficient research or insufficient data or for any other reason, and

(l) where the report is lengthy or complex, a brief summary of the report at the beginning of the report.

4 Supplementary report following change of opinion

(1) Where an expert witness has provided to a party (or that party's legal representative) a report for use in court, and the expert thereafter changes his or her opinion on a material matter, the expert must forthwith provide to the party (or that party's legal representative) a supplementary report which must state, specify or provide the information referred to in clause 3 (a), (d), (e), (g), (h), (i), (j), (k) and (l), and if applicable, clause 3 (f).

(2) In any subsequent report (whether prepared in accordance with subclause (1) or not), the expert may refer to material contained in the earlier report without repeating it.

5 Duty to comply with the court's directions

If directed to do so by the court, an expert witness must:

(a) confer with any other expert witness, and

(b) provide the court with a joint report specifying (as the case requires) matters agreed and matters not agreed and the reasons for the experts not agreeing, and

(c) abide in a timely way by any direction of the court.

6 Conferences of experts

Each expert witness must:

(a) exercise his or her independent judgment in relation to every conference in which the expert participates pursuant to a direction of the court and in relation to each report thereafter provided, and must not act on any instruction or request to withhold or avoid agreement, and

(b) endeavour to reach agreement with the other expert witness (or witnesses) on any issue in dispute between them, or failing agreement, endeavour to identify and clarify the basis of disagreement on the issues which are in dispute.