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SPECIAL INQUIRY

THE HONOURABLE THOMAS BATHURST AC KC

MONDAY 14 NOVEMBER 2022

INQUIRY INTO THE CONVICTIONS OF KATHLEEN MEGAN FOLBIGG

Mr I Fraser for the Ministry of Health
Ms S Love for the Commissioner of Police
Mr D Jordan SC with Ms V Garrity for the Director of Public Prosecutions
Dr G Woods SC with Dr R Cavanagh and Mr W Buxton for the Applicant
Mr D Graham SC with Dr T Waterhouse for the Australian Academy of Science
Ms K Richardson SC with Ms K Boyd for Dr A Cala
Ms S Callan SC with Ms J Roy and Ms N Wootton - Counsel Assisting the Inquiry

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CALLAN: Your Honour, nearly 20 years ago, Kathleen Folbigg was found guilty of manslaughter in respect of her first baby, Caleb, of murdering her subsequent three young children, Patrick, Sarah and Laura, and also, in relation to Patrick, of maliciously inflicting grievous bodily harm. Ms Folbigg is presently serving a 30-year sentence of imprisonment which expires on 21 April 2033. She exhausted all appeal rights many years ago, but by petition dated 2 March 2021, Ms Folbigg asked the Governor of New South Wales to pardon her in relation to these convictions. The Governor's power to do so is set out in the *Crimes (Appeal and Review) Act 2001* (NSW), which preserves in statutory form the royal prerogative of mercy. That legislation also permits the Governor to direct that an inquiry such as this be undertaken, and the Governor has appointed your Honour to conduct this inquiry. The inquiry provides for a process whereby the Governor obtains information for the purpose of considering whether she will exercise the prerogative of mercy.

On completing this inquiry, your Honour is to cause a report on the results of the inquiry to be sent to the Governor, where upon the Governor may dispose of the matter as she considers just. The nature of this inquiry is different from that of a judge and jury in a criminal trial. This inquiry must commence with the fact that convictions have been recorded but that questions or doubts have been raised sufficient to justify the Governor directing this inquiry be conducted. This inquiry may consider any information that may throw light on the convicted person's guilt. Your Honour is not bound by rules of evidence and may have regard to all of the information and evidence received, whether that information is favourable or unfavourable to the convicted person. It is not fettered by tactical or forensic decisions at trial or by the way the Crown or defence cases were conducted. That directly informs my role with Ms Roy and Ms Wootton along with the whole of the assisting team, which is to assist your Honour.

This is not an adversarial exercise. We have and will continue to engage

productively with the parties and the witnesses. Over the coming days, for instance, we will be scrutinising and testing certain evidence. This is aimed at assisting your Honour's consideration of all of the evidence and information, including its strengths and limitations. It ought not be mistaken for us advancing a position one way or another about the ultimate question, which is a matter for your Honour and your Honour alone as to Ms Folbigg's guilt. It is not our role to take a position but rather, as I have said, to assist your Honour in examining all relevant evidence and information. Your Honour's ultimate task is to consider the evidence at the trial and the conduct of the trial in light of the further evidence and submissions received in the inquiry in order to determine whether, overall, there is a reasonable doubt as to Ms Folbigg's guilt. The Act requires your Honour to form your own concluded opinion on this question and report this to the Governor.

In addition to furnishing a report to the Governor on the results of the inquiry, if your Honour is of the opinion that there is reasonable doubt as to the guilt of Ms Folbigg, your Honour may refer the matter, with a copy of the report, to the Court of Criminal Appeal for consideration of whether her convictions ought be quashed, which has a different legal character to the mercy a Governor may grant.

Your Honour, this is not the first inquiry into Ms Folbigg's convictions. During 2018 and 2019, an inquiry was conducted by the Honourable Reginald Blanch AM KC, culminating in a report issued to the Governor in July 2019 which concluded there was no reasonable doubt as to Ms Folbigg's guilt, finding the investigations undertaken since trial had failed to identify a reasonable, natural explanation for the four deaths and for Patrick's ALTE, individually or together, and that the only conclusion reasonably open is that somebody intentionally caused harm to the children, smothering was the obvious method, and the evidence pointed to no person other than Ms Folbigg. A significant volume of evidence and information was obtained in the 2019 Inquiry, which forms part of the body of evidence and information before your Honour.

Since 2019, further specific information about a genetic variant in the DNA samples of Sarah and Laura Folbigg has emerged. This is referenced in the direction issued on 18 May 2022 by the Governor, which has been marked Exhibit 1 in this inquiry. The direction commences that it appears that there is a doubt or question as to part of the evidence in proceedings leading to the conviction of Kathleen Megan Folbigg on 21 May 2003. It goes on to state:

> "That doubt or question concerns evidence that a genetic variation CALM2-G114R, identified in DNA samples from Sarah Folbigg and Laura Folbigg, has biophysical and functional consequences that may cause cardiac arrythmias and sudden, unexpected death in young children, such consequences being the subject of research published in March 2021 following the completion in July 2019 of the previous inquiry into said convictions."

Your Honour, I will come shortly to outline the case upon which Ms Folbigg was convicted and the effect of subsequent evidence and information gathered

to date.

At the outset, it is to be recognised that the central question at all times has been whether Ms Folbigg caused the death of one or more of her four children or whether they died of natural causes. There is not and has never been, for instance, a suggestion that another person caused their death. As the jury was instructed at trial, there were effectively three possibilities open on the evidence: identified natural cause; unidentified natural cause; or deliberate suffocation. The expert medical evidence at trial was significant. Some 22 experts, including forensic pathologists, gave their expert opinion on various matters. Since the trial, numerous further experts across a range of scientific and medical specialities have provided reports, including for the purpose of the 2019 Inquiry, in support of Ms Folbigg's Petition to the Governor in 2021, and for this inquiry. This corpus of medical and scientific evidence is weighty and dominates consideration of Ms Folbigg's convictions. However, it is not the only source of evidence relevant to the question of her guilt. Another significant category of evidence were diaries and journals she maintained when the children were alive. I will come to those in due course.

At earlier directions hearings in this inquiry, it has been indicated the focus of this inquiry will be relevant research and/or advances in medical science, and expert opinion in light of such research or advances, as to the cause of death of each child and the cause of Patrick's ALTE, or apparent life-threatening event. With that focus in mind, an obvious starting point is the genetic variant CALM2-G114R and its implications for consideration of the cause of death of Sarah and Laura Folbigg. This is the topic the current hearing block will begin to explore. However, as your Honour made clear in ex tempore reasons given on 6 September 2022 when addressing the proposed focus of this inquiry, your Honour is ultimately required to examine the whole of the evidence in considering whether there is a real possibility that one or more of the deaths of the children resulted from natural causes. It would be incorrect for the inquiry to focus only on evidence going to the genetic variant CALM2-G114R. To that end, the hearing block scheduled for February 2023 will also include specific consideration of Ms Folbigg's diaries and journals, and may also involve examination of other medical or scientific evidence, for instance in relation to the cause of deaths of Caleb and Patrick Folbigg.

What I now propose to do, in order to give a sensible framework for the coming days and the hearing in February 2023, is to first outline key evidence in the prosecution of Ms Folbigg, including about the life and death of each of her four children. Then I propose to summarise information and evidence obtained during the 2019 Inquiry of particular pertinence. Finally, I will briefly outline steps taken by this inquiry, including reviewing the evidence gathered to date. I propose to sketch out the essence of views expressed by experts relevant to the genetic variant CALM2-G114, including what appear to be areas of common ground and where dispute and disagreement emerge.

Detailing first the evidence giving rise to Ms Folbigg's convictions, by way of background, she met Mr Craig Folbigg in 1985; they married in 1987 when she was 20 years.

On 1 February 1989, Caleb Folbigg was born at 40 weeks. On 2 February 1989, Caleb was examined by Dr Barry Springthorpe, a consultant paediatrician, who noted noisy breathing and respiratory distress which subsequently improved.

On 17 February 1989, Caleb was examined by Dr Springthorpe, who diagnosed laryngomalacia, or floppy larynx. Three days later, on 20 February 1989, Caleb died aged 19 days. The evidence at trial was to the effect that he was put to bed at about 8:00pm. He was last seen by both parents about 10 or 10:30pm. Kathleen Folbigg fed Caleb at 1:00am, and he was asleep by 2:00am. She awoke at 2:50am to check on Caleb, and Craig Folbigg awoke to Kathleen screaming, "My baby, there is something wrong with my baby." At 2:59am, ambulance officers established that Caleb was in cardiac arrest. The final autopsy report of 9 May 1989 listed the cause of death as SIDS - that is, sudden infant death syndrome - with no external signs of injury. Your Honour, the term SIDS refers to the sudden death of an infant during sleep which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history.

A diagnosis of SIDS may only be made if there are no suspicious circumstances surrounding the death. It is to be relevantly compared with a cause of death being described as "undetermined", which indicates death from some unknown cause, be it natural, accidental, or inflicted - that is, homicide. SIDS was, at the time of the trial and remains to this day, a diagnosis of exclusion based on an absence of any other cause of death, and it is understood as being multifactorial.

Nearly 18 months after the death of Caleb, on 3 June 1990, Patrick Folbigg was born at 39 weeks. Ms Folbigg was aged 22. In light of Caleb's death, a sleep study was conducted on Patrick on 15 June 1990, involving ECG leads being attached to the chest and abdomen to detect spells of apnoea and periodic breathing over a nine-hour period. The results of that study were normal. On 18 October 1990, Patrick suffered an apparent life-threatening event, or ALTE, aged four months and 15 days. The evidence at trial indicated he was put to bed at 8:30pm. At about midnight or 1:00am Ms Folbigg heard Patrick coughing, but went back to sleep. At about 4:30am, Mr Folbigg awoke to Ms Folbigg screaming at the end of Patrick's cot. Mr Folbigg commenced CPR. Patrick was taken to the Mater Hospital. An electroencephalogram or EEG, which measures electrical activity in the brain appeared normal at that stage.

Between 19 and 20 October Patrick had approximately 15 fits. He was responsive to an anti-convulsive drug and valium. On 29 October 1990 Patrick was discharged. The diagnosis recorded was, "intractable seizures, probably viral encephalitis", meaning inflammation of the brain and also “bronchiolitis”. Patrick was subsequently admitted to hospital on 4 November 1990 following a further seizure. A CT scan conducted on 5 November 1990 demonstrated a deterioration at the back of his brain. An EEG indicated abnormalities potentially indicative of encephalitis or epilepsy.

By November 1990 Patrick was unable to fix on a face and was responsive only to bright lights. He was again admitted to hospital on 14 November 1990 following a further seizure, and then again on 22 December 1990. On 13 February 1991, Patrick died aged eight months and ten days. At about 10:00am, Craig Folbigg received a phone call from Kathleen Folbigg stating, "It's happened again, I need you to come home." Ambulance officers arrived at 10:10am and at that time Patrick was warm to touch. At 10:40am Patrick was pronounced dead at the Mater Hospital. The Final Autopsy Report recorded the clinical diagnosis as, "Encephalopathic disorder leading to intractable seizures. The underlying cause of encephalopathy not determined on investigation" and "Asystolic cardiac arrest at home leading to death."

Twenty months later, on 14 October 1992, Sarah Folbigg was born at 39 weeks. Ms Folbigg was aged 25. On 5 November 1992, a sleep study was conducted on Sarah, with normal results. When she was taken home, Sarah slept on an apnoea mat in her cot, although use ceased before her death. On 6 November 1992, Dr David Cooper reported to a paediatrician, Dr Henry, that Sarah had "quite long hyperventilation, hypopnoea event, one of them about 40 seconds." In late August 1993, Sarah suffered a cold or flu for which she was treated with flucloxacillin, later taken on 26 or 27 August.

On 30 August 1993, Sarah died aged ten months and 16 days. There were conflicting accounts as between Mr and Ms Folbigg as to events that evening, which your Honour will have to resolve in your findings. Mr Folbigg's version is that he woke at 1:10am and noticed Sarah and Ms Folbigg were not in the bedroom where he, Ms Folbigg and Sarah slept, and that a light was on. Mr Folbigg had originally told police that Sarah was in her bed, but at trial he said that was a lie he had given to police. Ms Folbigg's account to police was that Sarah had not left her bed that night. She had noticed Sarah was not breathing when she returned from going to the toilet at 1:30am. She denied she was feeding Sarah or had left the room prior to going to the toilet. Paramedics arrived at 1:30am. They observed Sarah was cyanosed around the mouth. That is a bluish colour caused by shortness of oxygen in the blood. And her airway was obstructed. At 2:10am Sarah was asystolic, meaning her heart had stopped, and ambulance officers ceased treatment.

The Final Autopsy Report by Professor John Hilton listed the cause of Sarah's death as SIDS. And his observations included, "Focal pulmonary collapse, modest pulmonary congestion and minimal oedema, occasional petechiae on pleura, epicardium and on/in thymus, congested haemorrhagic uvula lying anterior to the epiglottis and aspiration of gastric content." Professor Hilton's external examination included noting two tiny punctuate abrasions on Sarah's face, one immediately below the lower lip on the left side and the other slightly to the left side of the mid-line of the chin. The uvula was of normal size but appeared congested or haemorrhagic on its anterior surface, and one section of the larynx showed a light mixed lymphocytic inflammatory infiltrate deep to the respiratory epithelium.

Four years later, on 7 August 1997, Laura Folbigg was born at full term. At 12 days old, Laura underwent full biochemical blood and metabolic

investigations which were normal. A sleep study showed mild central apnoea and no obstructive apnoea which corrected itself by February 1998. During the first year of her life, Laura was often monitored during sleep using a Corometrics home cardiorespiratory monitoring device, which was a sleep mat designed to record and download breathing and heart information during Laura's sleep. Use of the monitor decreased after the first six months and ceased when she was 12 months. This monitoring did not indicate any problems.

On 22 June 1998 Laura was taken to Singleton Hospital and diagnosed by Dr John Cash, a visiting medical officer, with an upper respiratory tract infection and mild croup. On 14 August 1998 she was taken to Dr Paul Innis, GP, with flu like symptoms and diagnosed with a viral upper respiratory tract infection. On 19 January 1999 Laura was taken to Dr Innis with a macular red rash and her throat was red. She was prescribed phenergan. Three days later, at a further review by Dr Innis, Ms Folbigg reported Laura had fevers over the past few days. She was diagnosed with a viral rash. On examination, her throat was red, but no additional treatment was prescribed. On 5 February 1999, at her 18 month immunisations, Laura's throat and ears were clear.

On 1 March 1999, Laura died aged 18 months and 22 days. On the morning of 1 March Ms Folbigg took Laura to Mr Folbigg's workplace for a visit. She left about 11:30am. Laura fell asleep in the car on the way home, so she put Laura to bed for a nap. Ms Folbigg subsequently discovered Laura unresponsive, so placed her on the firm surface of the breakfast bar, commenced CPR and called 000. At 12:14pm, ambulance officers attended at the Folbigg home where Laura was lying on the breakfast bar, not breathing and with an unobstructed airway. At 12:17pm, an ECG monitor was applied and Laura was administered adrenalin. On 13 December 1999, Dr Allan Cala prepared a Final Autopsy Report. The cause of death was listed as "undetermined" with “incidental” findings of myocarditis and inflammation of the muscular walls of the heart.

The police investigation into the death of the four Folbigg children commenced on the afternoon of Laura's death on 1 March 1999. Detective Senior Constable Ryan attended at Singleton Hospital and spoke to both Mr and Ms Folbigg there. The same day, police attended at the Folbigg residence and conducted crime scene investigations. On 23 July 1999, Ms Folbigg participated in a lengthy interview with police. In June 2000, Kathleen and Craig Folbigg permanently separated.

Diaries and journals made by Ms Folbigg were obtained by police during the course of their investigation. These diaries and journals did not span the entire decade from the birth of Caleb in 1989 to the death of Laura in 1999. Rather, the diaries obtained covered the period February to March 1989, the year 1990, then June 1996 to April 1998, and there were diaries for 1999. I will come back to these diaries and journals.

On 19 April 2001, Kathleen Folbigg was arrested and charged with the murder of her four children. Subsequently, the Crown added a charge of one count of

maliciously inflicting grievous bodily harm in respect of the ALTE of Patrick Folbigg on 18 October 1990.

At trial, the Crown relied on evidence of the facts and circumstances of the birth and deaths of each of the four children. The Crown also relied, as I have said, on a significant number of expert witnesses, and I'll come to their evidence in a moment.

The Crown was permitted to rely on evidence relating to the deaths of each child and Patrick's ALTE in proof of the other counts. This is often referred to as evidence being “cross-admissible” in support of tendency and coincidence reasoning. Coincidence reasoning concerns the improbability of two events happening just by coincidence. Tendency reasoning rests on reasoning that a person has a tendency to conduct themselves in a particular way. As to coincidence reasoning in the case against Ms Folbigg, the Crown relied on evidence of similarities in the circumstances of the four deaths and Patrick's ALTE, which made it improbable, so the Crown said, that the deaths were merely coincidental.

These similarities included the following. First, all four were children of Kathleen and Craig Folbigg. Second, all four children were under two years. Third, the deaths occurred suddenly. Fourth, the deaths occurred unexpectedly, that is the children were healthy. Fifth, the children all died at home during sleep when in a bed, cot or bassinet. Sixth, the deaths all occurred when the only person at home or awake was Ms Folbigg, said by the Crown to give her the opportunity to have done them harm. Seventh, each child was discovered dead or moribund by Ms Folbigg when they were still warm to the touch and two still had a heartbeat. They were discovered by her during what she claimed was an ordinary check on their wellbeing and on three occasions when she said she was on her way to the toilet. Eighth, except for Laura, Ms Folbigg failed to render the children any assistance at all after discovering them.

The Crown argued these relevant similarities were so substantial and remarkable that when taken in conjunction with other evidence, including the expert medical evidence, the presence of hypoxia, that is the absence of enough oxygen to sustain bodily functions, and the absence of clear medical reason for the occurrences of the ALTE or sudden deaths, the only reasonable hypothesis left open was that Ms Folbigg asphyxiated them with the intent to kill or do grievous bodily harm.

Turning to tendency reasoning, the Crown contended that Ms Folbigg had a tendency to become stressed and lose her temper and control with each of her children and then to asphyxiate them. The Crown accepted that before the evidence could be used for tendency reasoning, the jury would need to be satisfied beyond reasonable doubt that in relation to any one of the children the accused had caused his or her ALTE or death. Then the jury could use that conclusion and the remaining circumstantial or tendency evidence to assist in deciding whether she was responsible for the other deaths or ALTE.

The Crown relied on two primary sources of evidence to establish the asserted tendency by Ms Folbigg to lose control and asphyxiate her children. First, the evidence of Craig Folbigg that detailed in relation to each of Patrick, Sarah and Laura, instances in which Ms Folbigg had lost her temper, become frustrated, growled or yelled at her children. The second source of evidence relied upon by the Crown were content of the diaries and journals written by Ms Folbigg. Indeed, over and above relying on these diaries for tendency reasoning the Crown case was that Ms Folbigg's diaries were an intimate, personal and exact analysis of what her thinking was during the times the diaries were available.

JUDICIAL OFFICER: Ms Callan, did Ms Folbigg dispute that she became frustrated, growled and yelled at her children during the police interview or in her evidence before Blanch J?

CALLAN: She did. She certainly disputed the characterisation.

JUDICIAL OFFICER: So I will also have to assess the reliability of Mr Folbigg's evidence on this account?

