SPECIAL INQUIRY

THE HONOURABLE THOMAS BATHURST AC KC

NINTH DAY: WEDNESDAY 22 FEBRUARY 2023

INQUIRY INTO THE CONVICTIONS OF KATHLEEN MEGAN FOLBIGG

AUDIO VISUAL LINK COMMENCED AT 9.06AM

<DOMINIC JAMES RICHARD ABRAMS, AFFIRMED(9.07AM)

<EXAMINATION BY MS ROY

Q. Can you tell the Inquiry your full name?
A. My name is Dominic James Richard Abrams.

Q. And your place of work?
A. I work in Boston, Massachusetts at Boston Children's Hospital.

Q. Professor, you provided your CV to the Inquiry at our request.

ROY: For the record, that's now Exhibit 34, tab 4, red page 156.

Q. I'm not going to take you through it, but I'll ask you to confirm some relevant details. You're a medical doctor?
A. Correct, I am.

Q. You obtained a degree in 1994 from St Mary's Hospital Medical School, Imperial College, London?
A. I did.

Q. You became a member of the Royal College of Physicians in Paediatrics in 1998?
A. I did.

Q. And obtained a certificate of Paediatric Cardiology in 2007?
A. I did.

Q. In 2008 you were conferred a Doctorate in Medicine in Cardiac Electrophysiology?
A. That is true, yeah, right.

Q. You are currently an Associate Professor of Paediatrics at Harvard Medical School?
A. Correct.

Q. And you are also currently a Cardiac Electrophysiologist at Boston

Children's Hospital?
A. I am.

Q. And a Cardiovascular Geneticist at Brigham Women's Hospital in Boston?
A. That's true.

Q. You are Co-Director of the Centre for Cardiovascular Genetics at Boston Children's Hospital?
A. I am.

Q. And that's the centre that you helped to found?
A. Correct, I did.

Q. You currently practice clinical medicine?
A. I do.

Q. In what capacity?
A. So, I will see patients in the clinic, we will review clinical notes from patients, organise investigations, make diagnoses and recommend specific management plans for those patients and also the wider family as well.

Q. And the Centre for Cardiovascular Genetics has a translational research group?
A. We do, yes. So, some of our members are much more involved in translational lab-based research than I am and obviously we very much collaborate together to ensure the sort of - the bedside collaboration as it's referred to, is streamlined.

Q. Is it right that the current focus of that work is CPVT, calmodulin mediated disorders and arrhythmogenic and restrictive cardiomyopathy?
A. That's true.

Q. I think, as you've said, you're not personally involved in laboratory-based research?
A. No, I'm not, no. I work with those who are, but it's not something I personally do or have done in the past.

Q. Can you tell us about your specific expertise and experience with respect to calmodulinopathy?
A. So, for many years I've managed patients with both Long QT Syndrome and Catecholaminergic Polymorphic Ventricular Tachycardia or CPVT. A lot of our patients have both those diagnoses. Those clinical phenotypes are well known to me and as of late we've seen an increasing number of patients who have variance in the calmodulin genes associated with one or either of those two.

Q. Are you able to tell us how many patients you have with the calmodulin variant?
A. So, we have four affiliated with our centre and I've been closely involved in three others.

Q. Four at the centre and three that you've otherwise been involved with? Is that right?
A. Correct yeah. Yeah, that's correct, so seven.

Q. Are you able to tell us how many of those have Long QT?
A. I think four have Long QT and three have CPVT phenotype.

Q. Have any experienced IVF?
A. Yes. One has a diagnosis of Idiopathic Ventricular Fibrillation. That's correct, yeah.

Q. Does that travel with either Long QT Syndrome or CPVT in that patient?
A. Not in that individual that we've identified to date, no.

Q. Do any have mixed phenotypes?
A. Not in any of the patients I've seen. No.

Q. Do any have other suspicious phenotypes that might be associated with their CALM variant, other than Long QT, CPVT and IVF?
A. Not that I can recall, no. No, nothing specific stands out.

Q. Have each of those cases then identified as either *de novo*, mosaic or familial?
A. I think there are no mosaics that we know of. Some parents are not available for testing but the vast majority have been *de novo*.

Q. Are you aware if any of those cases are actually familial cases?
A. We've never identified a familial case, but we haven't had access to all the patients. Not all of them are primarily managed by us but we've been asked to be involved in their care.

Q. To your knowledge, are any of those cases included in the International Calmodulinopathy Registry?
A. They're not, no.

Q. Coming to your involvement with this Inquiry, is it correct that you were first contacted by members of the solicitor team assisting the Inquiry in January of this year?
A. That's correct.

Q. And shortly thereafter on 13 January of this year you were retained to provide an expert opinion in the form of a report?
A. That is correct.

Q. You provided a report dated 27 January 2023?
A. I did.

ROY: For the record that's Exhibit 34, tab 4.

Q. In terms of the record, the provision of that report, there was an

administrative issue connected to your university that delayed providing the report to the Inquiry, so it wasn't actually received on 27 January. Is that right?
A. That's correct. Yeah, it was - yeah.

Q. For the purpose of preparing that report, you were provided with a large volume of material?
A. I was.

Q. And that included reports from other geneticists and cardiologists to assist in the Inquiry?
A. Yes.

Q. Were you able to review all of those reports and factor in the views when you provided your opinion?
A. Everything that was provided before I submitted my report, yes, I was able to review. Some came after I submitted my report.

Q. Subsequent to providing your report, you received another round of expert reports?
A. I did, yes.

Q. And you've had a chance to consider those?
A. I have.

Q. Prior to being contacted by the solicitor team in this Inquiry, were you otherwise aware of the Folbigg case?
A. I was aware of the case, yes.

Q. How were you aware of it?
A. I was aware of it through Professor Skinner. I was aware of it in the publication in Europace and I was aware of it from the lay media response to that paper and publications around the world.

Q. Prior to being retained as an expert, had you formed a view about the pathogenicity of the G114R variant?
A. I wasn't really available to access all the information obviously that I was before, but we were still, you know, learning about calmodulin at that time, but yes, it was obviously a variant of great interest but one that needs to be evaluated further.

Q. I'll come back to the substance of your report, but I'd like to begin by confirming your overall conclusions in relation to whether or not there is a reasonable possibility that Sarah or Laura Folbigg's deaths were caused by a fatal cardiac arrhythmia attributable to the CALM variant. At red page 144 of your report, Exhibit 34-04, you said, "whilst a cardiac arrhythmia related to the calmodulin variant cannot be definitively excluded, I believe the likelihood this was responsible for the deaths of Laura and Sarah is low." That remains your view?
A. That remains my view.

Q. Later down the same page in the final paragraph of your report, you say, "It is possible, but unlikely to my mind, that Kathleen Folbigg has a very mild form of CPVT and her two children died at a very young age during sleep, from either CPVT or another condition associated with calmodulin." Does that remain your view today?
A. That remains my view.

Q. You said that you cannot exclude the possibility that a cardiac arrhythmia related to calmodulin caused the deaths of the children, the girls. Accepting that you say the likelihood of this is low and the possibility unlikely, would you nevertheless characterise that possibility as reasonable?
A. I think it's - it's possible. It's an assumption that you could make based on identification of a variant in a deceased individual in calmodulin based on everything we know about the gene, the pathogenicity of variants in that gene and the associated phenotypes which have a very high risk of life-threatening ventricular arrhythmias. So, in that situation it is a reasonable assumption, but then you would want to explore the wider family further and therefore identify other members who may or may not, of the family, who may or may not have the variant, which in this case would be the mother of the two girls, and evaluate her. Based on the fact that she does not have a significant or definitive phenotype associated with many of the conditions that we know to date has been described as caused by calmodulin variants, that to my mind shifts weight away from this being a pathogenic variant that's responsible for an arrhythmic death of the two young girls.

As I alluded to in my report, really the only way you can be definitive about that is by recording their heart rate at the time of any subsequent or terminal event, and obviously we don't have that information so that's why we cannot definitively say that this was or--

Q. Do you recall seeing a letter from Professor Hugh Watkins concerning this case?
A. I do, yes.

ROY: That is at Exhibit 14, tab 10, red page 321.

Q. At this stage, at the time of writing this letter, Professor Watkins had been briefed with the reports of Professors Schwartz, Wilde and MacRae and I think that was, again, due to the delay we referred to earlier in relation to your report, he hadn't seen your report. On red page 322, Professor Watkins says this, "Looking at the three reports, what I see is that reasonable, thoughtful and authoritative experts are all looking at the same data but are drawing different inferences. This is because there is substantial uncertainty and so judgement, rather than interpretation of indisputable fact, is needed to reach a conclusion." Do you recall reading that?
A. I do.

Q. And so you agree with what he says there?
A. Yeah, I do, and I think it was - it is - there is uncertainty in this situation, as there are in many other situations like it, where we have a genetic finding in a

family with minimal clinical evidence of a condition and two episodes of sudden death, so therefore we have to try and use judgment to determine what was the cause of the deaths in the two individuals and to what degree that can be attributed to the variant in question. So, I don't think there is definitive evidence either to say yes or no and therefore it becomes - down to a degree of interpretation and judgment based on your views on the variants and the likelihood of that happening.

Q. Professor, your evidence comes at the conclusion of the cardiogenetic experts who have given evidence to this Inquiry and we also have the benefit of your comprehensive and very clearly written report, if I may say, which again due to its timing means that it also encompasses your responses to many of the other experts in the Inquiry. For that reason, I do not propose to take you at length through your report with the views you've expressed, particularly those that are concordant with those that have been expressed by others, and so I will instead step through essentially in order, areas of your report in relation to which the Inquiry would benefit from some elaboration. So coming to your report, which again is Exhibit 34, tab 4, red page 129, and I regret I don't think we've given you updated pagination. We can put it on the screen. It would be six pages into the PDF of your report, Professor.

JUDICIAL OFFICER: What is the red page?

ROY: 129.

Q. Do you see that page, Professor? It's coming up, I'm sorry, just a minute.
A. Okay.

Q. In the third from the bottom paragraph you describe in the second sentence:

> "A transcriptional analysis of fetal, infant and adult human myocardial samples demonstrating expression of all three transcripts, CALM3 expressed to the greatest degree, followed by CALM2 and CALM1, a difference which was statistically significant."

What does that mean?
A. Sorry, that study was by Dr Crotti back in 2013 when calmodulin was really in the very early days of our understanding and the first patients described. What they were able to demonstrate was that there were transcripts in all three of the genes, so messenger RNA which goes on to create the protein was identified in tissue from the heart in the small number of individuals they were able to test. So it tells us that the product of all those three genes is expressed in the human heart.

Q. It continues, the next sentence refers to transcripts from all three genes in 432 human left ventricular samples, reporting medium CALM1 transcripts per million 111.9, CALM2 205.4 and CALM3 119.5. Does that suggest that CALM2 is expressed at twice the rate of CALM1 and 3 in the heart?
A. It could do in that specific example, yes. There was a higher count in

CALM2, but how we interpret that into a clinical situation I think remains very unclear, but that was as it was reported in the GTEX database or the portal, which is tissues from, as I said, 432 individuals where there was myocardium available and they were able to express or able to analyse the expression of those genes, but how we interpret that beyond that I think is difficult to say at this stage.