CALLAN: Yes.

JUDICIAL OFFICER: Thank you.

CALLAN: The Crown at trial, your Honour, characterised certain entries in her diaries, particularly in combination, as admissions of guilt, suggesting the diaries were the strongest evidence that you could possibly have for Ms Folbigg having murdered her four children. Specific consideration of the diaries will form part of the next hearing block in February 2023.

After the jury returned a guilty verdict in May 2003, Ms Folbigg appealed. Her appeal against convictions was dismissed by the Court of Criminal Appeal but it upheld her challenge to sentence, which was reduced to 30 years. Ms Folbigg will be eligible for parole on 21 April 2028, and her sentence will expire on 21 April 2033.

An application for special leave to the High Court was heard and refused on 2 September 2005.

Then, on 27 November 2007, the Court of Criminal Appeal heard a further appeal against conviction in relation to an argument that the trial miscarried because a juror or jurors obtained information from the internet which revealed that Ms Folbigg's father had killed her mother, and a juror or jurors informed themselves away from the trial as to the length of time an infant's body is likely to remain warm to the touch after death. The appeal was dismissed with the Court of Criminal Appeal stating they were satisfied the irregularities were not material and did not give rise to a miscarriage of justice.

Can I turn, then, to address the relevant expert medical evidence called at trial in relation to the deaths of the four children. Specifically in relation to Caleb

Folbigg, Consultant Paediatrician Dr Barry Springthorpe gave evidence noting that Caleb had a congenital laryngeal stridor, almost certainly on the basis of laryngomalacia, but observed there was nothing in the way of “laryngeal webs or cysts” at autopsy and said the stridor had nothing to do with his death. Dr Springthorpe's evidence was that an airway could collapse in the absence of cysts or webs. He also said it was possible to smother a young baby and leave no external signs at all. In his view, the cause of Caleb's death remained a mystery.

In relation to Patrick Folbigg, Dr Joseph Dezordi had examined Patrick on admission to hospital in October 1990. In his view, the ALTE was likely caused by a fairly catastrophic event such as an asphyxiating event or a prolonged seizure.

Dr Gurpreet Singh-Khaira, a histopathologist who conducted the initial and final autopsy in respect of Patrick Folbigg, his evidence was that he could not exclude that Patrick died of a seizure leading to a catastrophic asphyxiating event.

Dr Ian Wilkinson, a Paediatric Neurologist, gave evidence at trial that he excluded encephalitis as a cause of Patrick's death, and he was no longer of the view he had expressed in Patrick's death certificate that epileptic fits led to the asphyxiation which caused Patrick's death. This change of view was affected by consideration of the other deaths in the family.

Turning to Sarah Folbigg, Professor John Hilton who had conducted the post-mortem examination of Sarah Folbigg, found her cause of death was SIDS. He gave evidence at trial regarding the deaths of both Sarah and Laura Folbigg. As to Sarah, Professor Hilton said the findings of the internal examination in the post-mortem were consistent with an asphyxia mode of death which would include deliberate smothering. In his view, the location of the petechiae and the changes to Sarah's lungs tended to favour a diagnosis of SIDS as compared to intentional suffocation. The inflamed uvula was a factor in a SIDS diagnosis and could have had some role in Sarah's death but he could not say for certain its position at the time of death as compared to the time of the post-mortem.

In respect of Laura Folbigg, Professor Hilton's evidence was that myocarditis was a highly significant finding. It did not exclude deliberate suffocation as a cause of death, but Professor Hilton disagreed with Dr Cala that a video of Laura apparently well the day before her death was significant in terms of whether the myocarditis was a cause of death. Professor Hilton's evidence was that Laura's abrasions were trivial and not suggestive of an effort of an adult to suffocate a child. Professor Hilton gave evidence at the 2019 Inquiry, and I'll refer to that in a moment.

Forensic Pathologist, Dr Allan Cala, did the post-mortem examination of Laura Folbigg and gave evidence in relation to the cause of death of all four children. As to Caleb, in his view, the cause of death was undetermined, and your Honour, it is to be recalled that "undetermined" encompasses unidentified

natural and unnatural causes of death.

As to Patrick, Dr Cala's evidence was that it was possible Patrick's ALTE had resulted from deliberate smothering, and the cause of Patrick's death was undetermined with suffocation not ruled out.

In relation to Sarah's death, he also considered this was “undetermined” stating SIDS was not appropriate due to her age and the abrasions on her lower lip. He said there was a “possibility” she died of an acute and catastrophic asphyxiating event.

In respect of Laura, Dr Cala's view was that she did not die of SIDS as she was too old for this diagnosis and had an intercurrent illness, myocarditis, which “might” have explained her death. He said if he had examined Laura in isolation without knowledge of previous infant deaths in the family, he might have given the cause of death as myocarditis but could not ignore “any known relevant family history of severe illness or premature deaths”. Furthermore, having watched the video of Laura Folbigg two days before her death, he was even more firmly of the view that myocarditis played “no role whatsoever in her death and was an incidental finding”.

In Dr Cala's view, Laura's cause of death was “undetermined” and was probably an acute catastrophic asphyxiating event of unknown causes, consistent with smothering including deliberate smothering. He could not exclude myocarditis but considered it “very unlikely” to be the cause of death.

Dr Cala's evidence was that in relation to all four children, there was an “exceedingly remote possibility, so remote as to be almost impossible, of some underlying undiagnosed genetic or metabolic disease which has so far not been detected.” He said he was “very suspicious that all four Folbigg children may have died as a result of deliberate smothering” but the medical evidence did not allow him to take this any higher than a “suspicion”. Dr Cala also gave evidence in the 2019 Inquiry, and I will address that shortly.

Can I turn, then, to Professor Peter Berry, a Consultant Paediatric Forensic pathologist who also gave evidence at trial. In his view, Caleb's cause of death was not SIDS because of the finding of haemosiderin in the lungs, which was not diagnostic of suffocation but meant the death should be investigated further, such that he would have given the cause of death as unascertained.

For Patrick, the ALTE was not part of the usual natural history of SIDS and Dr Berry considered it most unlikely to have been caused by an epileptic seizure. He would have given the cause of death as unascertained, ascribing it to brain damage following an unexplained collapse. Dr Berry said one had to consider the possibility of some kind of asphyxia episode that caused Patrick's ALTE.

As to Sarah, Dr Berry said the death resembled SIDS, albeit she was older than most SIDS deaths. Considered in isolation, he would have ascribed SIDS as the cause of her death. In his view, the displacement of the uvula was due

to dissection of the throat but accepted if it was displaced prior to death, it could have had an effect on respiration.

For Laura, Professor Berry stated that determining the significance of the myocarditis was “very difficult”. It presented as an “explanation for death” but could be an incidental finding. He also said there was no finding to exclude suffocation. Professor Berry stated that taken together, he would have ascribed the cause of death as suffocation because he said, "sudden death of four infants in the same family who were previously well…due to natural disease is unprecedented in my experience".

That view was also reflected in the expert evidence of Professor Peter Herdson, Consultant Forensic Pathologist, who adopted the so-called “Meadow's law” in his evidence at trial to the effect that the first unexplained death of an infant in a family may be attributed to SIDS; the second should be labelled "undetermined"; and the third should be considered homicide until proven otherwise.

JUDICIAL OFFICER: Can I just ask you this just about that?

CALLAN: Yes.

JUDICIAL OFFICER: As I understood the position, the trial judge excluded at least specific reference to “Meadow's law”.

CALLAN: Yes, your Honour.

JUDICIAL OFFICER: That evidence of Professor Herdson was admitted, I think, without objection, was it not?

CALLAN: I'll have to check that, your Honour.

JUDICIAL OFFICER: It certainly wasn't the subject of any appeal in any event.

CALLAN: Your Honour, there had pre-trial rulings to carve out the permissible grounds for the evidence.

JUDICIAL OFFICER: But whether it was excluded or not, Professor Herdson went very close to it, and in a different way, so did the Crown Prosecutor in his closing address.

CALLAN: Yes, your Honour, and I should say that that reasoning has been widely discredited in more recent times. It could be described as “unscientific”, and in a legal context wholly inconsistent with the prosecution bearing the burden of proof, and the accused person's entitlement to the presumption of innocence. Can I make clear that our submission is “Meadow's law” should not be accepted, nor should any evidence which relies upon such reasoning be given any weight in your Honour's task.

JUDICIAL OFFICER: Does that mean that at least in the case of Professor

Berry and I think Dr Susan Beal, their evidence has to be taken with some degree of care?

CALLAN: Yes. And Professor Herdson as well.

JUDICIAL OFFICER: Yes.

CALLAN: For that reason, your Honour, I have not elaborated for instance on the details of the evidence of Dr Beal or Professor Herdson. Can I move on to the evidence of Professor Roger Byard, Forensic Pathologist, who gave evidence in the defence case. His view as to the cause of death of each child was altered somewhat whether he considered the position individually or taken together.

As to Caleb, taken either individually or along with the other deaths, Professor Byard would label the death as undetermined, noting a history of breathing problems involving laryngomalacia or floppy larynx. He stated there was no positive evidence of suffocation of Caleb but that there can be suffocation with no positive findings at all in babies. In respect of the presence of haemosiderin, he stated that this was not indicative of suffocation and simply meant the case should be investigated carefully.

As to Patrick, Professor Byard's view was that approached individually, the cause of death was epilepsy against a background of possible encephalitis, but taken along with the other deaths he would label the cause of Patrick's death as undetermined but cannot exclude epilepsy. He could not exclude the possibility that the ALTE was the first manifestation of epilepsy or a seizure disorder. He also said Patrick's death was consistent with a seizure. There were no findings that could amount to proof of death by suffocation or that the ALTE was suffocation.

As to Sarah, taken either individually or along with the other deaths, Professor Byard would label the cause of death as undetermined with narrowing of the upper airway. He could not exclude the position of the uvula as being significant but that it could have been a post-mortem artefact.

Finally in relation to Laura, taken individually Professor Byard would say cause of death is myocarditis, but taken along with the other deaths, he labelled her cause of death as undetermined but cannot exclude myocarditis. He stated, myocarditis was “the sort of inflammation I have seen in a number of cases of sudden death in children.” In his view, the fact that Laura died when she was asleep did not exclude myocarditis, and as to the video of Laura shortly before her death, he could not place significance on it because infants and children could have significant and lethal diseases and show little external manifestation. A home video, he said, did not amount to a clinical examination.

Professor Byard stated he could not exclude the possibility that any of the four children's death and Patrick's ALTE was caused by natural causes.

Your Honour, I'll now turn to the genetic evidence at trial. Plainly enough there has been much by way of development in medical science since that time. Geneticist, Dr Bridget Wilcken, reported on the results of the testing and analysis of samples of blood of each of the four Folbigg children. Her evidence at trial was that something like a total of 50 genetic metabolic disorders which might be associated with unexpected death had been tested for and were excluded. Dr Wilcken provided a statement to the 2019 Inquiry, recognising there had been a great deal of development in genetic testing since the trial and recommended consulting with a clinical geneticist and genomic expert. In due course, that occurred, and I'll describe it in a moment. To complete the expert evidence at trial, Dr Owen Jones, a paediatric specialist called in the defence case, expressed the view there was no credible evidence for an inherited disorder of cardiac rhythm, such as long QT syndrome, in the Folbigg family. On this topic, as I've said, plainly there's been much by way of development, to which I will turn. In relation to Caleb, Patrick and Sarah, Dr Jones considered there was no evidence of an intrinsic congenital or acquired cardiac abnormality causing or contributing to their death.

In relation to Laura Folbigg, there was good evidence she had myocarditis, which was a well-recognised cause of unexpected sudden death. In his view, the absence of clinical features of heart failure could not be used to argue against myocarditis as a cause of death. In this respect, I note the evidence of Dr Ian Bailey, Paediatric Cardiologist, was that the ECG of Laura suggested she ceased breathing before her heart stopped, whereas Dr Jones disagreed the ECG could show this sequence. His evidence was that he was not in a position to comment on the probability of myocarditis being a cause of death of Laura Folbigg. Your Honour, that completes my overview of the medical and scientific evidence at the time of the trial.

JUDICIAL OFFICER: Would it be fair to say that the approach of the Crown at the trial was to seek to exclude identifiable natural causes, at least in each case except Caleb, and then rely simply on coincidence evidence and the diaries?

CALLAN: Yes, your Honour, insofar as the coincidence evidence permitted consideration of similarities. Yes, your Honour. Can I turn then to the 2019 Inquiry which was sought by petition from Ms Folbigg and ordered by Governor Hurley in a direction which indicated “the doubt or question concerns evidence as to the incidence of reported deaths of three or more infants in the same family attributed to unidentified natural causes”. The material before your Honour in this Inquiry includes the evidence tendered in the 2019 Inquiry along with the transcripts of the hearings conducted as part of that Inquiry. As has already been recognised at directions hearings conducted in this Inquiry, your Honour is not reviewing or assessing the merits of the 2019 Inquiry.

JUDICIAL OFFICER: Mr Blanch took a somewhat adverse view of Ms Folbigg's evidence, having seen her. You accept that I'm in a position to reach a contrary conclusion notwithstanding the fact that she's not giving evidence at this Inquiry?

CALLAN: You are, your Honour. It plainly has to be tempered by the fact that you are not in a position to observe her in the witness box, but we have both the transcript of the hearing and also the audio recording of that hearing.

A number of experts provided reports and gave evidence in the 2019 Inquiry which are of considerable assistance to the work of this Inquiry. Overall, your Honour, what shines through is the hard work and careful attention paid by a number of highly qualified experts to the very many topics addressed in that Inquiry. Their evidence deals with complex and, in some instances, emerging fields of medicine and science, which I will outline to the extent presently relevant. First, expert evidence was provided by Professor Rosemary Horne, Infant Sleep and SIDS Specialist, and Professor Dawn Elder, Paediatrician, who described advances in understanding about SIDS since the time of the trial, including as to major risk factors as well as refinements in the definition of SIDS, including the introduction of subcategorisation. These expanded subcategories accommodate some deaths which might have previously been described as undetermined.

Professor Horne, amongst other things, observed that genetic studies indicate up to 35% of SIDS cases might be explained by familial or genetic diseases such as cardiomyopathies, ion channelopathies or metabolic disorders that remained undetected during conventional forensic autopsy procedures. Overall, however, the underlying cause of the majority of SIDS cases still remains elusive and it is likely due to a multifactorial aetiology triggered by a combination of different genetic and environmental risk factors. Relatedly, Mucosal Immunologist and Foundation Professor of Pathology, Professor Robert Clancy AM, and Clinical and Mucosal Immunologist, Professor Caroline Blackwell, along with Clinical Microbiologist, Professor Paul Goldwater, gave evidence as to the role of infection in SIDS deaths. Professor William Rawlinson AM, Senior Medical Virologist, provided a report indicating the unviability of testing the Folbigg children's tissue samples for viruses. The next category of expert evidence which occupied a substantial portion of the oral hearings in the 2019 Inquiry was in the field of forensic pathology, and that incorporated the evidence of Professor Johan Duflou, Professor John Hilton, Professor Stephen Cordner, and Dr Allan Cala.

Common ground between the four Forensic Pathologists appear to have been, first, there was no unequivocal cause of death for the four Folbigg children. Second, earlier research as to there being an extremely low risk of more than one SIDS death in a family belied the possibility of genetic bases for SIDS, which would result in subsequent siblings having an elevated risk. Also, that SIDS and fatal suffocation can be indistinguishable in infants due to the absence of discernible physical injury. In respect of Caleb, Forensic Pathologists agreed that haemosiderin in his lungs was not a positive indicator of superimposed upper airway obstruction, and as to blood and froth around Caleb's mouth at the time Ms Folbigg found him, Professors Cordner, Duflou and Hilton described this as being not uncommon in SIDS cases. Dr Cala said this did not exclude the possibility of an external agent having been applied to Caleb's outer airway. In relation to Caleb's laryngomalacia, or floppy larynx, Professor Cordner considered this potentially made Caleb more vulnerable to

SIDS. Professor Duflou noted only 10% of cases of laryngomalacia can be fatal if untreated. Professor Hilton's view was that it could not be determined whether laryngomalacia contributed to death, and Dr Cala did not accept it caused the death.

As to the cause of Caleb's death, Professors Duflou, Cordner and Hilton would have ascribed Caleb's death as SIDS category 2, due to his age and laryngomalacia. Dr Cala would have ascribed Caleb's death as undetermined, owing to concerns about Caleb's age and the report of blood and froth around his mouth. He was unconvinced Caleb died of SIDS.

In respect of Patrick, the Forensic Pathologists agreed the cause of the ALTE was “unknown” or “unexplained”. They agreed that Patrick had an encephalopathic disorder at the time of his death, but encephalitis was excluded as the cause of death. Professors Cordner and Duflou considered Patrick's death to be the consequence of epileptic seizures due to the encephalopathic disorder, the underlying cause of which was not determined. Professor Hilton stated Patrick's death was part of an epileptic-type illness. Dr Cala was unconvinced that Patrick died of epilepsy. He would not have given epilepsy as a cause of death, and maintained that smothering could have explained both the ALTE and death. I pause to note that specifically on the cause of Patrick's ALTE and death, evidence was also given to the 2019 Inquiry by Paediatric Neurologist, Associate Professor Michael Fahey and Professor Monique Ryan. Professor Ryan has provided a further report to this Inquiry, and to the extent that topic is to be specifically explored, this will occur in the hearing block next year.

Turning to Sarah Folbigg, the Forensic Pathologists agreed that punctuate abrasions on her chin were not uncommon in young children, and were possibly the result of resuscitation attempts. They also agreed that the petechial haemorrhaging was unlikely to be significant. In relation to the swollen uvula, Professors Cordner and Duflou considered this was relevant to a SIDS diagnosis, Professor Hilton considered it was possibly significant, and Dr Cala said it was irrelevant to the cause of death. As to cause of death, Professors Hilton, Duflou and Cordner would ascribe the cause of death of Sarah Folbigg as SIDS, with Professors Duflou and Cordner classifying it as Category 2. Dr Cala would ascribe the cause of death as “undetermined”. He was unconvinced Sarah died of SIDS. In respect of Laura Folbigg, it was uncontroversial she had myocarditis at the time of her death. The Forensic Pathologists accepted this was a condition that can lead to death and which may or may not produce observable symptoms in a child of her age. Professors Cordner, Hilton and Duflou considered that myocarditis was the most likely cause of death. Dr Cala considered that Laura did not die from myocarditis, but it could not be positively excluded.

Finally, can I turn to the evidence obtained in the 2019 Inquiry on the topic of genetics and cardiology. The 2019 Inquiry obtained samples from each of the four children and Ms Folbigg. DNA was extracted from these samples and genetic data obtained and analysed. Mr Folbigg was invited to produce a DNA sample but declined to do so. I note he maintains that position. Two sets of

cardiac tests were performed on Ms Folbigg in April 2019 by Associate Professor Hariharan Raju, a Cardiologist and Electrophysiologist at Macquarie University Health Science Centre. Associate Professor Raju has provided an expert report to this Inquiry addressing Ms Folbigg's cardiac history and will give evidence shortly. A number of other cardiac and genetic experts provided evidence in the 2019 Inquiry, including the findings of their genomic sequencing analysis of Ms Folbigg and her four children. This body of evidence has been of considerable assistance, upon which the work of this Inquiry builds. The cardiac and genetic experts who gave evidence in the 2019 Inquiry were Dr Alison Colley, Dr Michael Buckley, Professor Edwin Kirk and Professor Jonathan Skinner, who together provided joint reports. Professors Kirk and Skinner have provided a further report for this inquiry and will be called to give evidence.

There was also evidence given in the 2019 Inquiry from Immunologist, Professor Carola Garcia de Vinuesa, Professor Matthew Cook, and then Dr, now Professor Todor Arsov. They have prepared a joint report for this Inquiry and will be called to give evidence. Finally, there was evidence provided in the 2019 Inquiry from a Genetic Cardiologist, Dr Kathryn Waddell-Smith, who addressed in general terms the approach to be taken to genetic variants when considering the cardiac health of a child.

Your Honour, before examining the significance of the CALM2 mutation found in Laura, Sarah and Kathleen Folbigg, it is useful to recognise what I might describe as a number of foundational matters in relation to genetics which emerge from the evidence. Like many organisms, much of the human body is made up of proteins. To borrow a description from Professor Kirk, proteins are the building blocks of cells and the padding between cells. Any time your body has a job to do, it gives it to a protein. If your cell wanted to make a car, every single mechanical and electrical component would be made from proteins, and so would the garage you parked your car in. DNA is a chemical that contains genetic information for building and maintaining an organism by coding for particular protein. The genome is a complete set of the DNA in an organism. Almost every cell in the human body contains that person's genome, which was inherited from a combination of their biological parents. The information in DNA is written in an alphabet of four letters, A, C, G and T. These stand for the four nucleobases which are the chemical building blocks of DNA. In DNA, those nucleobases are arranged into clusters of three which form into one of 20 different amino acids. A gene is a stretch of amino acids in the DNA that codes for a particular protein. A twenty-first arrangement of nucleobases tells the body to stop. That is to complete that particular protein code. As Professor Kirk put it, “you can think of nucleobases as the letters, amino acid names as the words they spell, and genes as sentences. Each sentence explains how to build a particular protein, and each molecule of DNA contains many of these sentences. It's a manual for building parts of the body.”

A genetic variant, which is what we're concerned with, in considering the variant CALM2-G114R, is a permanent change in the DNA sequence. There is considerable genetic variation between human beings. On average, a

person will have three million places at which their genome differs from their neighbours. Most of these sit in between the genes or in a place that does not affect the protein code for a gene. About 40,000 variants in each person on average result in a difference in the parts of the genes that code for protein. Most of these variations are benign or have very subtle consequences, and account for our differences, who has brown eyes, who has blue, who is tall, who is short, et cetera.

Variants are identified by comparing differences between the DNA of an individual who has been tested and the reference genetic sequence. The reference sequence is not necessarily normal or even representative of the most common sequence at any given base. But it is used as a common reference point by testing laboratories.

JUDICIAL OFFICER: Can I ask you this, what's the R stand for? For example, there's a difference between G114R and G114W. You may be coming to it.

CALLAN: Yes, your Honour, I am coming to it. The R stands for tryosine. But in your Honour, and I'll come to it - sorry, no the R stands for arginine, abbreviated as ARG or R. The G, as in G114R is glycine.

JUDICIAL OFFICER: I understand the G, it's the R and the W.

CALLAN: It's the R which arginine and the W which is the abbreviation for tryptophan.

JUDICIAL OFFICER: Where do they fit into these sequences?

CALLAN: Your Honour, I'll come to a slide in a moment to show you how the variations--

JUDICIAL OFFICER: I'm sorry.

CALLAN: No problem. Your Honour, while many gene variants have no or negligible effect, some genetic variations are disease causing or pathogenic. The term genetic mutation is sometimes used instead of genetic variant – for instance, in respect of variants that are capable of causing disease. But, for the purposes of this Inquiry the term variant will be used. The term phenotype means an observable trait. And in the genetic context refers to the clinical manifestation or presentation of a person's genes. Tall with brown eyes is a phenotype for the genes that demonstrate those traits. In the case of a disease causing or pathogenic genetic variant the phenotype is the observable manifestations of the diseases. For instance, in the case of cystic fibrosis which is a genetic disease cause by variants in a single gene, the phenotype is the observable signs and indicators of cystic fibrosis such as lung infections or pneumonia and the progressive damage to the lungs, digestive system, and other organs of the body which are a feature of that genetic disorder.

The field of genetics is at an age of rapid expansion and discovery. There was a huge amount of progress between the trial in 2003 and the Inquiry in 2019, with more progress since then. It is recognised that a great deal remains unknown. Various guidelines are available for the classification of genetic variants. Such guidelines have a particular application in clinical genetics. The purpose of such guidelines is to impose a level of consistency and rigor to the task of classifying genetic variants. The American College of Medical Genetics and Genomics and the Association for Molecular Pathology Guidelines, referred to as the ACMG Guidelines, were used to varying extents in the evidence before this Inquiry and in 2019.

A few preliminary points on variant classification use of these guidelines. First, variant classification is a probabilistic exercise in which different types of evidence are weighted and combined to assess the probability that a particular variant is disease-associated. Under the ACMG Guidelines, genetic variants are classified into one of five categories: benign; likely benign; variant of uncertain significance; likely pathogenic, and pathogenic. Benign means that there is a very high degree of confidence the variant is harmless. At the other end of the spectrum, pathogenic means a very high degree of confidence a variant is associated with Mendelian disease. That is a disorder caused by mutation in a single gene.

Likely pathogenic refers to a variant that is assessed as having sufficient evidence to be medically actionable. That is, in a clinical setting at a 90% confidence threshold. Variants that have some evidence of pathogenicity but insufficient to reach the 90% confidence threshold, or for which there is conflicting evidence, are categorised as variants of uncertain significance. There was in the 2019 Inquiry, and remains in this Inquiry, an issue as to whether the stringency of the ACMG Guidelines is suitable in the present context. It is also to be recognised that classification of a variant as pathogenic or likely pathogenic does not automatically imply that it has, or will, cause a clinically manifest disease. The reason for this will be addressed by various experts. Furthermore, of course, a person may carry a genetic disorder and yet die from something totally unrelated.

Of the hundreds of variants in the genetic testing of Ms Folbigg and her children, more than 20 were the subject of detailed analysis in the 2019 Inquiry and among those was a variant identified as CALM2-G114R. That variant found in Ms Folbigg and her two daughters, Sarah and Laura, has assumed particular ongoing relevance. That variant was not found in Caleb or Patrick Folbigg, and no other genetic evidence in any of the children looms large. That is to say, no pathogenic or likely pathogenic variant in genes could explain the unexpected death of four out of the four children. However, the CALM2 variant found in Kathleen, Sarah, and Laura Folbigg was novel. That is, it has never been seen before by the scientific community and the question was, could this variant be disease-causing.

Your Honour, the CALM genes, of which there are three, contain the body's instructions for making calmodulin. Calmodulin is a protein, which amongst other things, plays an important role in the regulation of sodium, potassium

and calcium, and consequently the electrical activity of the heart. Variants in the CALM genes appear to be very rare and scientific knowledge in this field is relatively recent and still emerging. What has been observed is that all genetic variants in the CALM genes will materially change the calmodulin protein or its function. But those that do - sorry, what has been observed is that not all genetic variants in the CALM genes will materially change the calmodulin protein or its function. But those that do are capable of causing a cardiac condition described as calmodulinopathy. Calmodulinopathy is a collective description for an arrhythmogenic condition. That is, a condition causing an abnormal heart rhythm.

The three primary conditions are Long QT Syndrome, Catecholaminergic Polymorphic Ventricular Tachycardia or CPVT; or Idiopathic Ventricular Fibrillation. Each condition is capable of causing sudden death. The reason for this is because for every heart beat to occur a rapid movement of sodium, potassium and calcium ions across the cardiac cell wall is required for depolarisation and repolarisation of the cardiac cells. If the channels through which the ions travel are defective, then repolarisation or depolarisation is abnormal and there is a risk of serious ventricular arrythmia. An abnormal heart rhythm causing sudden syncope, that is blackout, cardiac arrest, or sudden death.

The diagram in this slide shows a typical heart rhythm you would normally see repeating on an electrocardiogram or ECG. In the case of Long QT syndrome, as the name implies, the interval between the Q and the T waves is longer than normal, which you can see marked at the bottom of the diagram. This indicates it is taking the heart longer than is typical to electrically repolarise ready for the next electrical signal. Long QT syndrome produces a heart rhythm abnormality which can interfere with the heart's ability to pump blood around the body. It can generally be seen on an ECG. When triggered, Long QT syndrome can progress into lethal arrythmias.

CPVT involves rapid beating of the ventricles triggered by catecholamines, which are neurotransmitters such as dopamine and adrenalin. This interferes with the heart's ability to pump blood and can ultimately cause the heart to stop beating if not treated. CPVT is not always visible on an ECG, but can be, particularly if triggered by the release of neurotransmitters, for instance by exercising.

Idiopathic, that is of unknown cause ventricular fibrillation or IVF, causes an unpredictable rhythm that does not enable the heart to pump blood around the body at all. There will be no signs of a future IVF on and ECG, though if an ECG was applied while ventricular fibrillation was occurring it would certainly be immediately apparent. It is lethal if not quickly interrupted.

So, going back to the variants in the CALM genes which can produce a calmodulinopathy, one mechanism by which this can occur is if a CALM variant interferes with the ability of either or both the Ryanodine Receptor 2 or RyR2 gene, and the Cav1.2 or calcium channel voltage dependent L-type alpha 1C subunit gene, to inhibit the release of calcium in the heart cells, by reducing

the ability of the calmodulin protein to bind to calcium. The less able the calmodulin protein is to bind to calcium, the less able these channels are to stop or slow the release of calcium at the times necessary to maintain a normal heartbeat and rhythm.

You can see these channels on the slide. In green, which is the Ryanodine Receptor channel which releases calcium, the little yellow balls, from the sarcoplasmic reticulum into the interior of the heart cell. And in blue, which is the Cav1.2 channel to the extra cellular space, showing passage of calcium out of the heart cell.

As depicted in the diagram, the stronger the effect of the CALM variant on the calmodulin protein's ability to bind calcium, the more severe the disease. Calmodulin mutations or variations with minute or no measurable effect on calcium bindings, do not seem to affect the Cav1.2 channel. That is, the blue channel controlling calcium release from heart cells into the extra cellular space, and therefore have no effect on the QT interval. That is, such variants do not cause long QT syndrome. However, these variations may still lose the ability to inhibit the Ryanodine Receptor or RyR2 channel, causing CPVT. Calmodulin variants with some moderate effect on calcium binding can affect the QT interval, such that some individuals with this variant have a mild Long QT Syndrome; others display a mixture of CPVT and Long QT Syndrome, and yet others are diagnosed with CPVT. Calmodulin variants which strongly reduce the calcium binding effect can have a very severe effect on both channels and produce the more severe, and earlier onset, of Long QT Syndrome.

We move to the next slide, and this, your Honour, starts to provide some indication as to the location of the variant we're considering here. The focus of the evidence, as I've said, in the 2019 Inquiry was whether the CALM2-G114R variant ought be classified as a variant of uncertain significance, or likely pathogenic; that is, whether it possibly caused a calmodulinopathy, and thus the possibility this occasioned Sarah and Laura's death.

One of the relevant factors used to predict the pathogenicity of a new, or “novel” variant such as this one is looking at its location on the gene compared to known variants. The location of the CALM2-G114R gene is indicated on this diagram with the red arrow. The top two images on screen depict the location of known pathogenic mutations on the calcium bound calmodulin structure.

Your Honour, in the bottom half of the diagram, the three lines show the protein encoded from the CALM genes; that is, CALM1, CALM2 and CALM3. Each letter in the line represents the amino acid that should be produced at that location. The name of the variant in this case CALM2-G114R indicates it appears in the CALM2 gene; that is, the middle of the three lines. G, which is an abbreviation for "Glycine", indicates the amino acid that should appear at location 114. Your Honour may observe the location intervals are marked in increments of 20 along each line.

JUDICIAL OFFICER: Yes.

CALLAN: The red arrow indicates position 114, and the R indicates the amino acid is Arginine abbreviated as ARG or R, rather than Glycine. The significance of this change will be explored in the evidence. Each of the coloured circles above the three lines indicates a previously identified pathogenic variant, with the colours indicating the associated phenotypes - red for Long QT Syndrome, orange for mixed Long QT Syndrome CPVT, yellow for CPVT, and blue for IVF.

You can see from the third line the G114 variant is in the same location 114, as a recognised variant on the CALM3 gene, marked by the blue W on the bottom line. There the Glycine amino acid or G has been replaced with a W, the abbreviation for Tryptophan. The CALM3-G114W variant has been reported as causing IVF. Your Honour, I will refer to this further shortly. It has been the subject of particular consideration, and I should note that this diagram comes from evidence obtained in the current Inquiry, but if I may return to the position as it developed in the 2019 Inquiry.

Once the novel variant CALM2-G114R was identified in Ms Folbigg and her two daughters, the various genetic experts undertook the task of classifying it; that is, assessing the probability that it was disease-associated. They did so by reference to relevant known information and guided to varying degrees by the ACMG Guidelines.

As stated earlier, your Honour, there were a number of genetic variants which the experts analysed and classified. This variant was one of many. But focusing on the classification of CALM2-G114R, there was divergence in the expert evidence as to whether the variant should be classified as “likely pathogenic” or a variant of uncertain significance. Briefly, the joint view of Dr Colley, Dr Buckley, Professors Kirk and Skinner, was that it was a variant of uncertain significance.

Their reasons, in summary, was that while there was accumulating evidence to support a relationship between CALM variants and Long QT Syndrome, these had arisen *de novo*; that is, not inherited from their parents. Furthermore, while there were multiple lines of computational evidence supporting a deleterious effect of the variant on the gene, it was also observed in a healthy adult individual, namely Kathleen Folbigg.

Professor Kirk explained in oral evidence that the apparent lack of cardiomyopathic symptoms in Kathleen Folbigg indicated the CALM2 variant was unlikely to be pathogenic in Sarah and Laura.

Professor Skinner added in oral evidence there was no known link between sudden infant death of children, that is when asleep or at rest, and Calmodulin. Like Professor Kirk, he considered the CALM2 variant was unlikely to have been pathogenic in the Folbigg children, because at over 50 years of age, and having undergone various forms of cardiac testing, Kathleen Folbigg had not developed or exhibited any clinical sign of

cardiomyopathy. By contrast, the report of Professors--

JUDICIAL OFFICER: Is the reason they gave for saying it arose *de novo* because people who had it would have died before they had a chance to pass it on to their descendants?

CALLAN: Yes, your Honour. The view expressed in the report of Professors Vinuesa and Cook noted there were additional considerations that they gave weight to, including the region where the variant occurred and a lack of clarity as to whether Ms Folbigg was affected by Long QT Syndrome. Overall, they classified the variant as likely pathogenic. In oral evidence, Professor Vinuesa expressed the view that whilst Ms Folbigg might be completely healthy, that did not rule out that she could be affected by the variant, and in any event this did not render the variant benign.

Professor Vinuesa explained that her team classified the variant as likely pathogenic because in their opinion, “CALM2 is a novel variant. Precisely because they are novel the potential for pathogenicity is greater than those that might be already known, for example those proven pathogenic”.

Dr Buckley and Professor Kirk took issue with the correctness of several criteria used to arrive at this conclusion of likely pathogenic, suggesting it led to undue weight being placed on the likelihood the variant was pathogenic. Shortly after oral evidence in the 2019 Inquiry, an article by Lia Crotti and others was published outlining insights from the International Calmodulinopathy Registry. This Inquiry will hear evidence about the Register, including from one of its founders Professor Peter Schwartz.

The Crotti article was provided to the 2019 Inquiry and was then addressed in further written reports from various experts, including Professor Vinuesa as well as Dr Buckley and Professor Kirk. This body of further material was addressed in an addendum included in the report of the 2019 Inquiry.

The Crotti article described for the first time the CALM3-G114W variant which was classified as likely pathogenic. While this was not the variant found in Kathleen, Sarah and Laura, it provided further information to support a conclusion that CALM2-G114 variant was likely pathogenic. This was certainly Professor Vinuesa's view. However, Dr Buckley and Professor Kirk expressed doubt, emphasising the need to take into account clinical information as to the presentation of Kathleen, Sarah and Laura Folbigg in interpreting the significance of the G114R variant. However, they recognised, particularly given the rarity of the group of conditions and the full range of possible clinical manifestations is not known, that it was possible the variation CALM2-G114R was pathogenic.

Your Honour, that was the state of the evidence about CALM2 variant when the 2019 Inquiry concluded. I will now turn to subsequent developments in evidence and information about the CALM2 variant, including steps taken since the commencement of this Inquiry.

As I adverted to earlier, the CALM2-G114 variant is specifically referenced in Ms Folbigg's petition to the Governor, and the Governor's direction for this Inquiry to be conducted. Since your Honour's appointment as Inquirer in May of this year, a number of steps have been taken to progress the Inquiry. I propose to take a moment to outline briefly those steps in recognition of the work which forms the immediate backdrop to the hearing this week, and in February 2023, and to acknowledge the productive contribution of all the parties, and a number of experts, already.

Prior to the first directions hearing which occurred on 24 June 2022, the Inquiry obtained and reviewed all material tendered in the 2019 Inquiry, plus the trial transcript. That material was served on the parties and has been tendered as Exhibits 2, 3 and 4. The hearing of the 2019 Inquiry was live-streamed and while video recordings of that hearing are not available, audio recordings are and they have been added to Exhibit 4.

Material obtained from the 2019 Inquiry included photocopies of Ms Folbigg's diaries and journals that were available. The Inquiry has taken a number of steps to secure the originals of those diaries and journals, which have now been received into the Inquiry's possession. Also, the Inquiry took a number of steps to secure the available portions of the video recording of Ms Folbigg's interview with Police on 23 July 1999. In a moment I will tender that.

Directions hearings were conducted on 24 June, 27 July and 6 September during which various preliminary matters were addressed, including granting various parties leave to appear, namely Ms Folbigg, Mr Folbigg, the New South Wales DPP, the New South Wales Commissioner of Police, the New South Wales Department of Health, Dr Cala, and the Australian Academy of Science.

Those assisting your Honour have conducted two ‘all-parties’ meetings with the legal representatives for the parties. The first occurred shortly after the directions hearing, on 29 June 2022, and featured a productive discussion about the conduct of the Inquiry. Much more recently, there was an all-parties meeting last Tuesday, 8 November 2022. This featured another productive discussion about the conduct of this hearing segment, and preparations for the next hearing segment.

Your Honour, the 2019 Inquiry Report contained a comprehensive summary of relevant evidence. This was extracted by the assisting team into a table format and distributed to the parties in mid-August 2022 with a request that the parties indicate any aspects which they contended were not accurate. The parties engaged with this task in a timely way, for which we are most grateful.

Subject to some specific comments raised on behalf of Ms Folbigg, the Commissioner of Police, the Australian Academy of Science, and the DPP, the overwhelming majority of the evidence summaries appear to be uncontroversial. This provides the Inquiry and all involved with a significant volume of foundational facts and information, which has resulted in efficiencies and allows focus on the real issues in dispute.

Can I turn, then, to further evidence which has been received by this Inquiry to date. In relation to forensic pathology, supplementary reports have been received from Professor Hilton, Professor Cordner, and Professor Duflou. Dr Cala is expected to provide a supplementary report also. I will tender those reports in a moment.