Q. Would it be reasonable to conclude that a CALM2 variant might be more pathogenic than an equivalent variant in CALM1 or CALM3 from that data?
A. I don't think you can say that, no.

Q. Coming then to red page 131 which is two pages forward in your report, which again should come up on the screen, about half way down that page where the numbering starts, can you see that?
A. Yeah.

Q. Here you're giving examples of families that had been described in each of the three calmodulin phenotypes?
A. Mm-hmm.

Q. Over the page, red page 132, the third point you describe an Idiopathic Ventricular Fibrillation family?
A. Yeah.

Q. Four siblings suffered cardiac arrest with ventricular fibrillation documented in the two survivors in the absence of abnormality on the 12 lead ECG and cardiac imaging. Two other family members were identified to have the CALM1 variant were asymptomatic but all four surviving family members showed paradoxical QT prolongation in early recovery from exercise. First of all, just so I understand this report correctly, notwithstanding the lack of symptoms in the two family member carriers, I take it that as a result of QT prolongation this variant was still considered fully penetrant in this family?
A. We - yes, if you would define penetrance as paradoxical QT prolongation, I would say it was. Obviously this is not a study I wrote or was involved with, it comes from Amsterdam, but that would be my inference of that report, that when they, in the supplement - paper, they demonstrated the exercise tests and they all showed an abnormal response of the QT to exercise. Therefore I would personally consider that a phenotype of a sort of Long QT-type picture, albeit relatively subtle but it was evident.

Q. Why is it described as paradoxical QT prolongation?
A. Early in the recovery period from exercise the QT intervals should shorten, which allows the heart rate to increase, amongst many other things. So if it prolongs, that's an abnormal response and we see that in different forms of Long QT Syndrome.

Q. Is it paradoxical as against a healthy human, but not paradoxical as against a Long QT phenotype?
A. Correct. So it goes against those who don't have Long QT Syndrome.

Q. Coming then to red page 134 of your report, which is topic two and where you undertake the classification exercise applying the ACMG criteria, your conclusion, having applied that criteria, is that this is a G114R in CALM2 is a variant of uncertain significance and--
A. Yes.

Q. --you maintain that view?
A. I do.

Q. You apply first as moderate evidence the fact that CALM3-G114W has been determined to be pathogenic?
A. Mm-hmm.

Q. That's obviously in CALM3 and not CALM2. Do you consider those two genes relevantly interchangeable for this purpose?
A. I think that goes to a level of basic science in understanding how the protein works and whether the protein produced by the three different genes is identical in structure and function. I think we believe it is, but there may be elements of it that we don't particularly well understand that would be outside my knowledge and expertise, but sort of it's either very similar, if not identical.

Q. Next you've applied as supporting evidence the functional assays that were supportive of a damaging effect on the gene or a gene product?
A. Mm-hmm.

Q. Pausing there, when you refer to the functional assays, are you referring to those that were reported in the Brohus article?
A. Yes.

Q. Does this view also take into account the further assays that were conducted at the end of last year by Professor Toft Overgaard in respect of the punitive benign variant I10T?
A. So I think in terms of the functional assays, again sort of understanding the methodology of those and the specific critique, as I said in my report and say so again, is outside my areas of expertise. I do think that these particular assays are very difficult to reproduce exactly what happens in the human heart, and I've given examples of where functional assays have not correlated with what we see in the clinic. So, I think it's very difficult to do that and that's of no disrespect to the quality of the science at all, again that's outside my area of expertise to comment on, but I do think it's obviously been shown it's deleterious in those assays, but how you apply that I think obviously is an area that could again fall into the judgment with different individuals coming down to different opinions, but again I would defer to those who have more expertise in critiquing the methodology and the structure of the assays than me.

Q. Assigning that only supporting evidence and not, for example, moderate evidence, is that because you consider it outside of your area of expertise, or on what basis are you applying only supporting evidence?
A. I think it's because the variants were expressed in isolation without a wild-type variant, that's obviously stated from the very nice International

Calmodulinopathy Registry paper that these assays are prone to errors because they don't replicate exactly the human heart and that's why people have moved towards more looking at induced pluripotent stem cell cardiomyo science to try and replicate human cardiac physiology more accurately and so obviously those are challenging and, certainly in my experience as a clinician, we have ultimately seen reports where functional conclusions were drawn which ultimately did not correlate with what we saw in the clinic. So, that's why I gave it that criteria.

Q. Do I take it from what you said that because the assays that were reported by Professor Toft Overgaard at the end of last year, both in respect of the potentially benign variant as well as in respect of G114R's sodium channel effect, that they fall in the same category as the tests reported in the Brohus article?
A. Yes, they were all performed in in vitro heterogeneous models rather than in iPS cells.

Q. So they don't change your view as to the weight to be given to that criteria?
A. It wouldn't specifically change my view but again it's something that I'm not an expert in. I think ultimately my conclusion for the ACMG criteria came because I felt that there was discordance and that there were negative findings in the benign criteria as well as positive findings, and therefore to my mind I would call this a grade of unknown significance based on that specifically.

Q. We'll come to that in a minute. You also note the absence as a criteria from controls. You've not downgraded the criteria by reasons of the presence in the UK Biobank for CALM3-G114R variant?
A. Mm-hmm.

Q. Can you explain why not?
A. Because I haven't seen that variant specifically, so I haven't seen evidence that it's there. We looked for it very hard and I believe it's in the Regeneron database which we could not access anymore. I did specifically look for it but I didn't comment on it really because I haven't seen any data myself to support that.

Q. Next is the PP2 criteria missense variant in a gene that has a low rate of benign missense variation and in which missense variants are a common mechanism of disease. First of all I take it you accept that calmodulin are highly constrained genes?
A. Yes.

Q. Among the most constrained of all genes?
A. I think that comes down to how you define constraint. There are different ways that that can be looked at. Firstly by simply looking at the observed ratio to the expected ratio of variants, or the ratio of observed to expected variants, and certainly when you're doing it on that metric the calmodulin genes are very highly constrained. There is another mechanism using the sort of further computational analysis to create a Z-score to define constraint that way. In that case then the calmodulin genes appear less constrained because they

sort of fall into the - around from 18,000 or so genes they were around the 1000 mark, whereas in the observed or expected ratio they were in the highest 20 constrained genes ..(not transcribable).. those two, but I think that even allowing for both of those two things you only have to look at the genes and see how many benign variants are in it, which they do exist but they're rare, to understand that these genes are highly constrained.

Q. Specifically in relation to the Z or Z-score, is that - have you considered Professor Nyegaard's evidence in relation to the utility of those scores in relation to calmodulin?
A. I think they're helpful. Again, they give you useful information in terms of interpreting how a variant then goes on to behave in a patient, but I wouldn't sort of put huge weight on them in terms of understanding how the variant behaves specifically. What we also know and hasn't been analysed for the calmodulin genes is there may be certain areas of a gene that are heavily constrained and other areas of the gene that are less constrained. If you look in the Genome Aggregation Database, which is where a lot of this information comes from, that's been analysed for certain genes, so you can say certain regions are more tolerant to constraint than others. Looking at what we know so far, it would appear that there are more benign variants outside of the four calcium-binding EF hands, and that's the site where benign variation tolerates it somewhat better via gene than within one of those critical functional regions. So, I think again, rather than saying whether the whole gene is highly constrained although it may be on average, there may be regions that are less constrained and other regions that are more constrained.

Q. With respect to the actual locations in the gene, is that an area where you have specific expertise?
A. No. I mean, this is how I would interpret genetic findings when I'm looking at a patient in clinic. So, we would go through the Genome Aggregation Database, we would look at the functional data, we would draw a conclusion based on that and use that in the clinical scenario. So, the way I approached this case was exactly as I would do in clinic with a patient.

Q. I'm sorry, continue. I cut you off.
A. Have I gone into the detailed methodology of understanding how that constraint is derived and that stuff? No, I have not. So, it's something I use but I didn't create it.

Q. So, that was with respect to constraint. With respect to the actual location on the gene, you were referring to constraint in relation to certain locations. Were you referring to the EF hand motifs and the trend that's been noticed--
A. Yeah.

Q. In terms of other features of the calmodulin protein, it's structure, would it otherwise be within your area of expertise to comment on the significance of different locations on that gene?
A. No, I would say it would not.

Q. And would you defer to Professor Toft Overgaard with respect to the significance of different locations and particularly the location of G114R in that respect?
A. Yes. So, they've obviously had huge experience in understanding the functional structural effects of location - of variants in different locations. However, ultimately I would say what is the most important thing is how that variant plays out in the clinical phenotype of the individual patient.

Q. In relation to calmodulin being a highly constrained gene generally, would that fact warrant upgrading the PP2 criteria from supporting to moderate in this case?
A. I certainly wouldn't feel comfortable doing that myself. Although it is constrained to a significant degree, I don't know how, you know, in the absence of more robust clinical data, I don't know how we could demonstrate.

Q. Finally, in terms of supporting criteria in favouring pathogenicity, you apply the multiple lines of computational evidence supporting a deleterious effect on this gene. In that assessment have you included the recently reported deep mutational scanning model that Professor MacRae was involved with?
A. That's not included, no.

Q. You're aware that predicted G114R to be pathogenic?
A. I did. I saw that, yes.

Q. And why didn't you apply that? Why didn't you give weight to the results of that model?
A. Well, I think it's a very new technique that's yet to be validated further. It examines yeast, not humans. And I didn't feel I had enough experience of it to use it at this time personally. Others may feel differently but I didn't feel - having never used it before I didn't think it was appropriate to bring it in at this situation.

Q. Coming now to the countervailing criteria that favoured a benign status - this is in your report, Exhibit 34, tab 4, red page 135, where you address BS2, this is observed in a healthy adult individual for a recessive dominant or X-linked disorder with full penetrance expected at an early age. This is what you've referred to already, reflecting the fact that Kathleen Folbigg is a G114R carrier?
A. Correct.

Q. You've applied this as strong evidence?
A. Mm.

Q. You go on to say that based on what is known about calmodulin diseases to date, you believe it is reasonable to assume penetrance is expected at an early age and you've italicised "expected". Can you expand on that?
A. Yes. I think if we look at all the patients who've been described to date, initial papers in the International Calmodulin Registry and in papers that have been published since that time in 2019, the penetrance remains exceedingly high, so there are very few patients or none that I can identify where there

wasn't evidence of the disease. Therefore to my mind based on what we understand to date, it would be expected to - that we could demonstrate a phenotype at a relatively young age in someone with a calmodulin variant.

Q. For clarity, I infer from the whole of your report that you also accept that CPVT is a phenotype associated with calmodulin variants?
A. Yes. In my personal experience it is.

Q. Do you consider that there is any substantial doubt in your area of specialty, that CPVT is an established phenotype of CALM variants?
A. I think the experience is still very small because the number of patients with CPVT are less than those with Long QT Syndrome. But certainly I've seen patients presenting with a clear clinical phenotype of CPVT, who ultimately were found to have a *de novo* variance in one of the calmodulin genes. So, that to my mind would be good evidence for support it's a form of CPVT. How it is similar or differs to the classic form of CPVT which is associated with the ryanodine receptor, which is by far the most common form, I think time will tell as we get more experience in more patients with calmodulin variants.