In short, these supplementary reports from the forensic pathologists address the implications of the evidence about the CALM2 variant for their view about the cause of death of each of the four children. If any oral evidence is to be sought from these Forensic Pathologists, it is proposed this will occur during the hearing block in February next year.

As I mentioned, there has been provided a supplementary report from Professor Monique Ryan of 23 September 2022, and that will be tendered shortly. If she is to be called to give oral evidence, it is proposed this will occur in February next year.

We have also been provided with a report of Professor Peter Fleming, Consultant Paediatrician and Professor of Infant Health and Development Physiology at the University of Bristol in the UK. He addresses current knowledge in relation to SIDS and, amongst other things, the significance of the absence of observable physical damage to each of the Folbigg children. The Inquiry has also been provided with a report of Professor Ray Hill of 7 April 2015. He is an Emeritus Professor of Mathematics, addressing from a statistical perspective the likelihood of multiple deaths from SIDS in a family.

JUDICIAL OFFICER: That report wasn't before the Blanch Inquiry, was it, notwithstanding its date?

CALLAN: I'll have to check, your Honour. It's not my understanding it was tendered in the Blanch Inquiry, notwithstanding its date. In relation to cardiac and genetic evidence, what I might describe as further primary evidence relevant to the cardiac history of Ms Folbigg and Laura Folbigg has been received and will be tendered shortly. There is also, as I averted to, a report from Associate Professor Raju of 5 November 2022 outlining Ms Folbigg's cardiac history. On the topic of her cardiac presentation, I should note that during the 2019 Inquiry, it was suggested, for instance, by Professor Vinuesa, that in addition to the cardiac testing undertaken in April 2019, further cardiac testing of Ms Folbigg ought be undertaken. Those assisting your Honour have followed up on this. Professors Kirk and Skinner suggest a sprint test and an adrenaline infusion. Associate Professor Raju makes similar suggestions and also suggests an implanting loop recorder. However, Ms Folbigg has declined to participate in any further cardiac testing on the basis that the test carries some risk to her of cardiac arrest, and that a number of experts has said that her phenotype is not determinative of whether the variant caused the deaths of her daughters.

Turning to the further evidence which has been obtained specifically addressing the relevance and implications of the CALM2 variant in Sarah and

Laura Folbigg, those assisting your Honour have engaged a number of experts. Considerable input was sought from and provided by the parties so as to ensure the right experts were engaged, and they were asked the right questions. In particular, in early September of this year, correspondence was sent to all parties containing a table detailing experts in cardiology and genetics proposed to be engaged, topics and questions those experts might address in their report, and a list of documents and material to be provided to those experts. In responses provided by the parties, there was no suggestion the proposed experts were unsuitable. The DPP, Ms Folbigg and the Australian Academy of Science suggested several additional experts, proposed modifications and additions to the questions to be asked, and identified further documents to be provided to the experts. In early October 2022, a finalised list of questions and further briefing materials was issued to the cardiac and genetic experts. A number of reports have now been received. I will outline that evidence in a moment.

These reports have been provided to the parties and circulated to the other engaged experts. There has been no restriction on the experts consulting with one another, speaking with the legal representatives for the other parties, and there is no impediment to the experts listening to the evidence of their colleagues. Complementing this process, Ms Folbigg's representatives had informed the Inquiry that Professors Michael Toft Overgaard and Mette Nyegaard were providing a report. That will be tendered and they are to give evidence this week. I willl describe their evidence in a moment. Your Honour, as might be expected, this expert evidence makes reference to a number of relevant journal articles in respect of genetic mutations. These articles have been collated and will be tendered. Turning to developments since the 2019 Inquiry, in the information and evidence about the CALM2-G114R variant. Your Honour, in 2021 a group of 27 scientists published an article in the academic journal Europace titled "Infanticide vs inherited cardiac arrythmias." For convenience, we will refer to this by reference to the lead author, Marlene Brohus. A number of other authors will give evidence in this Inquiry.

The Brohus ”rticle is the research published in 2021 which is referred to in the governor’s direction for this Inquiry. The Brohus article reported the results of functional assays conducted on the CALM2-G114R variant. Those laboratory tests show that the G114R variant has adverse effects on the calmodulin protein’s function; specifically, adverse effects on calcium binding and regulation of the calcium channels I earlier referred to, involved in cardiac function. The results of those tests were compared with the results of other genetic variants known to occasion calmodulinopathy and sudden cardiac death, including in children. The authors of the Brohus article concluded that the deleterious effects of the CALM2-G114R variant, revealed by the functional tests, were sufficiently similar to those of other variants to indicate that calmodulinopathy is a reasonable explanation for the deaths of Sarah and Laura Folbigg. However, your Honour, the conclusions in the Brohus article are not universally accepted within the highly specialised scientific community that works at this cutting edge of cardiac genetics. Part of the work of this Inquiry is to better understand this work, and associated expert evidence,

about the CALM2-G114R variant.

One area to be examined is Ms Folbigg's cardiac health; that is, the nature and significance of her various fainting or near-fainting episodes. Relatedly, we will examine the relevance, if any, of the fact that Ms Folbigg has lived to full adulthood notwithstanding she carries the CALM2-G114R variant. On that topic, your Honour will hear evidence about several concepts, including: variable expressivity - that is, varying degrees of impact a genetic variant may have on different people, people with a gene who have, for instance, had no or mild symptoms of the associated disease through to people with the gene who have extreme symptoms; variable penetrance - that is, the capacity of a genetic variant to have no adverse effect at all on some people while having an effect on others; complete penetrance means a condition manifests in 100% of genetic carriers; incomplete penetrance means, for example, that 70% or seven out of ten people who carry a genetic variant might manifest the condition; mosaicism, another expression referred to in the evidence, means a genetic variant that occurs in only some copies of a person gene, so they may not be affected in a relevant way but may still pass on a genetic variant to their children, who will then be affected; and genetic modifiers - that is, other gene variants that affect how a particular variant expresses in a person. A gene modified might counteract the pathogenicity of a variant, make its effects milder or make its effects worse.

Another area to be considered is whether the results of functional testing of the CALM2-G114R variant and what phenotype this would predict, aligns with the presentation and circumstances of Kathleen, Sarah and Laura Folbigg (to the extent this is known). Further, how the presentations of Kathleen, Sarah and Laura Folbigg compared to known calmodulinopathy cases, and the relevance of undertaking such an exercise of comparison. This will include the significance, if any, of the fact that Sarah and Laura Folbigg died under two years of age and while asleep. And other differences in the presentations of Kathleen, Sarah and Laura Folbigg compared to other cases of calmodulinopathy recorded in the scientific literature and on the International Calmodulinopathy Registry. Can I turn then to the witnesses to be called. Later today, I will call Associate Professor Hariharan Raju at the Macquarie Health Cardiology Centre. He's a clinical specialist cardiologist, electrophysiologist and heart rhythm expert. As mentioned, Professor Raju conducted cardiac testing of Ms Folbigg in April 2019 and reported his results to the 2019 Inquiry. For this Inquiry, he's prepared a further report addressing Ms Folbigg's complete cardiac history. Professor Raju is a co-author of the Brohus article and has been Ms Folbigg's treating cardiologist.

In summary, Professor Raju says that Ms Folbigg has a long-standing history of transient loss of consciousness. He described her reported history of frequent syncope following exercise, and other incidents included during pregnancy or menstruation and during emotional stress. Before losing consciousness, she would feel nauseous and dizzy, and could take 30 minutes to recover after regaining consciousness. He describes these as stereotypical and either situational or reflex that is neurocardiogenic rather than arrhythmic. That is, he considers these episodes are unlikely to be related to a

cardiogenetic disorder. Associate Professor Raju reviewed the results of Ms Folbigg's cardiac testing, some of which he personally administered. These were generally unremarkable, apart from one occasion of a borderline QT length and inferior U-wave prominence of uncertain significance in an ECG from March 2003 and, on another occasion, a sharper than typical decline in blood pressure immediately post-exercise and isolated LVOT ectopy in the April 2019 ECG.

Professor Raju suggests further testing to complete Ms Folbigg's cardio phenotyping, including the implantation of a loop recorder to enable prolonged monitoring; that is, to seek to capture a transient syncope spell by continually recording Ms Folbigg's heart. Professor Raju concludes that, on the basis of available data, Ms Folbigg has either no evidence of any significant cardiogenic disease or, less likely, has an unusual presentation which cannot be readily tested for or has a true Idiopathic Ventricular Fibrillation; that is, a phenotype that does not manifest until it produces a fatal arrhythmia, which also cannot be tested for.

JUDICIAL OFFICER: How do I deal with that in circumstances where Ms Folbigg - and I'm not criticising, I should emphasise - has declined to undertake any further testing?

CALLAN: Your Honour, the effect of the evidence I anticipate before your Honour will be that whilst there is certain significance and relevance in understanding Ms Folbigg's phenotype from a cardiac perspective, that cannot be determinative of the position in respect of her children. However, all of the experts - there seems to be common ground in this respect - recognise that a particular challenge in the exercise being undertaken as to classifying and understanding this genetic variant, are the limited information available about, and in particular, the children, Sarah and Laura Folbigg. Your Honour will also hear evidence this week from two research scientists based at Aalborg University, Denmark. Professor Michael Toft Overgaard is the head of the Department of Chemistry and Bioscience, and Professor Mette Nyegaard is at the Department of Health Science and Technology. They report on a number of the functional assays which are described in the Brohus article, of which they were co-authors. Professor Toft Overgaard specialises in characterising protein structure and function with particular expertise in characterising the effect of calmodulin mutations on the calmodulin protein function. Professor Nyegaard specialises in human genetics with a focus on how calmodulin mutations cause disease and interact with modifier genes. In evidence, they will explain the essential function of the CALM genes and calmodulin protein. They will also address the functional assays conducted of the CALM2-G114R variant.

Professors Toft Overgaard and Nyegaard will describe the rarity of any variants, pathogenic or not, in the CALM genes across the general population, and the implication of this rarity, including impeding any meaningful statistical calculations.

Your Honour, late last week Professor Toft Overgaard informed the Inquiry of a

further functional assay which he had just conducted in relation to the CALM2-G114R variant. The results of that test were detailed in a further report provided to the Inquiry on Saturday, 12 November 2022. In order to ensure the other expert witnesses and the parties are afforded adequate time to consider this further report from Professors Toft Overgaard and Nyegaard, it is proposed that their evidence-in-chief will be taken this week. But then the hearing will adjourn with the balance of their evidence, along with the rest of the cardiac and genetic experts, to occur in February 2023. In this respect, those assisting your Honour recognise this is an area of science in which new developments or discoveries are emerging. Thoughtful consideration of the significance and implications of such discoveries is required and we want to ensure that this Inquiry continues to have the benefit of the considered contribution of the various eminent experts who have been engaged.

If I can continue with my outline of the cardiac and genetic evidence received to date by this Inquiry, Professors Carola Vinuesa, Matthew Cook and Todor Arsov have prepared a joint report. Previously, they gave evidence in the 2019 Inquiry. Professor Vinuesa is an Immunologist whose work has contributed to understanding pathogenesis of auto-immune diseases. Professor Cook is a Professor of Experimental Medicine at the University of Cambridge and an Immunologist whose work focuses on the analysis of the genetic and cellular bases of immune disease. Professor Arsov is a Professor of Genetics, an expert in Medical Genetics, the use of genomic technologies in diagnosis in clinical practice in genetic counselling.

Their joint report addresses, amongst other things, the significance of the CALM2-G114R variant in relation to the cause of death of Sarah and Laura Folbigg, including outlining developments in knowledge since the 2019 Inquiry, such as the analysis and conclusions in the Brohus article.

In summary, they say by reference to cases in the reported literature that the Folbigg scenario, involving the sleeping death of two infant children and lesser or no effects on their living mother, is not out of range of reported calmodulinopathies. That is, the calmodulinopathy phenotype is a spectrum which ranges from sudden cardiac death and cardiac arrest to various forms of syncope, and entirely asymptomatic cases. And the symptoms manifest at a range of ages and during a range of activities, including sleep. They also suggest that Ms Folbigg's history is highly suggestive of her having calmodulinopathy. However, whether or not she is symptomatic is not determinative of whether the variant caused the girls' deaths, because the functional study is described in the Brohus article indicate the CALM2-G114R variant is pathogenic (that is, disease-causing) and arrhythmogenic (that is, causing irregular heart rhythms).

They conclude there is a reasonable possibility the deaths of Sarah and Laura Folbigg were caused by fatal cardiac arrythmias attributable to the CALM2-G114R variant. Specifically, Professor Vinuesa concludes, "This was the most likely cause of their deaths." Professor Arsov considers this was the cause of their death, with a possible contribution from their respective viral infections at the time of death. Professor Cook considers the probability that

calmodulinopathy was the cause of death has only increased since 2019 and he is unaware of any new evidence which argues against this conclusion.

The Inquiry will also hear evidence from Professor Peter Schwartz, an internationally renowned cardiologist specialising in cardiac arrythmias of genetic origin. His main area of research has been sudden cardiac death and particularly sudden infant death caused by cardiac arrythmia, and Long QT syndrome, which he has been studying for over 50 years. He is a co-author of the Brohus article.

Professor Schwartz was one of 41 researchers who established the International Calmodulinopathy Registry. This registry collects demographic, clinical and genetic information of patients with variants in one of the three CALM genes. It currently has 135 enrolled patients, and is the foremost source of information about calmodulinopathy patients in the world. Professor Schwartz explains how the Registry can be used to assess whether the phenotypes of Ms Folbigg, Sarah and Laura fit with current knowledge of patients with calmodulin mutations. By comparing the CALM-G114 variant with similar variations recorded on the registry, in particular CALM3-G114W and the variant known as N98S.

Professor Schwartz explained why in his view the G114 variant has an intermediate affect on calcium binding which then allows modified genes and environmental factors to determine the phenotype that will manifest in a particular individual. In other words, he addresses why it may be the variant has manifested differently in Ms Folbigg and her daughters. Professor Schwartz agrees with Professors Vinuesa, Cook and Arsov that whether or not Ms Folbigg is systematic is not determinative of whether Sarah and Laura Folbigg were relevantly affected by the CALM2-G114 variant.

Professor Schwartz says while there is no evidence to prove that the CALM2-G114 variant is more likely to cause one phenotype, in preference to another, given it's apparent intermediate impact on calcium binding it is reasonable to suggest that it could cause “mild to no observable” Long QT syndrome, but “definitely” cause a high risk of CPVT or IVF, and that it could cause cardiac arrest during sleep, as has been observed in carriers of the G114W and N98S variants.

Professor Schwartz opines that as the number of patients registered on the registry grows (having doubled since the 2019 Inquiry), it is becoming evident that when a child of two or three years with a CALM variation dies suddenly, it is not possible or necessarily important to discriminate with certainty between CPVT, IVF and Sudden Infant Death. Professor Schwartz has provided a supplementary report of 8 November 2022 which presents the very latest information from the registry and responds to certain statements from other expert reports.

Can I turn then to the evidence of Professor Arthur Wilde, an internationally renowned cardiologist specialising in clinical aspects of heart failure and arrythmia, with specific research focuses including genetic factors contributing

to sudden cardiac death. Professor Wilde expresses concern about the conclusion reached in the Brohus article. Professor Wilde points out what he describes as a mismatch between the functional analyses of the CALM2-G114R variant recorded in the Brohus article and the clinical phenotypes of Sarah and Laura Folbigg.

In summary, he considers none of the three calmodulinopathy phenotypes, Long QT syndrome, CPVT or IVF, clearly fits the Folbigg case. In his view, Long QT syndrome is the most likely phenotype to cause the death of infants or young children. But the data suggests there has only been one recorded family with a pathogenetic CALM variant and a Long QT phenotype, where a parent who is likely a non-mosaic carrier of the variant is not affected by Long QT syndrome, which describes Ms Folbigg. That is, where a parent who is likely a non-mosaic carrier of the variant but is not affected by Long QT syndrome has children with a variant that are affected by Long QT syndrome. In other words, CALM variants capable of causing sudden death in infants are typically so severe in phenotype, that carriers do not survive to their reproductive years. And your Honour, that is an expression of the notion of a *de novo* variant.

JUDICIAL OFFICER: A *de novo*.

CALLAN: Professor Wilde went on to observe that in that singular family there was also at least some marginal indication of prolonged QT's in all variant carriers, even those not apparently adversely affected. Which is unlike Ms Folbigg. Professor Wilde also notes that the very limited ECG data for Laura Folbigg is not clear, but it does not seem to show excessive QT prolongation, as one would expect with CALM-associated Long QT syndrome. And I note there is no ECG data for Sarah Folbigg.

Professor Wilde does accept the possibility that modified genes could be responsible for variable expressivity, and he acknowledges the work of Professor Schwartz in successfully demonstrating this effect in relation to other Long QT-causing variants. In Professor Wilde's view, Idiopathic Ventricular Fibrillation does not fit as a cause of death because it is extremely unusual in young children and would also be inconsistent (although not impossible) with the results of the functional analyses reported in the Brohus article.

Professor Wilde agrees with the Brohus article that having regard to the functional testing, CPVT is the most likely phenotype for the CALM2-G114R variant. And, in his view, Ms Folbigg's history and ECG results are at least compatible with CPVT. However, he observes that based from clinical data of reported individuals and families with CPVT cardiac death during sleep at age 10 and 18 months is unlikely to relate to a CPVT phenotype. Importantly, he also does not agree with the Brohus article's conclusions that the effects of the CALM-G114 variant are experimentally similar to the N98S variant. This is significant because the N98S variant is associated with variable expressivity, as well as sudden unexplained death while asleep, and at a young age.

Other N98S cases are heavily relied upon for comparison in the Brohus

article. Professor Wilde considers the fact that the "calcium dependent inactivation" of the N98S variant is less than that of the G114R variant is highly significant, as this increases QT prolongation and therefore should increase the likelihood of sudden cardiac death at a young age and during sleep in the N98S patients, compared to the G114R patients.

In short, Professor Wilde does not take issue with the accuracy of the functional analyses conducted on the G114 variant. However, he considers the Brohus article overstates the persuasive force of the evidence suggesting the CALM2-G114R variant was responsible for the deaths of Sarah and Laura Folbigg. Indeed, in his view it is unlikely the variant is causal of the deaths of these children.

Can I next turn to evidence received from Professor Edwin Kirk and Professor Jonathan Skinner in a joint report to this Inquiry. Your Honour may recall as I mentioned, both gave evidence in the 2019 Inquiry. Professor Kirk is a Clinical Geneticist and Genetic Pathologist. Professor Skinner is a Paediatric Cardiologist and Electrophysiologist. Professors Kirk and Skinner make no criticism of the quality of laboratory work performed in connection with the Brohus article, which as they say, appears to have been completed to a high standard. But they point out limitations in terms of the conclusions and inferences which can be drawn.

Professors Kirk and Skinner apply the ACMG guidelines to the CALM2-G114R variant, observing the functional analyses reported in the Brohus article are not of themselves determinative of whether the CALM2-G114R is to be classified as pathogenic, likely pathogenic or a variant of uncertain significance.

Professors Kirk and Skinner observe that in a diagnostic laboratory (as distinct from a research laboratory), functional assays of the kind used to conduct the functional analysis recorded in the Brohus article are treated with caution due to their capacity to produce false positive results. For a functional assay to be used as a diagnostic test, it is necessary to have defined thresholds for identifying results as abnormal and to have evidence regarding the probability of false positives.

They cite, by way of example, two instances of functional assessments in other cases initially reported to show a variant was responsible for sudden nocturnal death and sudden infant death, but the variants were later shown not to be pathogenic. Professors Kirk and Skinner acknowledge the diagnostic exercise is severely limited in the present case by a lack of evidence as to the cardiac health of Sarah and Laura Folbigg before their deaths, and the inconclusive nature of the information available about Ms Folbigg. This limitation, as I have said, your Honour, is recognised by a number of the other experts, including Professors Vinuesa, Arsov and Cook.

Professor Skinner considers the single ECG available of Laura Folbigg cannot be used to exclude Long QT Syndrome as it could possibly be consistent with some QT prolongation; however, he says on any view it does not show the gross QT prolongation typical of a Long QT Syndrome associated with death in

infancy.

Professors Kirk and Skinner consider Ms Folbigg's phenotype to be of critical significance in whether the ACMG Guidelines produce a classification of uncertain significance or likely pathogenic. They observe that an assessment that Ms Folbigg is definitely affected by Calmodulin Disease would mean the variant classification would change from “uncertain significance” to “likely pathogenic”.

Like Professor Wilde, Professors Kirk and Skinner consider the functional analysis reported in the Brohus article suggest that CPVT is the more likely presentation for CALM2-G114R, but observed that CPVT would be an incredibly unlikely cause of death in the two Folbigg girls, having never been seen to manifest during sleep, or under the age of two.

Your Honour, based on the evidence furnished to date across all these genetic and cardiac experts, I expect it may be recognised that the task of classifying the CALM2-G114R variant is challenging in the present case because of the lack of evidence as to the cardiac health of Sarah and Laura Folbigg before their deaths, and the inconclusive nature of the information available about Ms Folbigg.

Further, I also expect it to be accepted by all that the presentation of Kathleen, Sarah and Laura Folbigg falls outside a “typical” CALM presentation, if such a thing can be said to exist. However, I also expect it may be said that this area of genetic understanding investigation is still very new. The Calmodulin variants are very rare and their pool of affected people is small, such that differences in the presentation of Kathleen, Sarah and Laura Folbigg compared to other recorded cases cannot rule out the variant as a potential cause of the girls' death.

In short, no expert is expected to tell your Honour that the CALM2-G114R variant definitely caused the death of either Sarah or Laura Folbigg. Equally, no expert is expected to tell your Honour that the CALM2 variant could not possibly have caused their death. Rather, the experts divide on where in that middle ground they land in terms of the likelihood that this novel genetic variant could have had any role to play in their deaths.

Your Honour, that concludes my opening for this segment. After hearing openings from any of the other interested parties, I propose to deal with the tender of documents, and then I will call Associate Professor Raju.

JUDICIAL OFFICER: Associate Professor Raju is available from 12 until 1pm, is that the position?

CALLAN: Yes, your Honour.

JUDICIAL OFFICER: Thank you. Does anyone else wish to say anything by way of opening?

WOODS: Yes, your Honour, if I may.

JUDICIAL OFFICER: How long, Dr Woods?

WOODS: 10, 15 minutes.

JUDICIAL OFFICER: That's fine. Just I want to get Professor Raju if I can, yes.

WOODS: Of course. I thank my learned friend Counsel Assisting for her helpful overview. From Ms Folbigg's perspective, I just want to say where she stands on a number of matters but in particular to remark upon the complexity of what confronts the Inquiry.

The processes, as a result of which human beings come to exist, continue to exist and cease to exist are in many respects mysterious, but biological and medical scientists have made enormous advances in recent decades towards understanding in these areas. It is not long since the human genome was unravelled for scientific examination and we will all have within immediate recent memory the extraordinary speed with which brilliant scientists were able to create effective vaccines against the disease COVID-19.

The scientific study of human cells and their interaction with life proceeds at pace, and new lessons from this work are learned and published if not daily, then regularly. As my learned friend has indicated, we will be privileged to hear in this Inquiry from highly qualified and distinguished experimental scientists, from equally highly qualified and distinguished medical practitioners who deal with patients, and with some who are both laboratory scientists and practising clinical doctors.

It is expected that about most matters mentioned here, the expert witnesses will largely agree with each other but there will be some areas of disagreement, or at least disagreement as to emphasis or significance or interpretation, and that is only to be expected in the complex fields of cardiology and genetics. However, this is not a Royal Commission into the state of medical science in these fields generally, but a specific Inquiry into a particular case.

Ms Folbigg has served nearly two decades of imprisonment, and the position we put forward on her behalf is that, for the reasons which will be exposed in this Inquiry, it is in fact likely that each of her infants died of natural causes and specifically, that the two daughters, Sarah and Laura, were affected by the recently discovered and pathogenic gene variant CALM2-G114R which, in our submission, either wholly caused or substantially contributed to the death of each infant.

JUDICIAL OFFICER: Well, I don't have to find what was likely to have caused their death. To find out, I've got to exclude any reasonable hypothesis inconsistent with homicide.

WOODS: Your Honour, that's so. I'm not inviting your Honour to, as it were, determine the matter on the balance of probabilities.

JUDICIAL OFFICER: I just want to make it clear that's not what I'm going to do.

WOODS: Of course, I understand that, yes. Now, my learned friend has given an extraordinarily effective analysis of the technology and the science. I don't pretend to explain in full scientific terms the way that the genetic variant in question here raises an issue before the Inquiry. I don't pretend to explain how it can interfere with a function of the complex and mysterious organ, the heart, which is so central to human survival. The expert witnesses will do so no doubt with great accuracy, which I won't attempt.

However, I will venture a layman's metaphor which I hope will not be too misleading. The heart pumps blood around the body and if it stops doing so, we die. It is central to survival that the heart beats according to a certain rhythm and if that rhythm is disrupted, there may be problems. Various chemicals inside the body, including calcium, potassium and sodium, are vital to this process which depends on the coordination of electrical impulses.

Here, the most important chemical element in question is calcium. Hence, the initials CA in the name of the genetic variant in question here in particular. Now, in symphonic performances an orchestra relies on a conductor to keep the various instruments in harmonious combination. Without a conductor, the woodwind section, the brass section, and the strings may be out of tune resulting in musical disaster. For a symphony orchestra, coordination is everything.

So it is with the human heart - coordination is everything. Electrical impulses within the heart work in a sense like the conductor of an orchestra. A dysfunction of the rhythm and the workings of the heart can, and only too often does, produce a human disaster, sudden and unexpected death due to what we commonly call a heart attack. In such an event, the possible disruption of electrical impulses governing the heartbeat can cause death, and it is an issue arising from the CALM2 variation which is the focus of the Inquiry.

Kathleen Folbigg has always denied that she caused the death of any of her children. She asserted that position when interviewed by Police in 1999; she did so again when she pleaded not guilty at her trial in 2003; and she reiterated that position when she gave sworn evidence and was cross-examined at great length in the 2019 proceedings in this matter.

Partly from the 2019 proceedings, there's been an exposure of the question of the relevance of this genetic variation. There is now a great deal of relevant material in the science of genetics which was not available to the experts who testified in the 2019 Inquiry and certainly was not available at the trial in 2003.

The ultra rare genetic variant which affected the two deceased daughters, Sarah and Laura, was not even discovered until nearly a decade after the trial

and conviction of Kathleen Folbigg, and the fact that this variant actually affected the Folbigg females was only identified a few years ago. The relevant evidentiary landscape has widened dramatically.

It is a truism of good scientific method, as it is a good legal method, that if the evidence changes so the conclusions to be arrived at may correspondingly change. At this point, I will not refer to the expert reports in any detail and my learned friend has done so in enough detail to make us aware of where we are heading.

Each of the proposed witnesses has provided one or more reports, but we expect that it will disclose three broad scientific perspectives: firstly, the biochemical; secondly, the molecular; and thirdly, the clinical. Some of the reports at various points result in an answer by the expert to a particular question posed, "That is not my area of expertise." These three distinct perspectives will provide, we say, an overall genetic picture of the variant and its relationship with this case. One particularly significant witness in the Inquiry will be Professor Peter Schwartz, the director of a renowned Italian Institute for the study of Cardiac Arrhythmias of Genetic Origin and a curator of the worldwide Registry of Calmodulin Mutations. The Counsel Assisting has correctly said that we don't expect that there will be any of the expert witnesses who reaches the proposition that the CALM mutation in this case certainly caused the deaths of these infants. Nor do we anticipate that there will be any of the experts who will definitively deny it or reject it, but there is a range of possibilities between the two positions and we will be urging the proposition that, at the very least, the material to be exposed in this Inquiry will demonstrate that it is certainly a very reasonable possibility that cannot be properly excluded that the deaths of these two children were indeed caused by this.

JUDICIAL OFFICER: I have to consider that, of course, in light of all the evidence, including the diaries.

WOODS: Of course. We'll get to that. We'll be certainly making strong submissions on that point.

JUDICIAL OFFICER: I understand that.

WOODS: But not now, of course.

JUDICIAL OFFICER: I just want to make it clear, as I did earlier on, I can't confine my attention purely to the scientific evidence, important as it is, I should emphasise.

WOODS: I'll just conclude by citing the most recent addendum report dated 8 November presented by Professor Schwartz. He concludes his addendum by saying:

> "As this is not a mere scientific discussion among colleagues and science, but it is part of an inquiry on trial for multiple infanticide … I

wish by habit to be very clear. I have no idea whether Mrs Kathleen Folbigg has killed her children or not and, even though it is not a nice thing to say, to a certain extent, I don't really care. That is not for me to say. What is for me to say is what, in my opinion, are the legitimate conclusions based on science and on what we know as of today in relationship to the consequences of mutations on a calmodulin gene. On this basis, the deaths of Laura and Sarah Folbigg are fully compatible with their CALM mutation. There is not a single scientific element to prove the contrary. If an infant or a child dies suddenly, the post mortem is negative and a CALM mutation is found, the diagnosis would be sudden death due to calmodulin mutation, end of story."

Your Honour, we note that your Honour will be looking at all the evidence, and in that respect I'm grateful to hear that we now have, I think, the video evidence of the 1999 ERISP. That's helpful.

JUDICIAL OFFICER: I don't know if we've got the whole of it, have we, Ms Callan?

WOODS: Anyway, parts of it. Anything will be helpful, your Honour. Of course, we will be making further submissions about the diaries at a later point, and we recognise that your Honour will be looking at all the evidence, but this part is significant. We anticipate that, in due course, the evidence about the genetics will support the respectable submission that the diagnosis for both Sarah and Laura ought properly be sudden death due to a calmodulin mutation. Unless such a conclusion could be compellingly ruled out, legal principle dictates that there would inevitably be a reasonable doubt about all the convictions under consideration here. Your Honour, referring to the totality of the evidence, I'm pleased to see that the efforts by Counsel Assisting have produced the actual original diaries, and we would seek access to those within the confines of the Court or adjacent to the Court from time to time as we arrange. Thank you.

JUDICIAL OFFICER: Does anyone else want to say anything at this stage?

FRASER: Just something very briefly, if I may, your Honour, on behalf of the Ministry of Health. Given the matters to be traversed in this hearing block, it appears to the Ministry that there is little that we can offer the Inquiry by way of assistance by being present for the evidence in this hearing block. In those circumstances, we would propose to withdraw from this hearing block prior to the commencement of the evidence. We, of course, would remain available at any time to provide any assistance we can to the Inquiry and we would propose to remain engaged with the Inquiry and reassess our level of participation in next year's hearings when their shape becomes clearer.

JUDICIAL OFFICER: I'm perfectly content with that, Mr Fraser. Come and go as you will.

FRASER: Grateful, thank you.

JUDICIAL OFFICER: Anyone else? Do you wish to tender some documents?

CALLAN: Yes, your Honour. I propose to tender what is described as a tender bundle. It is listed in an index dated 14 November 2022. It adopts a numbering format which will permit further relevant documents to be added at convenient locations in due course if that need arises.

JUDICIAL OFFICER: I am happy for that, providing the subindices are kept up to date.

CALLAN: Yes, your Honour. Can I, for the record, hand up a copy of the index. Perhaps that could be marked for identification so it's clear what is to be tendered today. If your Honour might, as I said, consider marking the exhibits in the manner set out in the index.

MFI #1 INDEX HEADED "INQUIRY OF THE CONVICTIONS OF KATHLEEN MEGAN FOLBIGG PROPOSED TENDER BUNDLE AS AT 14/11/22"

You will see we propose the numberings start at number 5, which follows on from exhibits to date, which the last exhibit was Exhibit 4.

EXHIBIT #5 DOCUMENTS RELATING TO PROFESSORS VINUESA, COOK AND ARSOV INCLUDING SEPARATE REPORT OF PROFESSOR VINUESA AND JOINT REPORT OF PROFESSORS VINUESA, COOK AND ARSOV TENDERED, ADMITTED WITHOUT OBJECTION

EXHIBIT #6 DOCUMENTS RELATING TO PROFESSORS TOFT OVERGAARD AND NYEGAARD INCLUDING JOINT REPORT OF 18/10/22 TENDERED, ADMITTED WITHOUT OBJECTION

JUDICIAL OFFICER: In due course, I would anticipate their more recent report will be added.

CALLAN: Yes, your Honour. As mentioned in the opening, there was a report furnished on Saturday, and so that can be tendered and added as, for instance, Exhibit 6-03.

EXHIBIT #7 DOCUMENTS RELATING TO PROFESSORS KIRK AND SKINNER INCLUDING JOINT REPORT OF 31/10/22, ADDENDUM OF 09/11/22 AND TABLE OF ARTICLES REFERRED TO TENDERED, ADMITTED WITHOUT OBJECTION

EXHIBIT #8 DOCUMENTS RELATING TO PROFESSOR SCHWARTZ INCLUDING REPORT OF 24/10/22, ADDENDUM OF 08/11/22 AND TABLE OF ARTICLES REFERRED TO TENDERED, ADMITTED WITHOUT OBJECTION

EXHIBIT #9 DOCUMENTS RELATING TO PROFESSOR WILDE INCLUDING REPORT OF 24/10/22, ADDENDUM OF 09/11/22 AND TABLE OF ARTICLES REFERRED TO TENDERED, ADMITTED WITHOUT OBJECTION

EXHIBIT #10 DOCUMENTS RELATING TO ASSOCIATE PROFESSOR RAJU INCLUDING REPORT OF 05/11/22 INCLUDING ANNEXURES TENDERED, ADMITTED WITHOUT OBJECTION

EXHIBIT #11 DOCUMENTS RELATING TO PROFESSOR FLEMING INCLUDING REPORT OF 11/10/22, EXECUTIVE SUMMARY DATED 03/11/22 AND TABLE OF ARTICLES REFERRED TO TENDERED, ADMITTED WITHOUT OBJECTION

EXHIBIT #12 DOCUMENTS RELATING TO PROFESSOR RYAN INCLUDING REPORT OF 23/09/22 AND TABLE OF ARTICLES REFERRED TO TENDERED, ADMITTED WITHOUT OBJECTION

EXHIBIT #13 FORENSIC PATHOLOGISTS' FURTHER REPORTS INCLUDING REPORT OF PROFESSOR JOHAN DUFLOU OF 09/02/22, REPORT OF PROFESSOR CORDNER OF 01/03/22 AND AFFIDAVIT OF PROFESSOR HILTON DATED 26/07/22 TENDERED, ADMITTED WITHOUT OBJECTION

EXHIBIT #14 MISCELLANEOUS ADDITIONAL EVIDENCE COMPRISING REPORT OF PROFESSOR RAY HILL OF 07/04/15, REPORT OF KAREN HALL OF 28/02/19, DOCUMENTS PRODUCED BY SYDNEY CHILDREN'S HOSPITAL NETWORK AND JUSTICE HEALTH AND FORENSIC MENTAL HEALTH NETWORK, AFFIDAVIT OF BILLY JO BUCKLEY OF 19/10/22, ECG OF MS FOLBIGG OF 24/03/03 AND EMAIL FROM MR FOLBIGG OF 26/10/22 REGARDING HIS DNA TENDERED, ADMITTED WITHOUT OBJECTION

EXHIBIT #15 JOURNALS AND OTHER ARTICLES COMPRISING 116 JOURNAL ENTRIES OR ARTICLES TENDERED, ADMITTED WITHOUT OBJECTION

EXHIBIT #16 OTHER DOCUMENTS INCLUDING SUMMARIES OF CHRONOLOGIES TENDERED, ADMITTED WITHOUT OBJECTION

Your Honour, as foreshadowed, can I also tender a copy of the video of the police interview with Ms Folbigg which occurred on 23 July 1999. Your Honour, can I do so by reference to a volume of material tendered in the 2019 Inquiry which is, in these proceedings, Exhibit 2-AZ. If it's convenient, could I ask that your Honour turn that up? I wish to refer to your Honour to the index to explain this ERISP video. Your Honour, if you have exhibit 2-AZ there, immediately behind tab 2-AZ should be an index described as the Diaries Tender Bundle Index.

JUDICIAL OFFICER: Yes.

CALLAN: If your Honour turns of item number 15 that is described as the ERISP of the video recording between Ms Folbigg and Detective Senior Constable Ryan, and it lists time sequences and question numbers. Your Honour, it is a copy of that video which was not available and is now available,

that I tender on this USB.

JUDICIAL OFFICER: Do I simply then delete the expression "Not available" in the index?

CALLAN: Yes, your Honour.

WOODS: So that's Tab 17, is it?

CALLAN: 15.

JUDICIAL OFFICER: Exhibit 2-AZ, Dr Woods, has got an index to it.

WOODS: 2-AZ.

JUDICIAL OFFICER: If you go to the index, which is immediately after AZ, Exhibit 2-AZ, I'm sorry.

WOODS: Tab 15.

CALLAN: Your Honour, to be clear for the record what has just been, if I can put it this way, absorbed into the proceedings is the ERISP video recording which is to be located at exhibit 2-AZ Tab 15. Your Honour, that completes the tender for now. I note that Associate Professor Raju is appearing by AVL. If it's convenient, perhaps, your Honour, we could take a short adjournment and that can be set up.

JUDICIAL OFFICER: I understand Professor Raju is available between 12 and 1pm and between 5 and 6pm tonight; is that the position?

CALLAN: Yes, he may be available shortly before midday. But your Honour, I note the time and we had an early start, so I'm in your Honour's hands.

JUDICIAL OFFICER: What I propose to do is take a short adjournment. Then we'll hear from Professor Raju and then adjourn for lunch. And then come back at 2 o'clock to sort out future course of the proceedings.

CALLAN: Yes, your Honour.

SHORT ADJOURNMENT

AUDIO VISUAL LINK COMMENCED AT 12PM

<HARIHARAN RAJU, AFFIRMED(12.01PM)

JUDICIAL OFFICER: Thank you for giving evidence to the Inquiry, Professor. If there's any material you wish to look at during the course of your evidence, please feel free to do so.

WITNESS: Thank you.

<EXAMINATION BY MS CALLAN

Q. Sir, can you tell the Court your full name and in brief terms your qualification?
A. Sure. My name's Hariharan Raju. I'm a Clinical Cardiac Electrophysiologist with an interest in cardiogenetic disease. I'm a Practising Clinician and trained as a Cardiologist and Eltrophysiologist both here in Australia and in the UK with a PhD that involved significant components of molecular genetics in Sudden Unexplained Deaths.

Q. Where do you presently work?
A. At Macquarie University Hospital and at Concord Hospital here in Sydney.

Q. It's the case, isn't it, Professor Raju, that in February 2019 you were asked by Ms Kathleen Folbigg's legal representatives to assess her cardiac health.
A. That's correct.