Q. Professor Schwartz has informed the Registry that the updated ICalmR database will include one case in a familial calmodulin variation in which the infant was diagnosed almost at birth with Long QT Syndrome and the father, well into adulthood, had no evidence of penetrance. Assuming that case was not mosaic, does that change the application of this criteria?
A. I think non-penetrance is a concept that you would have to determine how aggressively that individual was studied. How was the criteria of-non penetrance defined, so that individual may have had a normal resting ECG in terms of the QT interval, but if you put that patient on a treadmill you may have found a very different response. So, I think without knowing the specifics of the case it's very difficult to comment on that further. And I read that from Professor Schwartz's report, but again there was very little detail around the specifics of that case.

Q. If you assume just for the purposes of my question, that the possibility of a phenotype was thoroughly investigated and excluded in the father, would that warrant a downgrading or removal entirely of this criteria?
A. Well, I think it would be one individual from probably over 100 have been reported to date, so it comes down to a definition of expected. I think expected would be - to my mind it would be likely or probable that that happens, so I don't think that one case would change ‘expected’, to my mind.

Q. Second in relation to this criteria is Kathleen Folbigg's health. You documented the evidence available to you in respect of that at the appendix to your report, which is in Exhibit 34, tab 4, red page 153. I'm not going to take you through that in detail. But can I check that it's correct that you applied your own measurements to determine Kathleen's corrected QT interval from her exercise test?
A. I did.

Q. And you describe on red page 155, you describe various challenges

associated with assessing that data. But the maximal corrected QT at length as you corrected it was 456. So, you observed some lengthening of her QT during exercise but you consider that consistent with a normal healthy individual?
A. Yes. I didn't think it went to a range that I would consider suspicious for Long QT Syndrome considering the upper limits or normal for an adult female, albeit at rest, is 480 milliseconds and the way I assessed it is how we would assess any patient we were seeing with Long QT Syndrome, which is to assess the exercise test in detail and then to plot the results against controls. So, I felt that she fell within the control range that we would use.

Q. Professor Wilde described conducting his measurements by hand and considered that at three minutes into the exercise test, the corrected QT was in the range of 470. First can you explain why there would be different measurements between the two of you?
A. I don't know exactly how he measured it or how he did it. I use a technique whereby the images were removed from the PDF and then annotated, so I would link the dV IDP which is the steepest down slope T-wave and whether it intersects with the isoelectric line and then use that as the QT interval and then correct the heart rate using the preceding RI interval. I do one specific technique. How Professor Wilde does it, I wouldn't be able to comment, so he may use a slightly different technique that may give a slightly different answer.

Q. If you accepted his measurements produced a maximal of 470 three minutes into exercise, would that degree of prolongation place Kathleen within the range of other pathogenic calmodulin variant carriers with LQTS phenotype?
A. We have very little evidence or very little experience of the exercise test in calmodulin specifically. I would expect the QT to be relatively rigid and so not to accommodate particularly well in exercise than to shorten. But I would still feel personally that 470 was not excessive prolongation that would allow me to make a diagnosis of Long QT Syndrome at three minutes.

Q. Are you aware of variant carriers recorded on the Calmodulinopathy Registry who are described as asymptomatic but showing mild QT prolongation on exercise?
A. Yes. I've seen that, yes.

Q. Would a QT of 470 at three minutes of exercise, be consistent with that description, that is asymptomatic but mild QT prolongation on exercise?
A. I personally wouldn't feel that comfortable saying that was mild QT prolongation. I still would feel that falls within the normal range.

Q. Professor Wilde also considered the occurrence of ventricular ectopy during the exercise test was compatible with the CPVT phenotype. You've commented in your report, I'm taking you back to red page 137, Exhibit 34, you note that there was no complex polymorphic or bidirectional non sustained ventricular tachycardia as is typically seen in CPVT?
A. Correct.

Q. Professor Wilde had also noted in his report at Exhibit 9, tab 2, red page 27, he said, "This test is compatible with CPVT but lacks the most typical findings of CPVT, ie polymorphic bidirectional doublets, which is frequent in regular CPVT patients." Is that the same thing that you were referring to?
A. Yes. Yeah, I think it is. I mean, I think there was nothing - if I saw that test in isolation I do not feel I would be comfortable making a diagnosis of CPVT. It doesn't have the classic hallmark of it, so typically we start to see ventricular ectopy initiating around the rate of 110 to 120 beats a minute. This was later. And I didn't feel that the burden of ectopy, the degree or ectopy or the morphology was entirely - was consistent with a definitive diagnosis of CPVT. If there was another family member who had CPVT very clearly clinically diagnosed, then that might sway your inference somewhat, and, as I said in my report, there were two very well-known families with lots of family members who seemed to have a milder form of CPVT and that would be consistent with that picture. In the absence of having sort of a definitively identified proband if you like, like the first person in the family to present, and I think it's very difficult to make a diagnosis of CPVT on that alone is my personal opinion.

Q. One of the other comments Professor Wilde had made is that the typical arrhythmias, the polymorphic bidirectional doublets are rarely seen in CALM-associated CPVT. Is that your experience as well?
A. In the most recent patient, yes it was, but other patients are described in the literature who I specifically look back who do have bidirectional VT. The second patient in the Nyegaard paper from 2012, they show a very nice ECG of bidirectional VT and in a recent paper from Paris also had evidence that the individuals had evidence of bidirectional VT. So I think there was a school of thought that patients with calmodulin may not display the classical bidirectional VT that we associate with typical ryanodine receptor mediated CPVT, but I think in the very small number of patients we have available to us to date, that may not hold true and they may show the same clinical arrhythmic picture.

Q. To make sure I understand your evidence correctly, you don't exclude CPVT in Kathleen Folbigg?
A. I don't feel comfortable making a diagnosis of CPVT clinically based on the evidence we have.

Q. You don't feel competent making the diagnosis, but do you consider the diagnosis excluded by the evidence?
A. No, it's not excluded. I'd say it's unlikely.

Q. From your report, Professor, I think it wasn't clear whether you had also seen the 2003 ECG of Kathleen Folbigg. If we could briefly put in on screen, Exhibit 14, tab 6, red page 228. While that's coming up, I observe that Professor Raju had noted prominent inferior U waves in this ECG. Are you able to comment on that?
A. So, there are U waves evident in the inferior leads and I think you can see lead II, III and potentially lead aVF, which is the inferior lead, so that would be consistent with what Dr Raju said. The quality of the ECG is not great but the standard definition of a U wave is .2 mV, and looking at it from here I would

say those probably did not reach .2 mV to be significant U waves, I would say those U waves were probably within normal realms of physiology.

Q. From that or anything else to do with this ECG, are you able to draw any inferences with respect to either CPVT or Long QT Syndrome?
A. No, I would say it was not from that ECG.

Q. I've already asked you about CPVT. In your view has Long QT Syndrome been excluded in Kathleen Folbigg?
A. I think to the best of our ability and understanding it probably has. She has a normal QT interval at rest and I did not feel there was evidence of significant QT prolongation in the recovery period from exercise based on the measurements I made from the exercise test. So based on everything we've seen so far, again I would not make a diagnosis of Long QT Syndrome in her situation.

Q. You wouldn't make a diagnosis but would you positively exclude it?
A. I think on everything we have to date you can exclude it, yes.

Q. If she was your patient, would you recommend further testing?
A. Yes, there were other things that we can do. Obviously she has a history of syncope, so has fainted in the past. That's always something of concern as a cardiologist. So, we could do further monitoring over a longer period of time, either using patch monitors for several days or ideally an implantable loop recorder which would monitor the heart rate and rhythm at the time of any symptoms. Something we do on a very common a - treatment basis. I don't think there's anything else we could specifically do or that I would do with regard to Long QT Syndrome. Other tests have been used though that are not something I specifically use and I specifically refer to challenging the patient with epinephrine, an infusion, I don't do that. So I feel reasonably comfortable excluding Long QT Syndrome on the basis of the exercise test, and obviously as I said before, our experience regarding Long QT calmodulin, primarily because the vast majority of patients described to date are too young to perform an exercise test, so our understanding is still relatively immature, but basically at the moment I would exclude Long QT Syndrome in her case, yes.

Q. Can I ask why you don't use epinephrine tests?
A. When it was used earlier on we did try it. In my personal experience the false positive rate was quite high, so I felt much more comfortable performing an exercise test, it was just something that I felt better interpreting.

Q. Professor, we haven't previously asked you this directly, so if you're not able to answer immediately that's fine. Has Ms Folbigg been tested for Brugada Syndrome?
A. I'm not aware that she has, no.

Q. What would that testing involve?
A. Classically that testing involves using a drug. Depending on where you are in the world would depend on which agent you use and have access to, which is called a provocation test to try and bring out the classical ECG appearance

of Brugada Syndrome. That could be using a drug such as Ajmaline, or Procainamide, but I'm not aware that that has been performed in her.

Q. From the evidence available to you, would it be possible to exclude Brugada Syndrome in Ms Folbigg?
A. She has no features of Brugada Syndrome that I can detect so far, no. So I could exclude it based on what - on everything we know to date.

Q. You could exclude it?
A. I would exclude it, yes--

Q. You would exclude it?
A. --based on that she hasn't - features of--

Q. Do you consider that even though she hasn't had an Ajmaline test you would consider you have enough information to exclude it?
A. No, I said on the basis of what we have so far, then I think it's reasonable to exclude it, but obviously an Ajmaline test could be very helpful in this situation.

Q. If she was your patient in clinic, with the evidence you have now, would you be comfortable excluding it or would you encourage further testing?
A. No, I think if you wanted to exclude Brugada Syndrome definitively, you would want to perform an Ajmaline test. Again, it's not the perfect test but I think it would be a good assessment as to whether she has any potential predisposition to Brugada Syndrome, then an Ajmaline test would be the best way to do that.

Q. You're aware that Professors Kirk and Skinner downgraded this criteria, BS2 from strong to moderate to account for the possibility of non-penetrant individuals. Are you aware of that?
A. I don't specifically recall that in their report, no.

Q. Do you consider that approach reasonably open?
A. Yes, I think, you know, a lot comes down to how you interpret that exercise test and how you would define expected. You know, I still think whatever you use, however you interpret specific criteria as defined by the ACMG, to my mind approaching this patient and many others there's a significant degree of uncertainty and that would be front and foremost in my mind, that I don't think we have a definitive answer here. So, you know, we've had variants that are much more pathogenic as per the ACMG that ultimately turned out not to be the cause of the disease in a family. So, I think I would keep an open mind, but at the moment, based on the term expected, that's how I classified it, but obviously these things are open to judgments and debate.