Q. And this was sought in the context of there having been an identification of rare genetic variants in her DNA associated with cardiac arrythmia.
A. That's correct.

Q. And it's the case that in early 2019 you met with Ms Folbigg to take her history.
A. Correct.

Q. And in that respect, were you concerned to seek information from her which might be regarded as relevant to her cardiac presentation.
A. That's right.

Q. Now, I'll come to what you have indicated she told you during that first occasion when you met with her to take her history, but in general terms would it be fair to say that the history she gave you included describing a number of fainting episodes?
A. That's right, and that was the primary concern in the context of both her family history and the potential DNA variants that she carried.

Q. It's the case also, isn't it, that in April of 2019 you performed a number of cardiac investigations of Ms Folbigg.
A. So just one investigation. That was a stress ECG.

Q. I'll come to the detail of that in a moment but in the overall picture that you have come to develop in relation to Ms Folbigg's cardiac presentation, you

have had access to reports or results of other cardiac testing, haven't you?
A. That's right. So they have been historical ECG and echocardiogram which is a cardiac ultrasound that I have seen.

Q. Now, you provided a report in April 2019 in a previous Inquiry that was conducted into Ms Folbigg's conviction.
A. That's right.

CALLAN: Your Honour, for the record that report is located at Exhibit 2-BL in these proceedings.

Q. This year it's the case, isn't it, that you were asked to prepare a report setting out Ms Folbigg's complete cardiac history to the extent it is known, and you were asked to provide your opinion as to what that history indicates, and whether Ms Folbigg has any cardiac condition.
A. That's right.

Q. Now, you provided this Inquiry with a report dated 5 November 2022. Do you have a copy of that report with you there, sir?
A. I do. I will bring that up.

CALLAN: Your Honour, I note that that report has been marked Exhibit 10-02.

JUDICIAL OFFICER: And behind the same bundle are Professor Raju's earlier reports.

CALLAN: Yes.

WITNESS: Yes.

CALLAN

Q. Now, Professor Raju, your report of 5 November 2022 does it, amongst other things, capture information and views that you express in the earlier reports that you furnished in February and April 2019?
A. That's right. So I have not consulted with Ms Folbigg since 2019 and so all of the information is very much based on previous interpretation of data.

Q. Now, in your report of 5 November 2019, at the end of that report, which your Honour is red pagination 13, you express the view in the final paragraph of your report that:

> "Overall, on the basis of current available data, I believe Ms Folbigg has either no evidence of any significant cardio genetic disease or less likely so-called Forme Fruste Disease or a true idiopathic ventricular fibrillation phenotype. The latter two phenomena are acknowledged as demonstrating pre-disposition to arrythmia in the absence of manifest abnormalities on testing outside times of transient arrythmias."

Can I ask you, first, do you maintain the view expressed in that final paragraph?
A. So overall I still feel that it's - she doesn't have sufficient data to confirm a diagnosis. She has some minor abnormalities that in this context are difficult to say with confidence whether they relate to any of the phenotypes that might be associated with the DNA variant that she carries. So in principle, yes, that's my overall feeling.

JUDICIAL OFFICER

Q. Professor, could I reveal my ignorance. What's a Forme Fruste Disease?
A. Forme Fruste is where the appearance of a disease is so mild that it just isn't apparent when you do a basic set of tests.

CALLAN

Q. Is it the case that what you described as an Idiopathic Ventricular Fibrillation phenotype is not observable before it occurs?
A. That's right. So, idiopathic is a doctor's term for "otherwise associated with a completely normal heart," and so you transition from completely normal to life-threatening dangerous arrhythmia with nothing apparent beforehand.

Q. Does what you're saying there when you refer to a Forme Fruste Disease or a true Idiopathic Ventricular Fibrillation phenotype, perhaps in lay terms, mean if Ms Folbigg is affected by a cardiac condition, it's in a way that shows no evidence on testing?
A. Or shows very minimal changes on testing, insufficient to make a definitive diagnosis.

Q. In your report, you expressed the view that Ms Folbigg's history of losing consciousness or fainting is more consistent with neurocardiogenic syncope, also called vasovagal or reflex syncope, than arrhythmic aetiology. Can I ask you to assist this Inquiry first with an understanding of what a neurocardiogenic syncope refers to?
A. So, syncope is a term for transient loss of consciousness related to inadequate blood supply to the brain, and so that essentially means that it is caused by either heart or blood pressure issues. So, that's the first thing. So, reflex syncope - so, reflex is a thing that happens when a doctor taps your knee and your leg flies out and you have no control over it. It's something that humans and animals have been left with following evolution for various protective mechanisms. We have reflexes that are unconscious responses from our brain that control heart rate and blood pressure, and so those reflexes, for reasons that nobody fully understands, can be activated to lower your heart rate and/or lower your blood pressure to - which can then result in a transient loss of consciousness. So, that is reflex syncope and cardiogenic and - sorry, neurocardiogenic is a subdivision under that umbrella term of reflex syncope and vasovagal or vasodepressor syncope is a subcategory under that. And, so, they are all related to pathology that is in the reflexes rather than true cardiac pathology. And the important thing here is that it's the commonest cause of syncope across the board. So - in fact, depending on

who you ask, about one in three people will have a reflex syncopal event in their lifetime. Whereas, in comparison, obviously, life-threatening arrhythmias which can be a consequence of the kind of cardiogenetic disease that is being proposed in Ms Folbigg is completely different and pathological. So, reflex syncope is benign in the sense that it can cause blackouts and injury but it doesn't cause sudden death.

Q. Could you assist the Inquiry by the differences, if they may be, between a reflex syncope or perhaps more focused on, as you describe it, a neurocardiogenic syncope, as compared to a loss of consciousness caused by a disordered heart rhythm in terms of not only the cause but the manner of onset and other symptoms?
A. Sure. So, arrhythmic syncope in general has unpredictable and sudden onset. So, you go from being completely well to being unconscious, literally, like, from one beat to the next. And when it recovers, assuming that it does and it - obviously, the potential outcomes at that point are either sudden death, cardiac arrest or recovery, but when you recover, if you haven't had a cardiac arrest, the recovery is generally instantaneous because your blood - heart rate and blood pressure returns from completely disordered to completely normal. And that contrasts with the majority of neurocardiogenic events which often have a prodrome; so, things like pallor, dizziness beforehand, some autonomic features - so, kind of, nausea and vomiting - which often exist before and also afterwards alongside, usually, a period of minutes or hours of lethargy and fatigue. And those are all associated with, kind of, reflex or autonomic reflex activation.

Q. In your report of 5 November 2022, starting at the bottom of the first page, you describe Ms Folbigg as having a long-standing history of recurrent transient loss of consciousness that started in childhood. Could you assist us in relation to that history as you've described in your report?
A. Sure. So, I guess the first thing to say is that multiple recurrent syncopal episodes, although concerning for the individual involved, usually points away from a life-threatening pathology. And, I guess, the simple explanation for that is that if you had a predisposition to life-threatening arrhythmias, at some point one of those episodes is likely to result in cardiac arrest. And so, that's the first thing. The more episodes you have without cardiac arrest, the less likely it is that that is predisposed to cardiac arrest. I guess, I'll just talk through the reports as I've stated in my report. So, she gave me some specific examples, often post-exertion rather than in the middle - mid-stride or in the middle of peak exertion. That's important because both mid-exertion and post-exertion can be related to arrhythmic syncope, but post-exertion certainly is commonly seen in individuals who do a lot of exercise and is a consequence - or can be a consequence of reflex syncope. So, one of the red flag symptoms that we worry about in this context is exertional syncope, but on the basis of the reports that she gave me, it appeared that all of the events that she had - that she was able to describe in any detail sounded like they occurred following completion of a period of exercise rather than in mid-stride. So, you can--

Q. In - sorry, sir. You go on?
A. Sorry. And so, there's the report that I kind of describe here, saying at

school or as a teenager, running a sprint relay, she took 30 minutes to recover with lethargy thereafter. So, again, that points more towards a reflex aetiology rather than an arrhythmic episode. And then, again, following emotional stress, having a similar response with a prolonged recovery period. The one event that, I guess, I would be slightly more concerned about is the episode in the swimming pool that has been described also in, as I mention, an affidavit of a friend, which – Billy-Jo Buckley - which occurred in a swimming pool, which is one of the - again, one of the red flag events for, particularly, the condition CPVT and one of the subtypes of Long QT syndrome which, well, I'm sure we'll come on to discuss later. Having said that, there's not enough information about that event to clearly differentiate whether that might've been arrhythmic or not. The one thing that Ms Folbigg was able to tell me was that, again, this occurred after she had completed the swimming race which, again, is less indicative of an arrhythmic, although it's still somewhat concerning.

Q. As I understand your report, the event which occurred in the swimming pool was an event that Ms Folbigg described to you during your second consultation with her on 18 April 2019, and whilst she told you she was around the age of 12, you proceed on the basis that that is the same event that is described in an affidavit of Ms Buckley of October 2022, who suggests that it occurred at the swimming pool in year 9, so about age 15?
A. Yes, and that's obviously an assumption on my part. It is conceivable that there were completely independent events but they sound similar.

Q. That suggests an event which occurred in the early 1980s. Noting the passage of time until it's come to be described and considered by you, what information about that swimming event have you placed weight on? I think you've described a red flag about the fact that it was in association with swimming as a starting point. Is there something about swimming and the point in time the event occurred that has informed your opinion about what is known?
A. Yes. So, the red flag points towards potential arrhythmic cause because swimming is a particular association with CPVT, or Catecholaminergic Polymorphic Ventricular Tachycardia, which is one of the potential cardiogenetic disorders that her DNA variant can predispose you to. It is also associated with the commonest subtype of the Long QT syndrome, which is again another phenotype that is associated with her DNA variant. And so, that's why that particular event, ideally, would be interrogated in more detail, but, unfortunately, the amount of detail available to me in terms of how it occurred was fairly limited. The one thing that I have interpreted is that Ms Folbigg herself explains that it occurred having completed the race, and in the context of her other episodes, which sound very much like neurocardiogenic aetiology, I have made the assumption that she has a single pathology that explains all of these episodes rather than that she has two separate pathologies occurring side by side, although, obviously, either is potentially possible.

Q. Was there anything about the way that Ms Folbigg described recovery from that syncopal event at the swimming pool which informed your view or assumption about it?

A. Unfortunately, her recollection of her recovery of that - following that particular event was somewhat limited, and so I don't know exactly whether that was prolonged. But of course, if you lose consciousness in a swimming pool, your recovery can be more prolonged, even if the original precipitant was arrhythmic, so that's probably not such a helpful discriminator in this particular situation.

Q. Your report also details Ms Folbigg's description of other episodes of loss of consciousness in adulthood. I understand from your report, she gave you that history in your first consultation with her on 20 February 2019, effectively describing these events from fainting when her verdict was delivered in court and, thereafter, events which have occurred during her time in custody?
A. That's right.

Q. You express a view about those events, is this the case, based not only on her history to you but also available Justice Health documents and notes which were made contemporaneously?
A. So that - that's right. So I've put her history in the context of other descriptions and overall my impression is that every episode that I was - that she was able to describe sounds more likely to be neurocardiogenic aetiology, rather than arrhythmic.

Q. A document described as a Chronology of Cardiac Events, which was prepared by those assisting in the Inquiry, drew upon every piece of information in this Inquiry which references or suggests fainting or some other type of potential cardiac event involving Ms Folbigg was provided to you, wasn't it, Professor Raju?
A. It was, yes.

CALLAN: Your Honour, I note for the record that that document is being marked Exhibit 16-02 in this Inquiry.

Q. You state towards the bottom of page 2 of your report, "That the chronology of cardiac events describes multiple symptomatic events, though none are described in sufficient detail to help differentiate the likely mechanism or etiology." Does that insufficient detail inhibit you from expressing a view one way or another about what that information reveals?
A. That's right. And I think it's important to say that in matters in general the history of the event will often contribute 70 to 80% at least of your differential diagnosis and the - yeah, the detail in all of those is just insufficient when you're weighing up potential causes of transient loss of consciousness.

Q. I want to ask you next about the medical investigations which have been conducted in respect of Ms Folbigg from a cardiac perspective, both those that generated reports you reviewed, but also the test you conducted in April of 2019. Before doing so it would be of considerable assistance to the Inquiry if we could have your contribution with some, I might describe it as basic explanation of what you're looking for, for instance in an ECG and the types of calmodulinopathy conditions that have been given specific consideration in this Inquiry.

CALLAN: I'm going to ask to have displayed on a screen a slide that, your Honour, was used in my opening address.

Q. Sir, I understand that this diagram--

JUDICIAL OFFICER: I don't know if it's been displayed?

CALLAN: Sorry.

Q. Can I ask if you're able to see the display, Associate Professor Raju?
A. I can.

JUDICIAL OFFICER: So can I now.

CALLAN

Q. I understand the schematic presents what might be described as a generic normal sinus rhythm for a human heart as it would be recorded by an electrocardiogram?
A. Yes.

Q. Could you explain each of the relevant conditions which are listed, that is, Long QT syndrome, Catecholaminergic Polymorphic Ventricular Tachycardia and Idiopathic Ventricular Fibrillation?
A. Sure. So calmodulinopathy, so essentially means a disease of calmodulin can manifest in humans as one of three phenotypes as we call it. So phenotype is how a genetic disorder manifests itself. And that can be anyone of those three conditions that you have just listed, as seen on the slide. The Long QT syndrome, as the name suggests, is characterised by a longer than normal QT interval, which is the arrow, the blue arrow in the bottom right. And so individuals affected by the Long QT syndrome tend to have a prolongation of that in comparison to normal. Having said that, that's not a universal presence and we do know that a proportion, perhaps one in three individuals affected by the genetic risk for Long QT syndrome will have a QT interval within the normal spectrum. And the reason for that is that humans have, in the same way that humans have a variation in heights, they have a variation in QT interval which gives a spectrum of normal that overlaps with pathology. The second--

Q. Sorry, I might interrupt you there. If we can flip to the next slide which I think should include by way of example, what appears on an electrocardiogram for Long QT syndrome. If we start with the top left quadrant of the slide.
A. Yep. So as you explained the QT interval or the distance of time between the onset of the sharp peak to the end of the T-wave thereafter, just eyeballing that you can see that it is in excess of around - it goes to beyond halfway between the two sharp peaks, which although that's not a measurement is kind of a ballpark estimate that shows that that is prolonged. And so that is an example of a prolonged QT interval in an individual at rest. And alongside that you can see the rarity of that condition and the so-called congenital Long QT

syndrome is a genetic type of Long QT syndrome.

JUDICIAL OFFICER: Ms Callan, was that in the slides you had at opening?

CALLAN: I'm sorry, I missed the question, your Honour?

JUDICIAL OFFICER: The slide you're just showing Associate Professor Raju now, I don't think that was one of the ones that you referred to at opening, unless I was missing something.

CALLAN: No. This is a slide which has been specifically prepared to assist in this witness' evidence.

JUDICIAL OFFICER: Yes, I just wanted to be clear on that.

CALLAN: Yes.

Q. Can we go back to the last slide. The next condition of calmodulinopathy, if you could assist with, is Catecholaminergic Polymorphic Ventricular Tachycardia. Could you describe in brief, if possible, features of that condition and how it may present on an ECG?
A. Sure. So unlike the Long QT syndrome which has an abnormality in the QT interval and a variety of different but specific triggers depending on the subtype, CPVT is associated with an otherwise normal resting ECG and abnormalities are only apparent during a catecholaminergic surge. So that's essentially an adrenalin surge. And so the diagnosis of CPVT often requires either some kind of exercise provocation or a direct injection with adrenalin in order to provoke the abnormality.

Q. Moving to the next slide which includes these examples from ECG that you've provided, there's an ECG in the middle at the bottom of that slide which is described there.
A. So if you could just push back once more on the side if that's possible? But it is the - yeah, so this at the bottom left is the classical appearance of CPVT, which is again an exceptionally rare cardiogenetic disorder, which on the left during exercise shows what is the kind of characteristic by-directional VT where you have changing of the QRS kind of going downwards, upwards and alternating, until it then degenerates towards the right-hand side into Polymorphic Ventricular Tachycardia and thereafter Ventricular Fibrillation.

Q. Can I then ask you about Idiopathic Ventricular Fibrillation?
A. So, Idiopathic Ventricular Fibrillation is essentially it's the kind of the leftover diagnosis for individuals who are predisposed to ventricular fibrillation, which is essentially the most severe life-threatening dangerous heart rhythm. It essentially equates to cardiac death, without any apparent underlying heart disease, despite comprehensive testing. And so part of the reason why I haven't made prevalence for it here is that the prevalence has changed over the last years and decades as we have learnt more about different heart conditions that predispose you to life-threatening abnormal.

CALLAN: It's just frozen.

JUDICIAL OFFICER: The machine is having Idiopathic Ventricular Fibrillation.

CALLAN: Your Honour, I understand the witness is just dialling back in.

WITNESS: Sorry, have you got me again, I lost you for a while?

CALLAN: Yes.

JUDICIAL OFFICER: Yes, thank you, Professor.

CALLAN

Q. We're still on this slide which includes some examples of ECG. Does Idiopathic Ventricular Fibrillation indicate itself on an electrocardiogram?
A. No. So Idiopathic Ventricular Fibrillation means that not only is the ECG normal at rest but there is no apparent heart disease at all until a sudden and unexpected life-threatening event such - well, essentially ventricular fibrillation, which equates to cardiac death. And so that's - it's a - it's what you're left with as a diagnosis after somebody has ventricular fibrillation and you have ruled out or to the best of you ability ruled out all of the underlying cardiac diseases that can predispose to that.

Q. Is it the case, Associate Professor Raju, that there may be a cause or causes of these cardiac conditions? For instance Long QT syndrome or CPVT which is other than having been produced by CALM variants?
A. Absolutely. And in fact CALM or calmodulin variants are relatively recently described and are exceptionally rare conditions, so much so that as someone with an interest in cardiogenetic disease, Ms Folbigg is the first individual that I have personally been involved with who has a CALM variant where there is a suspicion that this may be causing disease.

Q. You indicated that these three conditions might be the product of or described as a calmodulinopathy, are there any other cardiac conditions which have been recognised as being associated with CALM variants?
A. I'm not aware of any. As I said, it's not something I have a specific expertise in because of the relatively recent description of them.

Q. Can I ask you still on the topic of cardiac conditions, about Brugada Syndrome. We see an example ECG associated with that syndrome on the right side of the slide. What is that syndrome, Professor?
A. So that is another cardio genetic primary arrythmia syndrome. That means that your heart muscle is normal but it has a stereotypical ECG or electrocardiographic appearance which is kind of right in the middle of that ECG whereby you have so-called J-point elevation after the kind of the sharp QRS deflection and a negative T-wave thereafter with a coved appearance or a straight line between the end of the QRS to the inverted T-wave. That condition is yet another cardio genetic disease that predisposes you to sudden unexplained death which, with an uncertain prevalence, tends to be more

severe in male individuals and tends to affect individuals predominantly in their 30s and 40s, and is exceptionally rare in children or exceptionally rare to manifest in children.

Q. Was Brugada Syndrome one of the cardiac conditions that you investigated as part of your assessment of Ms Folbigg in 2019?
A. It was one of the potential underlying causes, so you have to be aware that when I consulted with her, although the DNA variant had been identified, it wasn't clear at that point that it was responsible for any disease at all, and so I essentially kept an open mind in the context of her family history as to what might be the underlying condition that ties together all of the tragic events in her family.