Q. Thank you, Professor. My last question on the ACMG criteria, and the product of your assessment is variant of uncertain significance, that places it somewhere between 10 to 90% as I understand it in terms of the likely pathogenic range. Are you able to offer your view with any greater precision about where you would place the variant in that range?
A. I think based on everything we understand about calmodulin, if this was a

clinical case I was involved with, yes, it would be suspicious, because of the nature of calmodulin and what has been experienced to date with this specific gene or with these three genes. So although, you know, I feel at the moment it's a variant of uncertain significance, that could change with further information that's certainly towards the higher end of that range, and I, you know, and many people would argue this is a likely pathogenic variant based on the constraints of calmodulin as I think Professor Watkins did, and I wouldn't necessarily argue with that strongly either way. I think it's definitely something that is suspicious and would require much further investigation and those investigations often take time to identify specific features that would help you determine the causality of a variant in a specific gene.

Q. You've referred both in your evidence this morning and in your report to the very early presentation of the Long QT phenotype in young children. In your report at red page 136 you note that 58% of patients were detected in the perinatal period with electrocardiographic abnormalities. Apart from QT prolongation are there other types of electrocardiographic abnormalities that would be picked up?
A. The classic one is, because it's difficult to measure the QT interval in a foetus, so the classic one is a very slow heart rate, bradycardia, which is often how babies like this are identified. Because the QT interval is very long, that means that the next heart beat is delayed, so the heart rate is slower, and so that is a mechanism by which many patients with Long QT Syndrome are identified. It's something that I would specifically ask for in the clinic when seeing a patient, and certainly the last two patients we had with severe Long QT Syndrome both fell in that group. So that would be something else that would be helpful information.

Q. Would there be a consistent slow heart rate, or would that come and go?
A. There are criteria that have been applied that if the heart rate is low on a significant number of occasions, and I can't remember exactly what that number was, but a sort of scoring system has been proposed, back in April in 2013, that if their - your heart rate falls below a certain range on a number of occasions, then that patient should be evaluated for Long QT Syndrome. So, it seems to be fairly ..(not transcribable)..

Q. And to be clear, on a number of occasions, are we talking about one minute of lowered heart rate in a 24-hour period, or hours of a lowered heart rate in a 24-hour period?
A. So in this situation I'm still referring to the foetus, so this would be referred to at each foetal heart rate scan. So the heart rate would be measured at the scan and if it fell below a certain rate on a number of scans throughout the pregnancy, then that would sort of raise a flag that this could be Long QT Syndrome.

Q. You're not talking about sort of a momentary drop in heart rate, you're talking about at least a more consistent period of lower heart rate?
A. Correct, and it's because the QT interval is so prolonged that the heart rate can't really increase.

Q. The heart rate I take it is the sort of thing that you would expect to be picked up on routine monitoring during pregnancy, the foetal heart rate?
A. Exactly. Yeah, that's part of a routine evaluation of any foetus.

Q. That would have been true in the early 1990s?
A. I couldn't say for certain because I'm not involved in obstetrics, but I would imagine it would be, it's a pretty easy thing to measure, but I would have to defer to people who are involved in obstetric care at that time to be certain.

Q. Page 137 of your report at the bottom, you discuss what's known of Sarah's phenotype. You note that as best that can be determined her death was not adrenergic - would you help me with that one please?
A. Adrenergically mediated.

Q. Thank you. I almost got through it. You make the same observation that her death was not that because she was reportedly asleep in bed?
A. Mm-hmm.

Q. You make the same observation a little later about Laura Folbigg. Could REM sleep trigger the release of adrenalin or other catecholaminergic neurotransmitters?
A. I couldn't say for certain because it's not something that I have an understanding of the basic physiology of. But certainly as it relates to the clinical interpretation we do not think of sleep as an adrenergically heightened state, or rightly or wrongly I wouldn't be entirely sure based on the question you just asked, but certainly we classically see symptoms in the adrenergically mediated diseases. By that I mean in CPVT and some forms of Long QT Syndrome, we see those children and young adults who are active, exercising or in a heightened emotional state.

Q. In relation to Laura Folbigg's phenotype, which you discuss on page 138, red page 138, you note in respect of her single EEG that without access to the original copy, you consider the length of the QT interval cannot be confirmed?
A. Correct.

Q. You then note that data was downloaded on a regular basis via modem from a cardiorespiratory monitor over a 12-month period and considered to be normal and you refer to the report of Dr Chris Seton, who analysed that data.
A. Yes.

EXHIBIT 2-AC AT RED PAGE 5364 SHOWN TO COURT

Q. Dr Seton there notes his final observation at the bottom of the page, "My regular analysis of this data revealed no evidence of a primary or secondary cardiac conduction problem." Is that what you were relying on in your report, that observation?
A. Yes, specifically that observation, yes.

Q. And you didn't take into account the underlying monitor data?
A. That would be outside my area of expertise. I've never interpreted such

data so I wouldn't want to do that.

Q. I hear what you say about it being outside your area of expertise. I might ask you a few questions if you can help us at all in relation to that underlying data.

ROY: Can we have Exhibit 14, tab 9, red page 261.

Q. At the top left-hand I think it says “Corometrics medical systems, Event-link(r) system”. We have the name Laura Folbigg, aged 9 months, and the observation of the physician, "using the belt for the last couple of weeks 26.4." Are you able to assist us with what that would refer to in this context?
A. To be honest, no. Unless that, they're trying to hold the device onto her better with a belt. I wouldn't know quite what they mean by that having never used one of these devices.

Q. If we can turn over the page to 262, this and subsequent pages, you would agree appear to report individual events that were recorded by the monitor?
A. They would look like it, yes.

Q. And if we can zoom into the top left-hand box, the first one. It notes an apnoea start time of 3.11am. An apnoea duration of 34 seconds.
A. Yes.

Q. In the right-hand column it says “Alarm settings - apnoea: 20 seconds”. I'm drawing the inference that if the monitor notes an apnoea duration of longer than 20 seconds it would alarm. Is that how you would interpret that?
A. That seems a reasonable assumption, yeah.

Q. Across to the right there is another apnoea noted of 25 seconds?
A. I see that.

Q. That is 25/97 in the top left-hand corner.
A. Yes.

Q. Suggesting 25 December 1997 in the morning?
A. Yes.

Q. And then the next one listed would appear to be 26 December 1997?
A. Yes.

Q. The box underneath on the left, number 3, is 30 December, although it's partly cut off. It would appear to be 30 December 1997.
A. Mm.

Q. It doesn't note an apnoea start time but notes a heart rate start time?
A. Mm.

Q. And an extreme heart rate of 65 beats per minute?
A. Mm.

Q. Would that be an unusually low heart rate?
A. I think it has to be measured in the context of what I assume is an apnoeic episode there. So, you know, I have absolutely zero experience of interpreting these traces or their associated physiology in terms of apnoea and bradycardia which are well-known to go together in young children and infants, so in that situation I would have to get an expert opinion to advise me.

Q. I'm not going to take you through any more of them. If you take it from me that they show over a period of time a series of apnoea and low heart rates, and I've heard what you've said about not being an expert in this. You have taken the reviewing physician's view that there was nothing in this that concerned him and from his point of view this was normal and indicated normal function?
A. That's what the inference was by me on what he wrote, yes, that there were no alarming features.

Q. If you took these as reflecting regular episodes of apnoea and low heart rate in Laura Folbigg, would that concern you in relation to her state of health?
A. This may well fall well within the norms of physiology for someone of this age, and again I would need someone far more experienced than me on this to tell me what that was. Based on what Dr Seton wrote, I inferred that he felt comfortable that all of these did fall within normal human physiology and were not of concern to him.

Q. And episodes of apnoea are not specifically associated with Long QT or CPVT?
A. So, if you have a sustained arrhythmic event you will become apnoeic, obviously because you won't breathe due to ventricular arrhythmia resulting from Long QT or CPVT. But apnoea is also very common in young infants. I would infer that from what Dr Seton wrote these were again normal for someone of that age.

Q. Had you otherwise reviewed those reports?
A. Which ones?

Q. The ones that we just did have on screen, the traces.
A. The traces.

Q. And were you able to interpret the traces in any meaningful way?
A. No, because I have never seen those before so I wouldn't exactly know what those mean.

Q. They don't show you heart rhythm?
A. Not heart rhythm, no.

Q. Coming back to your summary of Laura's phenotype in your report, which is red page 138, in respect of her resuscitation ECG, you note a wide complex very slow ventricular escape rhythm that was documented and you described this as a non-specific finding. Can you explain that?
A. So, by the time I think that was recorded, she had been having CPR for

some considerable time, or cardiopulmonary resuscitation for some considerable time. And so this is what I would interpret as something of a terminal event related to heart function, so there was occasional spontaneous electrical activity. Whether that resulted in any contraction or not is unlikely or uncertain. But it looks like a very terminal event based on the rate and how wide the QRS complex is, reflecting a very, very sick and diseased heart at that point.

Q. To be clear, is it possible from that rhythm to exclude the possibility that there had previously been a ventricular fibrillation?
A. You can't exclude it, no, from that. Certainly not.

Q. You've expressed a very firm view in respect of the importance of phenotype as the starting point for a genetic analysis and the great difficulty that presents in this case, where the phenotype picture is limited. We've covered it in a few different ways, but I want to understand as clearly as possible whether you consider there is sufficient evidence to exclude the presence of a phenotype in this family or whether you consider the picture ambiguous.
A. I don't think we can exclude it because the phenotype in two infants could be just a cardiac arrest and sudden death. That's possible. But obviously a cardiac arrest has many aetiologies. So, we certainly see families where that is the situation, so that's possible. But ultimately what we're looking for in any family that we investigate is more definitive evidence of a clinical phenotype associated with a specific genetic finding to help us understand what that genetic finding means, and ideally doing that in the context of a wider family to evaluate that in multiple different individuals within the construct of the family.

Q. At the bottom of page 103 of your report in the second last paragraph, second sentence, you note, "Although IVF could present as sudden death with no prior symptoms, no calmodulin variant has been identified in 919 infants under 12 months of age who underwent genetic sequencing after sudden death." Can you explain why that's a relevant observation?
A. These were two studies where I took the combined data. One specifically was looking for the presence of calmodulin variants in sudden infant death and another was looking at all causes of sudden infant death from a genetic perspective to help understand. But there were no calmodulin variants I could identify in either of those studies. That could mean that it's sufficiently rare that there just wasn't one by chance. But I don't think from what we know from calmodulin we can say it doesn't cause sudden infant death, it just didn't in those individuals. So, whether the majority of very young infants seem to survive diagnosis in the current era is possible as well. And I don't know how historic some of those patients were, but it was a - based on what we know about calmodulin you could say it was maybe atypical or a bit surprising that there wasn't a calmodulin variant in that study or in either of those studies.

Q. Based on the apparent variety of calmodulin variants generally and the rates at which they appear in, for example, the gnomAD database, even if they were present at a multiple of the background population rate inferred from the gnomAD database, would you necessarily expect a single case to appear in a

cohort of about 900 infants?
A. So, if you think there's approximately 150,000 people in gnomAD contributions to that data and I think there were maybe less than 20 benign variants, from a mathematic perspective it's possible that there weren't any either benign or causative variants in that cohort of 919 patients.

Q. Is it meaningful having regard to the low rates of CALM variants to note the observation of the absence in that cohort?
A. I think it was interesting but I don't have a specifically good explanation as to why they weren't there. You could argue that a cohort of patients with sudden death could be enriched for calmodulin variants because we know it's a lethal genetic variant, so it was a little interesting that it wasn't there but I can't give you a specific reason as to why that may or may not be.