Q. When you describe her family history, what are you referring to?
A. The deaths of her four children.

Q. Can I take you now to the reports of cardiac examinations which have been conducted in relation to Ms Folbigg. There was in March 2003 an ECG which I think you reviewed in preparation for your 2019 report.

CALLAN: Your Honour, it's contained in Exhibit 14-06 at p 228. That's red p 228, your Honour.

JUDICIAL OFFICER: Thank you.

CALLAN

Q. This is referred to towards the bottom of the third page of your report, Associate Professor Raju. You noted a borderline QT was evident. Can you explain the significance of this?
A. So as we've been discussing, the Long QT Syndrome is one of the potential manifestations of a Calmodulinopathy, and this particular ECG struck me as being not diagnostically abnormal but of sufficient unusualness that it was worth making specific comment on. And so a QT interval is generally considered abnormal in females if the correct QTc is 470 which this didn't but it was close enough given the overlap between pathology and normal that maybe this was of relevance.

Q. You also refer to notched inferior T-waves and small U-waves. Can you explain the relevance of that?
A. So, notching of T-waves is one of the alternative manifestations of ECG abnormality seen in specific sub-types of the Long QT Syndrome which can be present even in the absence of QT prolongation and so that again struck me as being worthy of comment.

Q. You then in your report go on to refer to a subsequent ECG dated 24 December 2018 which you describe as entirely normal.
A. That's right and we know that individuals with these conditions can have intermittent or variable expression of their disease and so one of the things that in clinical practice that we will use is sequential repeated tests to look for

evidence of a condition, and so the presence of an abnormality on one day with a completely normal test thereafter doesn't tell you whether they have the disease or not, and usually you would in order to, with some confidence, say that there was expression of a disease you would want to see at least two separate ECGs that show diagnostic abnormalities unless the abnormalities are so gross that it can't possibly be within the spectrum of normal.

Q. I should note in terms of dealing with this material chronologically, there was an ECG of Kathleen Folbigg dated 17 May 2011. That was not included in the materials with which you were briefed and so it's not referred to in your report. Your Honour, a copy of it is at Exhibit 2-AH. I understand, Professor Raju, you have since been provided--

JUDICIAL OFFICER: Exhibit 2-AH?

CALLAN: 2-AH.

Q. That you have since been provided with a copy of that ECG.
A. That's right.

Q. And did you notice anything of significance in that ECG?
A. No. This - this looks unremarkable.

Q. So you would describe the ECG of 17 May 2011 as unremarkable or normal?
A. Normal.

Q. Okay. You describe the ECG of December 2018 as normal. You go on to note a normal echocardiogram in February 2019, and a further ECG dated February 2021, and your Honour, for the record, that ECG is contained in the material tendered this morning, Exhibit 14-04 red p 57. Now, as you recognise, Associate Professor Raju, you personally supervised a postural ECG and exercise provocation on 18 April 2019. Your Honour, the report of that is contained at Exhibit 2-BH. Could you first assist us, sir, with what was involved in that postural ECG and exercise provocation test you administered in April 2019?
A. Sure. So that's an ECG, the same as we've shown examples of so far, initially starting with Ms Folbigg supine and then with a continuous recording as she stands up. Thereafter, with a standard Bruce protocol which is a defined staged exercise program on a treadmill with increasing incline and increasing speed of the treadmill at three minute intervals until - essentially until you get - until the individual is unable to continue or you achieve over 100% of their maximal age predicted rate, and continued recordings for at least four minutes into recovery thereafter. I did also after the end of that exercise provocation do modified high precordial ECG traces to look for manifest evidence of the Brugada Syndrome which is one of the other conditions we were looking for.

Q. And just following through with that, what was indicated in that specific traces that you undertook looking for Brugada Syndrome?

A. So nothing of concern as in there's no evidence of the manifest Brugada Syndrome. Having said that, that there is, as with all of these conditions, the potential for a concealed phenotype which can only be unmasked by drug provocation testing which we did not undertake.

Q. Now, during what you've described as the Bruce protocol treadmill exercise provocation test, you report that "Ms Folbigg had moderate hypertensive response during exercise followed by a precipitous drop immediately post exercise". Can you explain what that means?
A. So it's a normal human physiology for your blood pressure to increase as you increase, and what - you know, what should be your peak blood pressure is kind of unclear but certainly it shouldn't go above the systolic or the first number in your blood pressure should not go above 220. Her blood pressure peaked at 200 which is unusual in my experience as in most people peak at somewhere between 160 and 180, and I note that she did tell me in her prior history that she had previously been diagnosed with hypertension but some weight loss had been sufficient to resolve that. Having said that, the most important thing is that when you stop exercising, your blood pressure should fall but it should - generally, most people it falls slowly. In her it fell immediately back to near baseline which is a finding we see in lots of highly trained individuals who do a lot of exercise and tends to associate with post exertional neurocardiogenic syncope.

Q. Would it also be consistent with an arrhythmogenic cause.
A. No, because an arrhythmogenic cause would associate with an arrhythmia at the time of a reduction in your cardiac output, so that was not - that was not seen.

Q. You also describe isolated ectopics of LVOT origin. What does that mean?
A. So, in - so after her exercise is completed, she had isolated ventricular ectopics, so the ventricles are your main heart pumping chamber, and ectopics are - singular isolated ectopics are a normal phenomenon in everyone, and the LVOT stands for left ventricular outflow tract, and although I can't say that without absolute confidence that that's where they were from, it certainly resembled an ECG appearance where it's likely to be from one or other of left or right ventricular outflow tract. The reason that's important is that the outflow tracts are the commonest site for idiopathic ectopy although you can also see them in a number of cardiogenetic disorders.

Q. You go on to refer to observing "non-specific upsloping ST depression at peak exertion of maximal 0.01 NV". What does that mean?
A. So, that's the - so the ST segment is the - is the component of the ECG just after the sharp deflection of the QRS, and ST depression is almost universally associated with some form of cardiac pathology but not in - not when seen during exercise and not when upsloping, so that's a description of I guess a normal variant change in your repolarisation at - with exercise.

Q. Associate Professor, since completing this report, I understand you've, amongst other things, been provided with a report of Professor Wilde, dated 24 October 2022, where he describes the exercise test results as consistent

with CPVT. Can I ask whether you agree with that?
A. Sure. So, one of the - kind of, the major differentiators is the presence of not just isolated ventricular ectopy but couplets, which is two ectopics, one after the other - or triplets - and when I undertook this test, I felt that the two episodes where it looked like there was a couplet and a triplet were probably artefactual. So, artefact is something that is non-physiological; ie not related to an abnormality in the heart at all. Arthur Wilde has reported on these as pathological, and I've - I have gone back to revisit it, and I have to say that it's very difficult to say with any confidence as to whether those specific things are real - ie heart rhythm abnormality - or artefact. If you work on the basis that they are genuine, then it's certainly consistent with CPVT because, although isolated ectopy is, you know, non-specific, anything more than that is unusual in the absence of some kind of underlying heart disease. And so, I would say that if those are genuine couplets - if it's a genuine couplet and triplet, then this is certainly consistent and, I would say, suggestive of CPVT. Whereas, if they are artefactual, then it's still consistent with CPVT but certainly not diagnostic of CPVT.

Q. How is it that an artefact can present in a test such as this on an ECG?
A. So, ECGs are electrical recordings taken from, essentially, the skin outside the body but focused on electrical activity within the heart. The difficulty is you have multiple things between the electrode on the outside of your body and the heart muscle. And so, electrical activity in your, kind of, muscles in your arms and legs and on your chest wall can give you artefact, but also movement of the electrode between - so, friction between the skin and the electrode can also cause artefact. And so, it can sometimes be difficult to tell the difference between those two phenomena.

Q. How do you go about distinguishing between artefact and what is genuinely going on with the electrics of the heart?
A. So, one of the things is whether you see clearly obvious artefactual things around the same time. So, obvious artefact is vertical - completely vertical abnormalities on the ECG, because there is - on an ECG, you have electrical potentials against time and there is no biological process that can realistically cause electrical changes that are instantaneous. So, completely vertical deflections of your ECG cannot realistically be biological or physiological. And so, there certainly were multiple, definitely artefactual events on the tracing, but these two - kind of, the couplet and the triplet that we are particularly concerned about - do not fall into that category. And then the next thing is the response that you see as a consequence of that event. I mention that because, if you have an ectopic - depending on where it occurs within the cardiac cycle - it usually, or it can have some kind of compensatory pause thereafter, and so you see a change in the normal heartbeat that comes immediately after the abnormal beat. And then finally, if it's physiological, the appearance of the ECG QRS Complex - so, that's the big sharp deflection - should be consistent with it coming from somewhere within the heart. So, you asked me about the ectopics of LVOT origin. So, we can look at ECG traces which have not just a single vector or direction but 12 separate leads, and we kind of look at that. Each of those leads gives us information in a single direction, and we kind of compile that in our brains to try and work out

what the three-dimensional electrical activation of the heart is. And if it's a physiological signal, then it should be consistent with coming from somewhere within the heart, whereas artefactual abnormalities can essentially look very, very unusual or they can look unusual. And part of the reason why I thought it was artefactual is that there is an inconsistency between two of the leads which I felt meant that it was highly unlikely to be physiological. But, as I said, unfortunately, none of these things are absolute.

Q. I understand you've also been provided a copy of Professor Wilde's supplementary report of 9 November 2022. Relevantly, he notes that, having printed out the exercise ECG and measuring the QT interval himself, he considers it becomes slightly prolonged after three minutes of exercise and that this is particular to long QT syndrome type 1. Do you agree or have a view about that observation by Professor Wilde?
A. So, I have the greatest of respect for Professor Wilde. I've - having seen that, have gone back and revisited the ECG. I have to say that I don't think it prolongs to that level, but measuring the QT interval on an exercise test can be difficult when each one of the little squares constitutes 40 milliseconds, and we're looking for differences of under 10 milliseconds. And so, yeah, I - there are - the QT interval is, I guess, in a grey area.

Q. You also indicate in your report that on 18 April 2019, Ms Folbigg was fitted with a 24-hour halter monitor, and that was, as I understand it, for the purposes of undertaking 24 hours of recording of her heart rhythm. Why is that undertaken, Associate Professor Raju?
A. So, that's essentially another mechanism to look for intermittent abnormal heart rhythms. So, particularly when you're looking for evidence of CPVT or Long QT syndrome, which can provoke arrhythmias during exercise, amongst other triggers, it's sometimes useful not just to do a treadmill but also a halter monitor. And if there had have been couplets or triplets on that, then that would've caused me to revisit the abnormality that I had attributed to artefact on her treadmill, but that was - nothing like that was seen. I should mention that, unfortunately, she was put on bed rest for the entirety of that 24-hour monitoring period so it's not such a useful investigation. The point of doing an ambulatory monitor, as the term suggests, is that you ambulate, but I think - that was something I had no control over.

Q. You described the results of the halter monitor as normal. You also noted it showed very rare, late-coupled ventricular ectopy and very rare atrial ectopy. Can you assist us with what that means?
A. Sure. So, the ectopy is premature beats that are not from your normal biological pacemaker site, and, in essence, this level of - or this number of ectopics is completely normal in pretty much anybody, actually, and so I wouldn't put any weight on that on its own.

CALLAN: I'm conscious of the time, Associate Professor Raju. We've hit 1:00pm and I gather that you have another professional commitment. I understand we're proposing to resume with your evidence at 5:00pm today, if that's satisfactory?

WITNESS: It is. If you would like to continue for another 15 or 20 minutes, I'm happy to carry on.

JUDICIAL OFFICER: I think we should, if it's not inconvenient to anybody. Thank you, Professor; that's helpful.

CALLAN: Yes, thank you, Professor, we will.

Q. In terms of, as I said, just dealing with the chronology of cardiac investigation, after the exercise test and halter monitor that you were involved in, in April 2019, there was an ECG in February 2021. You describe that as "showing sinus bradycardia of rate 55 beats per minute, likely physiological but within normal limits." Can you explain what the significance of the sinus bradycardia rate is?
A. So, sinus bradycardia - so, sinus is your normal, biological pacemaker. That's where - that's a normal heartbeat. We, to some extent arbitrarily, define a normal heart rate between 60 and 100 beats per minute. So, anything less than 60 is bradycardia. Bradycardia just means low heart rate, but it's - we see this a lot in trained individuals, and so again it's not indicative of any kind of pathology, in my mind.

Q. What do you by trained individuals?
A. So, people who do exercise.

Q. What is it about people who do exercise that might mean that their resting heart rate is lower than the average?
A. So, that's a controversial topic even amongst cardiologists. There are two primary hypotheses, but there's the first, which is the vagus nerve, which is where vasovagal term comes from. But there's a suggestion that, as you train, your vagus nerve becomes more predominant and that causes your heart rate to lower in the same way that it does in neurocardiogenic syncope but as a persistent thing. There's also increasing evidence to suggest that, as you train, you have a change in your expression of iron channels, which are electrical currents that determine your underlying heart rate.

Q. Having regard to your own work in taking Ms Folbigg's cardiac history and the examinations that you've undertaken and had access to in terms of her cardiac presentation, along with the view, for instance, expressed by Professor Wilde, and, as I understand it, you've also been provided a copy of the reports of Professor Schwartz, what is your conclusion, sitting here today, as to whether Ms Folbigg presents with any of the calmodulinopathy phenotypes?
A. Sure. So, I should clarify, I haven't been shown Professor Schwartz's report. I have seen Professor Wilde's report. Unfortunately - so, in the absence of her genetic risk, I would say that her tests are not completely normal but they're not of sufficient abnormality to be diagnostic of any of the phenotypes associated. Having said that, when you put the minor abnormalities that you see in an ECG from two decades ago and some - on the treadmill, it is certainly plausible that she has a very mild phenotype that either Long QT syndrome or CPVT, but I'm not persuaded that that's the case. But it's certainly plausible.

Q. You use the term "Absent genetic risk", what did you mean?
A. So, obviously when we do tests in medicine we start with what we call a pre-test probability that someone has a disease. We then do a test and then that moves your kind of barometer of likelihood of them having the disease upwards of downwards. And so if you start with a she carries a genetic risk and this genetic or DNA variant is likely or you know, more than 50-50 likely to be associated with a disease, then the interpretation of those tests is slightly different than if she didn't have that. And so I guess I should clarify that the time when I did her testing it wasn't clear that the calmodulin DNA variant carried any risk at all. As in that was still, certainly at the time to me was not available, I wasn't aware of the functional data until sometime thereafter. Now, if from a personal point of view, if I integrate the genetic data with the minor abnormalities on her ECG it certainly fits, and so I would agree with Professor Wilde's assessment that this is consistent with CPVT. But in and of itself her cardiac data does not support a diagnosis of CPVT.

Q. Are you able to express any view as to whether you can exclude Ms Folbigg as a Brugada syndrome phenotype?
A. Because no drug provocation testing was undertaken I can't say that with confidence. But I think that's probably also less likely.

Q. In that respect in your report in the penultimate paragraph suggest, "In light of the family history and DNA mutation analysis", which I understand is that a reference to the CALM2 variant?
A. That's right.

Q. You say, "It would be prudent for Ms Folbigg to have further evaluation as previously recommended to complete her cardiac phenotyping."
A. That's right.

Q. You go on to describe what that evaluation might include. You speak of a "Provocation ECG testing with sprint exercise protocol and epinephrine infusion." What is that?
A. So we've already talked about the Bruce Protocol Treadmill exercise ECG. So the sprint protocol, rather than three minute stages with progressive escalation, goes from a standing start to maximal exertion almost immediately. The reason that that can be helpful, particularly in the diagnosis of CPVT, is that it is the sudden adrenergic burst that tends to provoke abnormalities. And so that can be helpful, (a) from that point in time, but (b) also because it gives us the opportunity to repeat an exercise provocation test, given the uncertainty about whether they were couplets or triplets or just artefactual abnormalities. The epinephrine infusion, epinephrine is essentially - well, it's now the internationally recognised, albeit originally American term, for adrenaline. So it is literally an injection of adrenaline with continuous ECG recordings, which is now recognised as probably slightly less useful than exercise or sprint provocation. But it may be helpful. And I guess particular in individuals where it's unclear what the underlying phenotype is, a lot of the diagnosis comes from repeating things and unfortunately we haven't really had the opportunity to do that.

Q. You go on to suggest that “The most important contributor to establishing whether her episodes of transient loss of consciousness are truly related to arrythmias is to achieve symptom rhythm correlation, ie document her heart rhythm at the time of an episode of unconsciousness or near unconsciousness." You then go on to describe that "This could be achieved by prolonged heart rhythm monitoring such as an implantable loop recorder." How does an implantable loop recorder work?
A. So we talked about the Holter monitor which conventionally is worn like and ECG with dots on your chest for 24 hours. Unfortunately, as I said, that was undertaken while she was on bed rest. Ideally you would have that kind of level of heart rhythm recording for weeks, months, years. Obviously the logistics and the convenience of doing that are just unrealistic. And so an implantable loop recorder, as the term "implantable" suggests, is a heart rhythm recording device that you implant within the chest wall just under the skin in front of the heart that, depending on which device you have, has a battery life of between three, four, four and a half years, and works somewhat like CCTV where it's continuously recording your heart rhythm and then if the device, the computer in the device detects something it sees as potentially abnormal, it will then snapshot that for you to have a look at. Or you also get a remote control with it, which when you lose consciousness you then regain consciousness and you press the button on the device, it will then store the last, depending on how it's programmed, but usually the last four minutes of your heart rhythm. So provided you regain consciousness within four minutes you will then have a recording of your heart rhythm at the time when you lose consciousness. I have got one of those devices, so if you can see it this is it here. And you can see this in comparison to my pen, it's that kind of device that literally squirts under the skin and is closed with one suture. And it stays within the chest wall for, as I said, up to five years or so.

Q. Just to assist for the purposes of the transcript, Associate Professor Raju, you just held up an example of an implantable loop recorder. And in terms of the dimensions of that device by reference, for instance to standard size pen, is it approximately one inch or one inch and a half long?
A. Yes. So it's probably two inches long and I'm going to transfer to metric, so about 5 millimetres thick in that dimension, and just under 10 millimetres wide in that dimension, probably four centimetres long.

Q. You've explained it's that type of device which could be implanted under the skin immediately in front of the heart and it has the functions that you've described in terms of recording continuously the electrical activity of the heart?
A. That's right.

Q. Can I just ask this, would you describe yourself as Ms Folbigg's treating cardiologist?
A. Not really. I believe that I'm the closest thing that she has had to a treating cardiologist, to my knowledge, no other cardiologist has consulted with her. But I was requested to consult with her by a legal team and then requested to do a specific test by the legal team, which is not the normal patient physician relationship. A normal patient physician relationship is that I consult with the patient and we, between myself and the patient, we have a

conversation about what subsequent tests are required and what management is required. And we've never really had that.

Q. When you describe this further testing, which you suggest would be prudent to complete her cardiac phenotyping, do you do so in the hopes that that will make clear the diagnostic position?
A. So yes. Obviously for Ms Folbigg it's actually fairly important to establish whether she does have a cardiogenetic phenotype, because if she does then that requires management. And so for her own health that's an important thing to try and establish.

Q. Are you suggesting these tests only because they might help the work of this Inquiry in understanding the pathogenicity of the CALM2 variant?
A. No. As in my kind of suggestion was made on the basis that she didn't, at the time or to my knowledge now, have a treating cardiologist and I think she should.

Q. Finally, Associate Professor Raju, you were named as a co-author of an article published in Europace with the lead author Malene Brohus, do you recall being a co-author of that article?
A. That's right

Q. Can I ask what your contribution was to that article?
A. Sure. So, my primary contribution was the patient history as the only cardiologist who had consulted with her and then final review of the manuscript before publication.

Q. In terms of the patient history, you're speaking of Ms Folbigg's cardiac history?
A. That's right.

Q. There is some description of her cardiac history in the article as follows. "The mother recorded recurrent episodes consistent with transient loss of consciousness that started in childhood. These episodes have been associated with intercurrent illness, physical exertion, including swimming and running, emotional stress and pregnancy. Her ECGs, cardiac exercise stress test and 24 hour Holter were normal." Is that information that you contributed to the article?
A. That's right. It's obviously summarised in the interests of keeping the scientific manuscript brief because the focus of that manuscript was obviously on the genetic and the functional data.

Q. Having undertaken the exercise requested of you by this Inquiry, reviewing Ms Folbigg's now more complete history and the cardiac investigations and reports undertaken to date, we've already dealt with your conclusion. That is your view as to whether Ms Folbigg has evidence of any significant cardiogenetic disease. Does your conclusion cause you for instance to deviate from any of the conclusions expressed in the Brohus article about the CALM2-G114R variant?
A. No.

Q. Why is that?
A. Because there are multiple potential explanations why an individual who carries an identical genetic risk may have an absent or difference in severity or difference in expression of the disease.

Q. Thank you for extending your time, Associate Professor Raju.

JUDICIAL OFFICER

Q. Thank you, Professor. I understand you're available again between 5pm and 6pm; is that right?
A. If required, absolutely.

Q. Will we be able to get a message through to you to tell you if you are required?
A. Sure.

Q. Thank you very much, Professor.

WOODS: Your Honour, I would just have one question.

JUDICIAL OFFICER

Q. You may be able to get away with coming back if you can deal with one question from Dr Woods for Ms Folbigg.
A. Yeah, happy to.

<EXAMINATION BY DR WOODS

Q. Professor Raju, you refer to the Holter test, and you said she was put on bed rest for 24 hours.
A. That was my understanding.

Q. And that was inconsistent with the preferable way of doing the ambulatory test which would have involved her walking about and doing things.
A. That is correct.

Q. And did you recognise that the custodial circumstances in which she found herself made the arrangements difficult.

JUDICIAL OFFICER: I think that's self-evident, Dr Woods.

WOODS: I think so.

WITNESS: Yes.

WOODS

Q. Just one other of the one question. The device that you talked about the loop, the implantable loop, that sits in front of the heart, does it, under the

skin?
A. It sits essentially under the left breast, in simple terms.

Q. And it's about two inches long and half an inch wide, roughly?
A. Yeah, a little bit less than half an inch wide, but yes, it's smaller than most people's little finger.

Q. Very well. Thank you.

JUDICIAL OFFICER: Just bear with me one second, will you? Mr Jordan, do you have any questions?

JORDAN: Your Honour, I can ask two questions if I'm permitted.

JUDICIAL OFFICER: Yes.

<EXAMINATION BY MR JORDAN

Q. Associate Professor, the implantable loop recorder, how long does it take to implant into a person?
A. So as I describe it to patients, it takes me longer to clean their skin for the sterile process than it does to actually do the procedure. It's - the operation itself is five minutes.

Q. Are there any risks associated with implanting that recorder?
A. So as with any medical procedure there are but they are fairly minor, so bleeding, bruising, infection. If any of those things are uncontrollable, which is exceptionally rare as in less than 1 in 1000, then you may have to explant the device prematurely.

Q. Thank you.

JUDICIAL OFFICER: You have nothing in re-examination I take it, Ms Callan?

CALLAN: No, your Honour.

JUDICIAL OFFICER: Ms Richardson is not here.

Q. Professor, thank you so much for your--

GRAHAM: Sorry, Mr Bathurst, I was proposing to seek leave to ask questions of Professor Raju but could I say this: given what he has said, could I take a raincheck and advise your chambers as soon as possible whether I do wish to seek leave on behalf of the Academy.

JUDICIAL OFFICER

Q. Professor, you may have to come back. I can't reach a final view but I think it's unlikely. Can I thank you for your patience this morning and we'll get a message through as soon as it's necessary.

A. That's fine.

Q. Thank you.
A. Thank you.

<THE WITNESS WITHDREW

AUDIO VISUAL LINK CONCLUDED AT 1.20PM

JUDICIAL OFFICER: Yes, Ms Callan.

CALLAN: Your Honour foreshadowed the parties reconvening at 2:00pm or perhaps 2:20pm, permitting time for lunch.

JUDICIAL OFFICER: When Professor Raju's gone, I'll just deal with Mr Graham and then we'll adjourn.

CALLAN: Yes.

JUDICIAL OFFICER: Mr Graham, I didn't give you leave to ask questions in circumstances where, what I might describe as the two principal protagonists have got anything to ask. I quite frankly don't see any reason to give you leave.

GRAHAM: Yes, that's correct that I need to seek leave to ask any questions.

JUDICIAL OFFICER: What did you want to ask?

GRAHAM: Well, that's why I want to consider--

JUDICIAL OFFICER: No, I'm not having that. I'm not dragging the witness back for that reason. Professor Raju is not a particularly controversial witness. I think in future that if you wish to ask questions, you better make it clear what they are because what I'm going to do is consider it on an ad hoc basis.

GRAHAM: Yes, I anticipated you might. The difficulty with that is, however, I can think beforehand of potential topics I may seek leave to ask and may or may not get, but what evidence happens at the time influences that. So it's difficult--

JUDICIAL OFFICER: But at the present time, as I understand it, you can't indicate to me any particular questions you want to ask.

GRAHAM: That's correct and--

JUDICIAL OFFICER: In those circumstances, I'm not going to bring a witness back on the off chance that you do. I would need some convincing that the questions are necessary to the Inquiry.

GRAHAM: Well, for instance, your Honour, it's unclear to me whether Associate Professor Raju has been given the Addendum to Professor Toft Overgaard and Nyegaard's report which is--

JUDICIAL OFFICER: Well, he says he hasn't as I understand it. Look, it may be that some parties - I was coming to this after the adjournment. It may be that some parties may wish to ask further questions arising out of that Addendum. That's why regrettably I'm going to have to adjourn this Inquiry. And I'll include you in that but on the basis that you'll have been given leave. I'll want to know specifically what topics you want to address. Now, that can be done at some future time if absolutely necessary but I'm not having him back here on the off chance today.

GRAHAM: Well, certainly the topics that I wish to ask him about are the history, Ms Folbigg's history.

JUDICIAL OFFICER: He's given that evidence in great detail. Mr Graham, I've given you leave. I went further than I was initially inclined to, and I don't want to have this debate with witnesses when you're not indicating to me any particular area which hasn't been covered apart from Professor Toft Overgaard and Professor Nyegaard's report which he hasn't seen. In those circumstances, I'm not inclined to give you leave.

GRAHAM: Well, I can say to your Honour the areas are history, stress testing, the interpretation of the ECG, diagnosis of CPVT, diagnosis of Long QT Syndrome and the variants associated with that. Those are the topics that I wish to—

JUDICIAL OFFICER: Thank you, Mr Graham, I understand that. In my view, they've all been adequately covered by the questions that have already been asked. So leave is refused.

GRAHAM: And that's what I wanted to consider, your Honour.

JUDICIAL OFFICER: Leave is refused. The Inquiry will now adjourned.

LUNCHEON ADJOURNMENT

I think there's someone else who wants to make an application to appear; is that correct?

CALLAN: Yes, your Honour. Those assisting your Honour have been advised that Ms von Reisner, as I understand it, wishes to make an application to be heard. She presented an application in written form. Could I provide a copy to your Honour? I understand this is something that was brought to your attention on Friday.

JUDICIAL OFFICER: Yes, I think I have a copy.

MFI #2 APPLICATION OF MS VON REISNER

Ms Von Reisner, I understand that you wish to make submissions to the extent that it's a possibility that the vaccinations received by three of the children were the cause of their death; is that correct?

VON REISNER: Yes, your Honour. I have done up-to-date—

JUDICIAL OFFICER: Yes, I appreciate that. That is a matter that I could only accept evidence from qualified medical practitioners. Whatever your opinion is, and I understand you've done a fair bit of research on it, it's not something that I can properly receive, so I'm afraid I can't give you leave to appear.

VON REISNER: Your Honour, I will not give my opinion. I would like in — the report would be included lists—

JUDICIAL OFFICER: Ms von Reisner—

VON REISNER: --of the biochemicals which were particular days injected to three babies, Patrick, Laura and Sarah.

JUDICIAL OFFICER: Ms Von Reisner, please—

VON REISNER: Those are Commonwealth official evidence.

JUDICIAL OFFICER: Ms von Reisner, please. Part of the evidence before the Inquiry is very detailed evidence of the vaccinations, the treatment the children have received at the various hospitals where they were born or where they - in the case of Patrick - was treated over the period of his life. There's no additional evidence to that that I need to assist me. I understand what you're saying, and we do appreciate your attempt to help, but I'm afraid I cannot give you leave. Thank you.

VON REISNER: Your Honour, but those evidence are official records of the Commonwealth of Australia regarding--

JUDICIAL OFFICER: Ms von Reisner--

VON REISNER: --these babies.

JUDICIAL OFFICER: Ms von Reisner, I'm trying to make myself clear. I'm not prepared to give you leave, I'm sorry. If you wish, you can resume a seat at the back of the Court, but I don't propose to hear you any further.

VON REISNER: Can I have the written decision regarding this that I can ask for review of the decision?

JUDICIAL OFFICER: I'll give you a written decision at the conclusion of the Inquiry.

VON REISNER: I don't ask to provide my opinion. I just would like to provide Commonwealth official documents printed and published by Commonwealth

themself.

JUDICIAL OFFICER: I understand this. I'll give you my reasons now. Take a seat while I give you the reasons.

Ms Koidu von Reisner has sought leave to appear at the hearing and has supplied me with a great deal of material, some which she describes as Official Commonwealth Records.

The thrust of her application is to make submissions that those records give rise to a reasonable possibility that the deaths of the four Folbigg children were due to vaccinations which were received during the short period of their lives.

I have considered the material and I do not propose to grant leave for the following reasons.

1. The medical records of each of the children have already been tendered at the Inquiry.

2. To the extent that Ms von Reisner expresses opinions on the material she has received she is not qualified and does not have sufficient expertise to give it.

3. Giving leave in those circumstances would only unnecessarily complicate and add to the burden already upon the Inquiry and those assisting in it.

In those circumstances, leave will be refused.

I direct that this judgment be made available to Ms von Reisner in due course, so if she wishes to make any application or review she can do so.

CALLAN: Your Honour, I note that in the Listening Device Transcript in unredacted form, which was tendered this morning and marked Exhibit 16-06, your Honour made a non-publication order in relation to certain content of that Listening Device transcript. Your Honour's order was made on 1 November 2022 and was expressed to continue until the conclusion of the first hearing block, that is this week. In the interim the parties have been given an opportunity to consider their position in relation to the non-publication order. Your Honour, Mr Craig Folbigg is not legally represented in this Inquiry, but was granted, as your Honour would recall, leave to appear as a party. He has communicated through those assisting your Honour that he seeks a continuation of this non-publication order effectively until further order of the Court, on the basis that the particular content may be misconstrued, if I can put it that way. And I raise that, your Honour, in circumstances where Mr Folbigg is not legally represented.

JUDICIAL OFFICER: Does anyone here oppose a non-publication order up to and until the conclusion of the Inquiry?

WOODS: Of the whole Inquiry, your Honour?

JUDICIAL OFFICER: Yes.

WOODS: Your Honour, we would seek the variation of the order such that it be made available to us.

JUDICIAL OFFICER: I'm sorry, yes.

WOODS: And if necessary any relevant submissions we make about it can be in writing to your Honour.

JUDICIAL OFFICER: Dr Woods, the order that was made was that the content may not be published until after the conclusion of the first hearing block, except to, among the other exceptions are lawyers acting for Ms Folbigg and Mr Folbigg.

WOODS: Very well, that's all right.

JUDICIAL OFFICER: So you're covered as far as that's concerned.

WOODS: We're covered with that, yes.

JUDICIAL OFFICER: All I'm simply doing is extending that order up until the conclusion of the Inquiry.

WOODS: Yes, that's fine.

JUDICIAL OFFICER: Has anyone else got anything to raise as far as that's concerned? Very well, I make that order.

That the non-publication order made on 1 November 2022 will be extended up to the conclusion of the Inquiry.

Where do we go from there?

CALLAN: Next is future progress of the matter. As was reflected in some comments I made during the opening this morning, although it had been anticipated that the witnesses for the whole of this week and possibly next week would cover the spectrum of experts who deal with cardiac and genetic matters, upon being advised by Professor Toft Overgaard late last week of a further functional test that he had just conducted, it seemed that a different approach was warranted so as to ensure that the parties and all the other relevant experts had sufficient time to consider the content and implications of that work. In those circumstances we propose, as I indicated in the opening, to take the evidence-in-chief of Professor Toft Overgaard and Professor Nyegaard and that will commence tomorrow morning. But to adjourn the proceedings at the conclusion of their evidence-in-chief.

That means that there will, it is foreshadowed, be a number of the cardiac and genetic expert witnesses called during the hearing block that's to commence in February 2023, it seems plain enough that that means that more time will be

required in February 2023 than was previously foreshadowed. And more generally, those assisting your Honour have proposed variation and further orders over and above the order your Honour last made on 6 September to accommodate a process for circulating a proposed witness list otherwise dealing with matters preparatory to the February hearing block and seek confirmation from your Honour that the second hearing block which is to commence on 13 February of 2023 will have an estimate of three weeks. And there are certain timetable orders proposed by way of exchange of written submissions in advance of closing oral submissions. Can I ask if your Honour has a copy of the short minutes of order?

JUDICIAL OFFICER: I do. Have they been circulated?

CALLAN: They were circulated before the luncheon adjournment. I note order 8 has the parties serving written submissions on 7 April, and I understand that's Good Friday. So I expect, fairly, that date should be changed.

JUDICIAL OFFICER: You better make it the 6th, I suppose.

CALLAN: Otherwise, your Honour, the parties have had sufficient opportunity to consider their position, they might address your Honour on any difficulties with that timetable.

JUDICIAL OFFICER: Before they do, one matter I was concerned about was the delay which would not prolong the giving of my report. The proposed timetable achieves that object of no delay. I'd only say that the second hearing block will commence on 13 February with an estimate of three weeks but it will continue until that hearing block is finished. I think three weeks should be enough but I do want to have the matter concluded as quickly as possible. It's in the interest of Ms Folbigg and in the interest of the community generally.

CALLAN: Yes, your Honour.

JUDICIAL OFFICER: Having said that, has anyone got anything to raise as far as the proposed amendment is concerned? Dr Woods?

JORDAN: Your Honour, in the interim I would raise one matter if I might?

JUDICIAL OFFICER: Yes.

JORDAN: Would your Honour consider extending the proposed period in order 8 to 14 April?

JUDICIAL OFFICER: I don't have a difficulty with that.

CALLAN: There's no difficulty from our perspective, your Honour.

JORDAN: Thank you, your Honour.

JUDICIAL OFFICER: Dr Woods?

WOODS: Order 8 would be to the 14th?

JUDICIAL OFFICER: 14 April.

WOODS: Otherwise, we have no objection to that, your Honour.

JUDICIAL OFFICER: Thank you. Ms Richardson. I assume silence signifies assent? Have you got any trouble with the timetable?

RICHARDSON: No, your Honour.

JUDICIAL OFFICER: Mr Graham, I'll give you leave to ask?

GRAHAM: With your Honour's leave to address your Honour, no problems with the orders.

JUDICIAL OFFICER: Thank you. Who have I missed? Ms Love.

LOVE: I've got no problem with the orders.

JUDICIAL OFFICER: I don't think Mr Fraser is here so he'll have to take it or leave it. I'll make those orders. I think so people who are listening to this live know what's going on, I should read them out.

1. A proposed witness list in respect of the second hearing block will be circulated to the parties by 13 January 2023.

2. Counsel Assisting will serve on the parties a list of any further evidence proposed to be tendered in the second hearing block on 13 January 2023.

3. If any party wishes to place written evidence before the Inquiry for the second hearing block, that evidence should be provided to the solicitor assisting the Inquiry by 23 December 2022. Counsel Assisting will indicate by 13 January 2023 if it is proposed to tender that evidence at the second hearing block, and if not, any party may provide a written application to the solicitor assisting by 20 January 2023 for consideration by me.

5. If a party wishes to have a witness called to give oral evidence before the Inquiry at the second hearing block, a written application is to be provided to the solicitor assisting the Inquiry by 20 January 2023.

6. Counsel Assisting will indicate by 25 January 2023 if it proposes to add that person to the witness list, and if not, any party may provide written application to the solicitor assisting by 30 January 2023 for consideration by me.

7. The second hearing block in the Inquiry will commence on 13 February 2023 with an estimate of three weeks.

8. Counsel Assisting is to serve written submissions by 24 March 2023.

9. The parties are to serve written submissions by 14 April 2023.

10. The listing for the delivery of oral submissions from 26 April to 28 April 2023 is confirmed.

As I indicated, the effect of those orders would be that it should not delay the publication of my report at all. If that's to be achieved, however, it's getting a little bit tight. There has to be fairly strict compliance with the timetable. I do urge the parties to try not to, as best they're able, to have evidence, particularly scientific or medical evidence, attempt to serve it late so it would delay the hearing further. I don't wish to say any more to that. In saying that, I emphasise I'm not being critical as to what has occurred to date.

That brings me to the more immediate. Professors Toft Overgaard and Nyegaard are to be called tomorrow. It was proposed to have a 9.30am starting time. Has anyone got objection to that? I know it was originally set for 11:00am but - yes, Mr Jordan?

JORDAN: It is really a personal matter but Dr Pritchard is being sworn in tomorrow.

JUDICIAL OFFICER: What time is that? 9.30am presumably?

JORDAN: Yes.

JUDICIAL OFFICER: I think in those circumstances, Ms Callan, we should start at 10.30.

CALLAN: Yes, your Honour.

JUDICIAL OFFICER: So we will commence at 10.30am tomorrow. Now, it is proposed, Ms Roy is it, I think you were going to take these witnesses?

ROY: Yes, your Honour.

JUDICIAL OFFICER: Do them together?

ROY: Yes, we'll call them together.

JUDICIAL OFFICER: And what procedure are you going to adopt? Are you going to address questions to each of them separately or--

ROY: I'd like to take their evidence together, your Honour.

JUDICIAL OFFICER: Take their evidence together?

ROY: Yes.

JUDICIAL OFFICER: Whichever of the two wishes to answer your questions?

ROY: Yes.

JUDICIAL OFFICER: I understand. Is everybody happy with that course? Okay. Very well, that's what will happen. Is there anything else, Ms Callan?

CALLAN: Not on my list, your Honour, no.

JUDICIAL OFFICER: I'll adjourn until 10.30am tomorrow.

ADJOURNED PART HEARD TO TUESDAY 15 NOVEMBER 2022 AT 10.30AM