Q. In the subsequent study of three cases out of a total of 258 cases, that would be significantly greater than the background expected rates in the population, is that right?
A. Yes, and those were older cases, older patients who died suddenly with, I believe in all cases the cardiac arrest was triggered adrenergically or - and I think all those three were probably included in the Calmodulin Registry from what I could understand between the two.

Q. On page 139 of your report in the middle of the page, you note that CPVT appears not to manifest under two years of age for reasons that are not well understood. I obviously take it from that that you've never seen it manifest under two years of age?
A. I have not, no.

Q. Have you otherwise seen very young children of Laura's age experience ventricular fibrillation?
A. Yes, we have seen ventricular fibrillation at a very young age.

Q. Have you seen that in the absence of structural abnormalities?
A. Yes.

Q. Is that unusual?
A. It's very rare, but it exists.

Q. Finally you note in your report that you would not consider sudden death a phenotype as it is non-specific and if it were--
A. Yeah.

Q. --used here it raises the issue of non-segregation. Can you explain that?
A. So I think if you - I mean if you are using purely sudden death to say this is a part - definitively a part of an arrhythmia syndrome such as Long QT or CPVT, without any other evidence, it could be that there's no supporting evidence from an ECG or any recordings to prove that either way. So, I focus very much in this paper on the two girls, but obviously there were two other sudden deaths in the family and non-segregation means that those two boys did not have the calmodulin variant. So you therefore can't infer the

calmodulin for all four.

Q. So, by reference to the boys, that's, you've not included sudden death as a phenotype in your analysis?
A. Can you repeat that sorry?

Q. I'll do it differently. In forming the views that you have, have you taken into account the fact of Caleb and Patrick Folbigg's deaths?
A. No, I've - I mean not in any great detail, I focussed very much on the role of calmodulin in the two girls.

Q. You said not in great detail, are they relevant at all to your assessment?
A. My assessment was very much focussed on the role of the calmodulin variant, but obviously it is important when, you know, the family as a whole to factor those in, although I didn't really spend much time on that, there are no other genetic variants invoked in the two boys, which were way outside my area of expertise, but obviously you have four children with a similar outcome of sudden death, two of whom have the calmodulin variant and two of whom do not. So from the calmodulin perspective it's very difficult to say this is a cause of everything because you have two individuals who do not carry the variant. So that would be how I would find on the segregation.

Q. The fact that the calmodulin variant would not explain all four deaths, has that influenced your conclusion that the calmodulin variant was not likely to be responsible for the death of the girls?
A. No.

Q. I'd like to explore a couple of hypotheticals with you.
A. Mm-hmm.

Q. I'd like you to assume that this family came to see you while Laura was alive, around a year old, but all other features of this case remain the same, including the deaths of the two boys who are not carriers, and Sarah's death who was a carrier, and you're aware that Laura is a carrier. First, from what you've already said, I assume it goes without saying that you would be seeking to have Kathleen and Laura further tested?
A. Correct.

Q. Would that include an implantable recorder for Laura?
A. Yes.

Q. Assuming that for whatever reason there was some delay before any of the additional testing could occur, perhaps the family was located remotely, would you be advising her parents to look for signs in relation to arrhythmia?
A. Yes, we would recommend or I would recommend strongly that the family were trained in basic life support, had an automated external defibrillator in the home and were aware how to use it. That's something that many families would adopt, particularly if they live in a more remote area where health care is further away.

Q. Would that be only because of the deaths of the prior three children or would it also be because of the calmodulin variant?
A. No, I think it would be primarily driven by the prior family history, but obviously you have a very suspicious variant in the calmodulin variant in Laura, you have some discordance between the phenotype of the first girl who died with the calmodulin variant and her mother. That has been eluded to by many of the other experts that's very common in inherited heart disease, so that's not in any way unexpected or strange. But then you would really want further information as to what this variant was doing and that would be through clinical monitoring through repeated evaluation, repeated ECGs, echocardiograms, et cetera. So I think if this situation arose in the current era, then someone will be monitoring very, very carefully in clinic on a regular basis, then we would have the benefit of an implantable loop recorder which automatically transmits any events direct from the home to the hospital. So, we would get it almost in real time.

Q. Continuing the hypothetical, assuming that Laura Folbigg did then pass away, in the circumstances of which were described in this case, if this family then consulted you about making reproductive decisions with respect to a fifth child, would you encourage them to screen their embryos for G114R?
A. I think it would to some degree depend on the evidence you had related to Laura passing away. If we go back to the question you've just asked me, did she have an implantable loop recorder that was in when she passed away, that's going to give you very clear evidence as to whether this was arrhythmic or not. So I think if it was arrhythmic then you would strongly advise that that would be a good idea. Not everyone goes forward with that, it involves quite complex in vitro fertilisation, so some families elect not to do it, some families elect to do it, but it's certainly something that's an option for many families these days and requires counselling from people who are much more - than me, but we do often refer families for that, yes.

Q. Assuming that you had all of the information that you have now, so you don't know, you don't have a recording of her death, would you still advise them about screening of their embryos for G114R?
A. I think there will be many other factors that you wanted to include in that overall picture, particularly the likelihood of success of effectively IVF in this situation, and their desire to have another child. Some people would infer that their desire to have another child was so great they would accept the risk and not go forward with IVF or pre-implantation genetic diagnosis. Other people would want to definitively exclude that. I think many of these decisions become very personal about the family concerned rather than any robust medical advice and that's the way I typically approach situations such as this.

Q. Would I understand you to be saying that you would discuss the fact of G114R with the family and discuss their options in relation to testing?
A. Yes.

Q. Professor, do you consider Laura's myocarditis would've increased risks posed by the G114R variant?
A. I think if we - you know, if - I think myocarditis can cause life threatening

arrhythmias in its own right, so I know that's been extensively discussed and I'm not an expert pathologist, so I won't go into that further, but obviously anything that can increase inflammation within the heart, you could reasonably infer could have a proarrhythmic effect.

Q. So yes, potentially?
A. Potentially yes, potentially, yeah.

NO EXAMINATION BY DR WOODS

<EXAMINATION BY MR JORDAN

Q. Good evening, Dr Abrams, can you hear me all right?
A. I can hear you very well, thank you. Good evening.

Q. Thank you. I won't take much of your time. Earlier in your evidence you confirmed that you had received later reports from some of the other experts provided to you after you had completed your own report, do you recall that?
A. Yes.

Q. I want to ask you whether there is anything in any of those later reports that you would now like an opportunity to respond to?
A. I think there were many points that were valid and well made, obviously from multiple experts in the field for whom I have enormous respect. I don't think that anything that I read specifically changed my opinion in great detail and I still feel that we have a lack of phenotypic evidence in a variant that's suspicious but unproven and uncertain. I felt there was a lot of, obviously, discussion around that point by other experts but I don't think in everything that we understand from the clinical picture related to the family I wouldn't fundamentally change what was said in my report.

Q. Are you aware that only very recently Professor Schwartz has provided the Inquiry with yet another report from him dated 20 February 2023?
A. I don't believe I've seen that one.

JORDAN: At that, I'll just leave it at a matter for your Honour and those assisting your Honour as to whether it's appropriate for Dr Abrams to be given an opportunity to respond to that most recent report from Dr Schwartz.

JUDICIAL OFFICER: Thank you, Mr Jordan.

JORDAN: Thank you for your time.

NO EXAMINATION BY MS HORVATH, MS LOVE, MR HASTINGS AND DR WATERHOUSE

JUDICIAL OFFICER: Thank you, Professor, I'm glad we didn't keep you for as long as we anticipated at the start. Thank you very much for your time.

WITNESS: My pleasure, thank you. Bye.

<THE WITNESS WITHDREW

AUDIO VISUAL LINK COMMENCED AT 10.28AM

ROY: Your Honour, I have one matter, a tender of a supplementary report from Dr Cala, who describes the circumstances in which he took and examined new slides in relation to Laura Folbigg's heart. The proposed tender would be Exhibit 13, tab 04A. That has been circulated. We are otherwise commencing at 2pm with--

JUDICIAL OFFICER: Wait. Mr Jordan, are you suggesting that we should supply Professor Schwartz's latest report to Dr Abrams?

JORDAN: Your Honour, I think maybe it wouldn't go quite so far as a suggestion, just I'm raising it for your Honour's consideration in circumstances where there is this difficulty where there's inevitable to and fro as reports are provided.

JUDICIAL OFFICER: There has to be an end to it somewhere, that's the difficulty I have.

JORDAN: Yes, I accept that, and that's why I'm--

JUDICIAL OFFICER: If I supply Professor Schwartz's report to Dr Abrams, presumably it would have to be supplied to every other expert in the field, who would no doubt then respond. That's the difficulty I have. I will consider it but at the moment I'm inclined not to unless someone specifically asks me to.

JORDAN: Yes, your Honour. I just also note this, while treading softly on the issue, part of the reason I raised that is that Professor Schwartz has, it would seem, again, provided more in this latest written report than he indicated he would provide when he gave his oral evidence.

JUDICIAL OFFICER: That's undoubtedly correct.

JORDAN: That perhaps just highlights the problem.

JUDICIAL OFFICER: Yes, thank you.

ROY: Your Honour, we are otherwise scheduled to commence at 2pm with Dr Matthew Orde, that is by AVL. Then Dr Cordner, he's also by AVL at 3pm.

JUDICIAL OFFICER: Yes. The Inquiry will adjourn until 2 o'clock.

LUNCHEON ADJOURNMENT

AUDIO VISUAL LINK TO CANADA COMMENCED AT 2PM

MATTHEW MILBURN ORDE

Yes, Ms Callan.

CALLAN: Your Honour, before we resume with the evidence, and I note that Dr Orde is in the witness box as it were by AVL, Mr Jordan for the DPP has indicated to those assisting your Honour of an objection to the report of Dr Garstang, and if it's convenient, perhaps that objection can be dealt with at a suitable time later this afternoon.

JUDICIAL OFFICER: Yes, all right, we'll deal with it if convenient after the conclusion of Dr Orde and Dr Cordner's evidence.

CALLAN: Yes. If we find that there is sufficient time in between, then that might commend itself.

JUDICIAL OFFICER: Yes.

Q. Dr Orde, good morning or good evening as the case may be. Will you take an affirmation?
A. Yes. Good afternoon to you.

<MATTHEW MILBURN ORDE, AFFIRMED(2.06PM)

<EXAMINATION BY MS CALLAN

Q. Could you tell us your full name?
A. Yes. Good afternoon. My name is Dr Matthew Milburn Orde, that's spelt O-R-D-E.

Q. It's the case that you are a Specialist Forensic Pathologist?
A. That's correct, yes. Currently working and based in Canada.

Q. You work currently as a Consulting Forensic Pathologist as part of Canadian Forensic Medical Consulting, currently contracted to provide autopsy services to the Saskatchewan Coroners Service in Canada, is that right?
A. That's correct.

Q. You're also a Clinical Associate Professor at the Faculty of Medicine of the University of British Columbia and an Associate Professor--
A. Yes.

Q. --in the College of Medicine at the University of Saskatchewan in Canada?
A. That's correct.

Q. It's the case, isn't it, Dr Orde, that you received an engagement by way of letter of instruction from the legal representatives for Ms Folbigg?
A. That's correct, I did.

Q. And you were asked to review material provided to you and express your opinion as to cause of death of each of the four children?
A. In essence that's correct, yes.

Q. That gave rise to a report in which you set out your views dated 21 December 2022?
A. Yes.

CALLAN: Your Honour, for the record that's at Exhibit 13, tab 5.

Q. Dr Orde, in respect of your background and qualifications, are they set out in an up to date curriculum vitae which is annexed to that report?
A. Yes. Yes.

CALLAN: Your Honour, for the record that commences at red page 109.

Q. I am sorry, I interrupted you, Dr Orde?
A. No, that's okay, I wasn't going to say anything.

Q. We see there your detailed curriculum vitae including your education and qualifications, your employment history and your research and publications?
A. Yes.

Q. I'll take you to it in more detail in a moment but is it the case that you prepared a supplementary report dated 12 January 2023?
A. I did.

CALLAN: That, your Honour, is to be found at Exhibit 13-06 commencing at red page 124.

Q. As I said, I'll come to it in due course, Dr Orde, but that supplementary report specifically addresses the circumstances in which you came to inspect microscope sections of Laura Folbigg's heart tissue and your views upon conducting that examination?
A. That's correct.

Q. In relation to the material with which you were briefed, you list that in your report commencing at paragraph 11 - which, your Honour, is--

JUDICIAL OFFICER: First report.

CALLAN

Q. --first report I should say, page 3 of the first report, red page 72?
A. Yes, I do.

Q. At paragraph 11 you list the letter of instruction and various reports and other material with which you were briefed?
A. Yes.

Q. At paragraph 12 you advised that in 2018 you say, in preparation for contribution to an ABC television documentary you had the opportunity to personally review microscope tissue sections prepared from Laura Folbigg's heart?
A. Yes, that's correct.

Q. Can I ask, what were the circumstances in which you came to make comment to the ABC television documentary in 2018?
A. Sorry, could you repeat the question? I didn't quite catch all of that.

Q. How did you come to make a contribution to that ABC television documentary in 2018?
A. You'd need to ask the producer of that program really, but I think in essence I was a recognised Australian forensic pathologist who had recently moved overseas, who knew a little bit about the background of this case and therefore was probably quite well placed to provide some independent commentary.

Q. In terms of what you knew about the background of the case, was that gleaned from publicly available information?
A. Yes, I mean there's general conversation in the forensic pathology community about cases of interest, and this is one such case. I can't recall any specifics. It wasn't something which I was overly familiar with, I was

simply aware that there was a case involving some issues involving myocarditis, but not a great deal more than that at that stage.

Q. To be clear, having regard to your employment history, have you worked as a forensic pathologist in Australia?
A. Yes. Between 2005 and 2013 I worked as a Staff Specialist and in the latter years a Senior Staff Specialist Forensic Pathologist in the Department of Forensic Medicine in Sydney based at Glebe.

Q. In the course of your work at the Department of Forensic Medicine at Glebe over that period of time, did you have access to evidence or reports in relation to the, if I can put it generally, the Folbigg matter?
A. Well, I guess I could potentially have had access to, but no, I don't recall ever looking at any materials relating to this case whatsoever.

Q. The documents that you were furnished that you list at paragraph 11, to the best of your recollection when they were furnished to you for the purposes of preparing this report, that's the first time you'd seen that material?
A. The majority of this material, yes. Obviously in preparation for the television documentary in 2018 I did undertake some research myself reviewing various documents that were available in the public domain. I was also - given some further documents by the producers of that documentary. I remember I was given access to a report prepared by Professor Cordner which I believe was prepared by him in 2015.

Q. Yes. As I understand it, as described at paragraph 13 of your first report, during the preparation of your first report, you accessed some further documents from the Inquiry website and they're listed there?
A. Yeah. Yes.

Q. In your report, when you address Patrick's death, if I could take you to that. It's page 32 of your first report, red page 101.
A. Yes.

Q. At paragraph 114, you refer to, "contemporary clinical opinion appears to favour this", that is, the epilepsy, as “having been due to an ongoing encephalopathic process rather than a discrete hypoxic-ischaemic cerebral event"?
A. Yes, that's my understanding, based upon review of the literature which I undertook, and it's the - I'm referring here not just to the epilepsy, per se, which is a consequence arising from his brain condition, but the underlying brain condition itself, which first manifested on 18 October 1990.

Q. When you say contemporary clinical opinion, what are you referring to?
A. The reports prepared by - if I can just refresh my memory, please. Professor Ryan, I recall, and the other report was prepared by - excuse me - I believe it was Professor Fleming.

Q. You provide a footnote, coming back to paragraph 114 of your first report, as to the value - you say there may be value, this is footnote 72, in seeking the

additional input of an expert paediatric neuropathologist.
A. Yes.

Q. As I understand it, you refer to the opinions which have been expressed by Professor Fleming and Dr Ryan in support of what you state at paragraph 114.
A. Yes.

Q. In that regard, that reflects the recognition you give to their areas of specialisation, which fall beyond yours, as a forensic pathologist?
A. Yes. I think there is some overlap here that, clearly, it's a pathological condition or a disease in Patrick's brain which could be assessed by a pathologist following autopsy. I didn't have access to the original materials, I didn't have access to the slides, I didn't examine the brain and so on, so, really, the best evidence I had to try and identify the cause or the causes of Patrick's brain condition was really based upon the clinical evidence and, therefore, I would certainly defer to these experts, rather than my own independent opinion in this regard.

Q. Can I turn to the topic of Laura's myocarditis.
A. Yes.

Q. At page 14 of your first report, red page 83, do you see at paragraph 50 you refer to photographs of the microscope tissue sections of Laura's heart in Professor Cordner's report and, it seems, set out again in the Brohus article. You then describe what those photographs of the microscope tissue sections appear to demonstrate. Do you see that there?
A. Yes, I do.

Q. Do you regard photomicrographs as equivalent to inspecting slides for the purposes of forming a view as the presence and extent of myocarditis?
A. No. The photomicrographs provide a quite localised snapshot, and it's all down to the issue of sampling. I mean, the person taking the photographs would have a wide range of view of these tissue slides, but they would have elected to take photographs from just a very small proportion of those tissue sections. So the photomicrographs only give you a small view, in essence, but I do recall looking at the slides myself and I believe they're likely one in the same as examined by Professor Cordner, and these photographs look in keeping with what my recollection of these sides showed at the time.

Q. Is it your evidence that you place primary weight on what you saw when you inspected the slides in terms of your view as to the presence of myocarditis?
A. Yes, I formed quite a strong opinion when I examined them in 2018, and I recall doing that quite clearly, but what I can say is that the photomicrographs really support that opinion which is in my brain.

Q. Can I turn then to the circumstance in which you came to view those slides in 2018, as addressed in your supplementary report, Exhibit 13, tab 6, red page 124. You say at paragraph 2 that you're unable to find any notes documenting your observations, or any captured photomicrographs, and so the

comments in that supplementary report you've made are from memory alone.
A. Yes.

Q. Your recollection is that you were provided with several slides for examination in around 2018, and--
A. Yes.

Q. That's the first sentence of paragraph 3. First sentence of paragraph 4, "I believe these slides were shipped to me by Professor Cordner". Where were you located at the time?
A. I was at that stage working at the Vancouver General Hospital in British Columbia.

Q. Had you had any communication with Professor Cordner before the slides were sent to you?
A. I don't recall the specifics. Undoubtedly, I'm sure there would have been some brief communication between the parties just confirming the address and so on, but I can't think of any other in-depth conversation, no.

Q. By the point you had the communication with Professor Cordner, had you been requested by the ABC to make some contribution to the documentary they were making?
A. I can't recall the exact order in which events unfolded. I think it's likely that I would have been invited to contribute to the documentary by ABC and then I would've followed that with communication with Professor Cordner with a view to receiving those slides for examination.

Q. I recognise the passage of time and you don't have contemporaneous notes, but was it you who sought out slides for examination?
A. I must confess, I can't remember that specific detail.

Q. In any event, you received several slides for examination, you recall--
A. Yeah.

Q. --in or about March 2018?
A. Yes.

Q. You say at paragraph 4, it seems likely the slides would have been the very same sections examined by Professor Cordner and referenced in his report. Do you say that because of the photomicrographs attached at the appendix to Professor Cordner's 2015 report?
A. That's part of it and, as I said before, the photomicrographs are simply reminiscent of the image in my mind of having looked at these slides myself, so I think there's some definite similarity there, which could be explained by them being one in the same set of slides. The other thing which I recall is that Professor Cordner in his report describes examining seven slides, and I use the word "several" to indicate around about, you know, five to eight, nine, something like that, sort of, number of slides. So the number again seems compatible. So putting everything together, yes, it seems likely to me that the slides I examined were one and the same as those examined by Professor

Cordner.

Q. But you have no way of determining now whether they were the same or were newly cut slides, for instance, from the blocks?
A. Firstly, no, I don't. I do remember Professor Cordner indicating to me that the slides were valuable and that they needed to be treated with care, so from that I would infer that they were likely the only copy he had, but that may be incorrect. I could also advise that the personnel at the Victorian Institute of Forensic Medicine in Melbourne may have access to data relating to which slides were shipped to me, which could probably confirm the answer to your question.

Q. In relation to your views, having regard to the slides, you set them out at paragraph 11 and 12 of your supplementary report, red page 126?
A. Yes.

Q. You, beginning of paragraph 11, first sentence, state, "this was an unequivocal case of potentially significant lymphocytic myocarditis, with clearly identifiable associated myocyte damage", and then you go on to use a colloquial expression, but over at paragraph 12, red page 127, you recognise, "absent the application of a formal inflammatory scoring protocol, any descriptors applied will necessarily be somewhat subjective".
A. Yes.

Q. The way you choose to describe it is, "somewhat patchy and variable in its intensity".
A. Yes.

Q. Overall, “could be described as ‘moderate’ - though with some areas of lesser inflammation and some with more".
A. Yes.

Q. Your recollection is there were features of myocarditis present in each and every section of the heart tissue which you examined.
A. Yes.

Q. Recognising what you've already said about the descriptors being somewhat subjective, can I take you back to your first report at page 34, red page 103. At paragraph 123, you state your opinion is, "what appears to be quite florid and widespread acute myocarditis".
A. Yes.

Q. Do you stand by that description, having regard to the way you described it in your supplementary report?
A. I do, with the proviso that the terms used here don't indicate any specific degree of pathological reliability. They're not intended to be objectively quantitative. They are more subjective words, which to me indicate to the reader the extent and degree of information present in these slides, and at the point in this first report of mine, the use of the word "florid" here was to try and convey to the reader that the appearances in the microscope slides were to my

eye quite striking and readily apparent, as being those of myocarditis of a degree of severity, which I think would undoubtedly have posed a risk of sudden death.

Q. Your use of the word "florid", you intended to convey "striking and readily apparent".
A. Yes.

Q. And when you said, "widespread", just reconciling that with your description in your supplementary report at paragraph 12 of being "patchy and variable".
A. Yes. I think all of the descriptors apply.

Q. You didn't mean to convey anything - when you said the description, "widespread", you don't regard that as different to describing it as "patchy and variable"?
A. Well, this is really an issue of semantics and, as I've tried to stress in the answer to this discussion, it's difficult for me to - use precise terms to convey exactly what was present in the slides. I haven't quantified using any scoring protocol the amount, degree and extent of inflammation present, so these words, as I say, are somewhat vague and subjective. If you look at the papers describing myocarditis and how descriptors ought to be applied, one of the key papers describing the so called Dallas criteria suggests that the degree of inflammation could be scored as mild, moderate to severe and that the extent or spread of inflammation could be described as that of focal, confluent or diffuse. So applying those terms, I would say that the degree of inflammation was on the whole moderate but with some areas with more intense inflammation, other areas with less intense inflammation. As to the coverage of the inflammation in the slides, it's difficult to give a definitive answer to that because the inflammation was certainly present in each and every slide I examined so on that basis it could be considered diffuse, but, as I say, within each slide the amount and extent of that inflammation was somewhat variable. So, using the terms suggested in the paper describing the Dallas criteria I would probably use the words “confluent” to “diffuse”.

Q. The criteria that you've just referred to, Dr Orde, you've extracted a reference to that at paragraph 7 of your supplementary report?
A. Yes, I have.

Q. That is as to the Dallas criteria which defines myocarditis?
A. Yes, and also in paragraph 10 of the supplementary report where I talk about the specifics regarding the grading of the extent and degree of inflammation.

Q. Yes?
A. But they're drawn from the same source in essence.

CALLAN

Q. I want to take you to two descriptions that have been given to Laura's myocarditis by Dr Cala.

A. Yes.

CALLAN: The first was given during evidence in the 2019 Inquiry. For the record in that Inquiry it is transcript page 198, red page 201 - I'll just get up the reference - your Honour, Exhibit 3 is the recording, Exhibit 4 is the transcript. It is 20 March 2019 which is day three of that Inquiry.

Q. Dr Orde, can you see that on the screen there?
A. It is very small, I don't know whether you can make the print any larger, can you?

Q. We're going to take you from line 14 through to line 30, if we can zoom in on that.
A. If I may transfer this from one screen to another at my end, which means I'll be looking away from the camera, but if you bear with me this makes it easier for me to read.

Q. Thank you. Are you able to read that, Dr Orde?
A. Yes.

Q. Do you see there, when asked Dr Cala says, "I've described it", and I want you to assume that's the myocarditis as he observed it in the slides that he looked at, "as moderate, up to moderate"? He recognises he had said in certain of his previous reports "light and patchy", and he refers to moderate and the suggestion that there might be a discrepancy. Then he goes on to say:

> "What I say is that in areas of examination of the heart, in particular the left ventricle, the inflammatory infiltrate was light and patchy, in other words, it was small in amount, a small number of lymphocytes aggregated around the cardiac cells but it was accentuated in areas as I have described it, in portions to the middle of the left ventricle to put it up to moderate, to put it up maximally to moderate intensity."

Do you disagree with that as an available description of the myocarditis in the slides you saw?
A. On the whole I think the terms Dr Cala has used here are reasonable. Again, stressing that any descriptors are necessarily somewhat subjective, but yes, I think the terms he's used here are reasonable. He simply seems to focus on the word moderate here, which is the word I had chose to apply myself in the supplementary report I prepared. If I may, might I attach a little commentary here myself in relation to the grading of the myocarditis?

Q. Yes, of course, Doctor.
A. I mean from the perspective of a forensic pathologist who is trying to gauge whether or not the appearances in a microscope slide, microscope slides to provide a potential cause of death, the question is, is the extent of myocarditis enough to potentially account for a death. It's not really is this mild, moderate, severe, is it patchy or extensive and so on. That really doesn't have any real bearing on this. What I can say to you is that in my experience based on the

reading I have done, my literature reviews in the - my experience as a forensic pathologist, that the extent and degree of inflammation present in Laura's heart slides certainly to my mind provided a very compelling potential cause of death. I think if you look at the literature Dr Cala himself appears to agree with that contention in as much that he states, I believe, that in certain circumstances he will be prepared to give myocarditis as a cause of death. So I think we're all centrally in agreement that the inflammation in the slides does amount to a potential cause of death, and to me that's key.

Q. You say at paragraph 13 of your supplementary report, second sentence:

> "The degree and extent of cardiac inflammation associated with this disease, together with the presence of identifiable damage to heart muscle cells, would in my opinion provide a highly plausible explanation for her untimely death, either independently or in combination with other factors".

That is Laura. What are the other factors that you're referring to?
A. Any other factors that may exist. So I'm speaking here in very general terms and the issue is, as I tried to elude to in my first report, was that it's quite difficult to identify the cause of death at autopsy in many instances. In many cases what we identify are one or more potential causes of death and then the potential causes have to be weighed up in the context of the circumstantial information under autopsy findings, toxicology results and so on. So we need to look at the big picture. So, in this sentence here I'm trying to say that just looking at the slides in isolation, these findings provide a very good potential cause of Laura's untimely death. What we can't say as forensic pathologists by just looking at the slides is that that is definitely the cause of death because of course we don't know what else may have gone on. And in these deaths here, I guess the clear issue before us now is was smothering an issue, and that's not really something that we can explore easily, in relation to this, in this sentence here all I'm trying to draw the attention to in the sentence is that on the basis of the slides I see a potential cause of death but I can't exclude other factors having contributed.

Q. On that topic, could I take you in your first report to page 36, paragraph 134, red page 105?
A. Paragraph 36 was that?

Q. Sorry, page 36 of your report, paragraph 134?
A. Apologies. Yes.

Q. Not at all. There you stress, "if the common understanding – that … 'asphyxial' deaths can occur without identifiable evidence at autopsy is … accepted, it logically follows that one or more of these children *could* have been smothered, but not positively detected at autopsy"?
A. Yes.

Q. With that in mind, can I take you to paragraph 136 over the page, red page 106, where you refer to the absence of any identified injury in the facial regions

of any of the children being notable? In arriving at the views that you have about the cause of death of these children, Doctor, did you regard that as it were negative finding?
A. Absolutely. I think certainly I'm aware of the repeated reference in the literature of the alleged fact that children can be smothered without evidence being visible at autopsy, and that's often accepted as gospel, but really the evidence base for that I think is quite weak and there are certainly cases out there which indicate that asphyxia can be a factor and it can be essentially invisible to a pathologist at autopsy. Certainly it's possible. But if we look at these children here, they are of differing ages, Sarah and Laura would I believe have had teeth at the time, which would provide a hard under surface if the lips were pressed against them, which would therefore increase the probability of injury. There's also some evidence to suggest that children, when asphyxiation is attempted, vigorously struggle. So certainly applying those - those data items to this discussion, I think the negative finding in these cases, that is that there were no signs of forcible smothering, is I think quite persuasive evidence that smothering did not occur, at least in the older of these two children.

Q. In part of your answer there, Doctor, you recognised that the evidence base is weak. That is in support of the common understanding, if I can describe it that way to use your words at paragraph 134, that asphyxial deaths can occur without identifiable evidence at autopsy. In relation to the weakness of that evidence base, do you accept that is in circumstances where this is a topic that's not capable of experimentation?
A. Of course, that's always the problem with forensic pathology, we can't experiment on young children.

Q. You said that there was literature which suggests that children would struggle. What's the literature you're referring to there?
A. There is a paper describing video recorded instances of alleged smothering by carers in a UK, I believe emergency department. Certainly that paper provides descriptions of the children's responses to alleged asphyxial events. That's certainly one factor, but, as ever, in forensic pathology when the evidence base is somewhat thin or lacking, we have to also refer to our physiological understanding as physicians who've examined individuals to our life experiences, it clearly follows to me that if one attempts to forcefully smother an individual, there will typically be a struggle that will ensue. The--

Q. I'll just pause you there. Just to be clear, the literature that you're speaking of of the covert video surveillance, that's reported in the article of Samuels and others from 1992 referenced in Professor Fleming's report?
A. Yes, I believe I referenced it in my own report, too, somewhere. I'm just struggling to find it. Yes, I think it was in Professor Fleming's report.

Q. I interrupted you. In terms of what you went on to speak about in terms of the mechanics involved in a suffocation, can I ask you to confine yourself to the position of an infant or child under the age of two, just to make it a pertinent application to the present case.
A. Yes, of course. All I was attempting to say is that our own life experiences

and my and other doctor's experiences as physicians who've interacted with young children would suggest that if we accept the premise that smothering wouldn't be an instantaneous event, that is, it will take a while to smother a child, often some struggling will ensue between the smotherer and the child victim. The amount of struggling will depend upon the rapidity of the smothering event, and also the physiological build of the child and the circumstances in which that smothering occurs, but I think it's clear that certainly - it would likely be significant struggling, which may give rise to the possibility of injury to the face, certainly to the lining of the mouth, certainly in an individual with teeth, which gives rise to - heightened rise to the possibility of injuries to the mouth and face.

Q. You accept that the potential for injury depends on a number of variables you've described, including the mechanism and, as you've said, the physiological build of the people involved, rapidity, and potentially other factors?
A. Yes, of course, and I'm not intending to be precise here. I have no hard data to support this conclusion. All I'm saying is it makes sense, on the basis of my and other physicians' experience of interacting with children, with some understanding of the process of asphyxiation, that a struggle would typically ensue, and that struggle would, I think, give rise to the possibility of injury to the face, notably to the inner aspect of the lips, especially when the individual involved has teeth.

JUDICIAL OFFICER

Q. Can I just ask you something about that, Doctor. In your first report at paragraphs 96 and following, you refer to a series of articles, which on one view are inconsistent with the proposition you're just putting?
A. Yes, your Honour, it does seem that--

Q. Those articles were written, I think, one in 2016, another in 2021, and they reach a somewhat different conclusion than Professor Fleming reached on his investigation. I'm just wondering why, in light of what you just said, it seems generally accepted by pathologists that smothering can be extremely difficult to detect.
A. Yes, I think there's certainly some truth to those statements, but these statements, I think, are often given validity by the extent to which they're repeated and, I think, undoubtedly, in the case of young infants who are unable to put up much of a struggle, it certainly would be possible to smother a young infant without leaving significant signs of injury such as could be detected at autopsy. Is that also possible in older infants, in older children? Again, it's a possibility, but I think the probability of such an outcome, that is no injury being identified at autopsy, would reduce as the child becomes older. So it's a matter of degree and extent, and I certainly accept the premises set out in these publications I've listed, but, yes, it is possible, I think, to smother a young child without leaving marks at autopsy, but is that necessarily always the case? And that's the distinction I'm trying to make, that I don't think it already - always would be the case, and certainly in the case of older children, I think one can regard the absence of injury as, in effect, a

positive autopsy sign that perhaps smothering did not take place.

Q. Would it depend to some extent on the degree of force that was inflicted in the attempt to smother the child?
A. Yes, of course. It would all depend upon the circumstances, and unless the event is witnessed, no one would ever know the answer to that. So it's all about extent.

Q. If it was done, for example, in a fit of rage, it would be more likely that injury was shown, as compared to, say, a cold-blooded attempt to smother the child trying not to leave any trace?
A. You would think so, but, again, you know - I think you used the word "violent" there. The use of the word "violent" does conjure up a certain manner in which the smothering would perhaps occur, but, again--

Q. I'll use the word "forcible".
A. Forcible. I mean, "forcible", yes, suggests a certain manner of the smothering, forcibly pressing down against the nose and the mouth and, therefore, that force would, I think, likely increase the risk of injury, but it depends exactly how the force was applied. You know, is this just a hand against the face or hands against the face, is the face down into the mattress, is the face down into the pillow or a pillow on the face. There are lots of variables we don't know the answer to here, but, yes, I think it logically follows that the greater the degree of force, therefore, the greater the risk of injury to the children's faces being identified at autopsy subsequently.

CALLAN

Q. Coming back then to what appears at paragraph 136 of your first report, Dr Orde, insofar as you regarded the absence of any identified injury to the facial region as a negative finding, that necessarily involves making assumptions about the method or mechanism of asphyxia which you would expect would leave some form of physical trace?
A. Only in as much that the converse would also apply. So if I am to accept the premise that smothering can occur without leaving signs of injury, that also imbues into the conversation the means by which that alleged smothering would have occurred. It's exactly the same in the converse that the absence of injuries, perhaps, also suggests that smothering didn't occur, but, again, it's dependent upon the specific circumstances. So either way, I don't think this evidence is conclusive. I'm not suggesting that in any shape or form. All I'm saying is that given the very limited amount of evidence data points we have in these deaths, this is one more little bit of information which maybe persuades me that smothering didn't occur, rather than did occur, but on this basis, can it be certain? Absolutely not. I'm not suggesting that at all.

Q. Just to be clear, doesn't it follow from your recognition of the way the - as it - the argument can go both ways that the preferable position would be to regard this as a neutral finding, because otherwise there are - you're building assumptions into methods of asphyxiation?
A. Well, I don't think the available data enables us to say whether or not this is

simply a - we can take a neutral stance. So I'm trying to set out in my report here, and I'm trying - probably failing badly in front of you today to try and set out, again, my opinion, that, sure, the absence of injury might suggest, if we accept the premises of the literature that children can be smothered without injury, it might suggest that smothering is a possibility. All I'm trying to say is that there's another side of the coin which we also need to consider, that the absence of any injury perhaps suggests the smothering didn't occur. I don't know how much weight we can place to either of those points in this discussion. All I'm saying is it's not a simple one-sided scenario that the absence of injury is entirely given weight and supports - and would support the diagnosis of smothering or other forms of asphyxia. There's another argument that says, "Well, actually, perhaps the absence of injury suggests the smothering didn't occur", but I don't think I can quantify those arguments meaningfully to you today.

Q. Doctor, just finally, at paragraph 139 in your conclusion, red page 106, you make the point that there is, in your view, no reliable positive medical evidence to suggest any or all of the children were smothered or otherwise intentionally killed. That comes back, doesn't it, to the absence of positive signs of smothering in the four children?
A. Well, that's part of it, but there's also, of course, the issue that there are other potential competing causes of death identified via the history, the circumstances and the autopsy findings. So it's not just the absence of injury. I'm saying there are other factors coming into place in this discussion at this point.

Q. You wrote, "no reliable positive medical evidence". That's, surely, a reference to the absence of signs of smothering, isn't it?
A. Yes, I - in essence, that's correct, and the use of the word "positive" there does try and convey that point that, really, there's nothing here to suggest that smothering did occur, but, I guess, bundled into that discussion, there's also the fact that there were other issues identified, as I say, by the history, the circumstances of the person or the findings that perhaps suggest other factors may have been operative other than smothering, though, yes, it's not positive in the lack of other - or the positive - the - also the co-association of other factors which suggest that smothering didn't occur.

JUDICIAL OFFICER

Q. Just one other matter I'd like to raise with you, and you may have answered this. If you could go back to paragraphs 114 and 116 of your report, page 101 in our copies. On page 2.
A. Yes, your Honour.

Q. Does the conclusion that you reached in paragraph 116 depend upon your acceptance of the reports of Dr Ryan and Professor Fleming to which you refer in paragraph 114?
A. The first part, that is the immediate cause of death, I think would be due to epilepsy. I don't think that depends upon their opinions. The second part, 1(b), yes, that depends on what their opinion is and, to me, the underlying

cause of Patrick's epilepsy is an encephalopathic disorder. I don't know what the aetiology is. It was suggested that at the time of trial that there was one potential factor. There were reports and, I think, the history now suggests that other factors may have been operative and, really, I don't think we know the answer. So, yes, I'm reliant upon the opinion of others as to the undetected nature of the encephalopathic disorder.

NO EXAMINATION BY DR WOODS

<EXAMINATION BY MS HORVATH

Q. Doctor, I appear for Dr Cala, can you--
A. Good afternoon.

Q. Thank you. You referred to the fact that Professor Cordner provided you with the slides in 2018. Is it fair to say that you had read his 2015 report before you received the slides?
A. Yes.

Q. You had previously worked in Glebe and you, I take it, were aware that this Inquest for Laura would have taken place at Glebe; or at least in New South Wales?
A. I don't think I was at the time actually, no.

Q. No, I withdraw that, let me try again. You were aware that the autopsy in relation to Laura would've taken place in New South Wales?
A. Yes.

Q. That was the New South Wales forensic pathology group that you had worked for previously?
A. Yes, that's correct.

Q. You didn't ask New South Wales for a copy of the slides?
A. I have a vague recollection that I did but I think they were unwilling to let the slides be shipped overseas. I can't remember the specifics of that discussion.

Q. You say I think that you recall that all of the slides that you saw, and correct me if I have misstated it, do you believe they were fairly consistent with the images that are seen in the Brohus article?
A. No. I think that the images in that article probably identify and depict areas of more intense inflammation. As I've said, my recollection is that the extent and degree of information was somewhat variable, turned out to be there would be areas with less intense inflammation. I think those pictures would be chosen to highlight the more severely diseased areas.

Q. Yes. Do you recall whether myocytolysis, and I may have mispronounced that, but cell death, was that observable in your recollection on each of the slides you saw?
A. I can't recall whether myocytolysis was visible in each and every

slide. What I do recall is that it was actually quite easy to see, it was unequivocally present and, you know, when we deal with cases of really subtle myocarditis, which may just be an incidental finding and the question is, is this really - did this really provide a cause of death, then sometimes on those sort of occasions we can struggle to identify features of myocyte damage or damage to the heart muscles themselves, or as you termed it, myocytolysis. In this case really I think that the finding was quite clear cut and readily apparent in at least some of the slides. I can't say whether that observation was present in all slides.

Q. What you're looking for, isn't it, correct me if I'm wrong, you're looking for evidence of myocytolysis, that's what - that's the key factor that you're looking for, correct?

A. Well, it depends really what the purpose of the diagnosis is, and in the clinical setting really all the literature or the majority of the literature out there is based upon the clinical setting, that's in dealing with living patients who have what's called endomyocardial biopsy. So biopsies taken through the - like a little jib's being passed through blood vessels into the heart and little nips of tissue being taken. That's somewhat different from the autopsy diagnosis and in the clinical setting, at least looking at the Dallas criteria as they called, it was felt that there was certainly merit in definitely positively identifying myocytolysis before the diagnosis of myocarditis is given just to make sure that the diagnosis was a firm diagnosis and to avoid cases where inflammation might be borderline and not really clinically significant. In the autopsy setting it's a little bit different because of course we're not just looking at small bits of tissue, we're looking at large sections - incorporating much more of the hearts than we can assess down a microscope, but therefore the whole assessment is such - why I think different from the clinical setting.

Q. What you're looking for though, isn't it, is at least in Laura's case, weren't you looking for evidence of essentially corridors of dead and surviving tissue which could facilitate the occurrence of an arrhythmia?

A. Well, I don't think we know the precise answer as to why myocarditis can result in arrhythmias. Is it the inflammation? Is it the damage to the myocytes? Is it the organising fibrosis or scar tissue? I don't think we know the answer to that and maybe it's all three. Certainly, yes, we look for myocytolysis because that gives us a clue that the heart muscles have actually been damaged. You might want to take into account clinical parameters, you know, how was the subject clinically, were they exhibiting signs of heart failure? Were biomarkers deranged? Were there other markers clinically indicating heart damage? But looking just at the myocyte damage in microscope slides, yes, it's a key part of the diagnosis, but I would stress that's primarily in the clinical setting. But yes, it is, even in the autopsy setting that is one of the key features we look for and in Laura's case, to me, myocyte damage was unequivocally present.

Q. I understand, and I don't think any of the forensic pathologists disagree with the fact that myocyte damage was present, Professor Arthur Wilde, a Professor of Cardiology, in his report, I'm just going to ask if you agree or disagree with this, he said that:

> "In the case of extensive myocarditis the heart is susceptible to cardiac arrhythmias. This is because myocarditis may create 'corridors of surviving tissue in the heart' which will facilitate the occurrence of arrhythmias."

And just for the record, that's Exhibit 9-02 at red page 37.
A. Could you repeat that sentence, "corridors of"?

Q. His term was, "'corridors of surviving tissue in the heart' which will facilitate the occurrence of arrhythmias".
A. Again I'm just going to change screen if I can to make this larger for me.

Q. Certainly. It should be about in the middle of the page for you now under the bold question.
A. Yes, I see that.

Q. My question is, is that something with which you agree, disagree or can't say? And I don't mean to be impolite by saying that.
A. Well, I - I don't really answer this for sure, I am not a cardiac pathologist or cardiologist, but here the - this proposal seems to be one of theory as to how myocarditis might result in arrhythmic conditions, and that certainly seems valid, but maybe other factors might also be at play but I don't think I know the answer to that.

Q. It's the case also, isn't it, that myocarditis appears often as an incidental finding observed at autopsy and not the cause of death?
A. Yes, that can be the case, yes.

Q. So what you're looking for, I take it, is evidence that, especially with a child, there are florid features of the disease, correct?
A. Well I think you're looking for features of disease that suggest that this is a potential cause of death, and then you're weighing that up against other potential factors in death that there may be.

Q. Going back a step, and I won't be long, the slides that you saw in early 2018, it's correct that you took no notes of the review of the slides, correct?
A. That's correct.

Q. You don't recall how many there were?
A. How many slides?

Q. Correct, how many slides there were?
A. As I stated, I said several, I can't recall the specific number.

Q. What you have though seen - and you haven't seen them again in the last five years, the slides?
A. No.

Q. All you have seen I take it, in terms of Laura's tissue, is the Brohus article, is that right?

A. Sorry, can you repeat the question?

Q. Of course. What you have seen in terms of visual representations of Laura's tissue is the Brohus article, number one?
A. Well certainly I've seen that, and I've seen images in Professor Cordner's paper of 2015--

Q. That was going to be my number two.
A. --but the main thing belying on this is my mental image in my mind of my recollection of looking at the slides myself. Without--

Q. Can I just - I'm sorry, I wanted to direct you to my question so that it's clear what is you have seen over the past five years. It is the Brohus article with pictures, that's right, that's one thing?
A. Yes.

Q. The second thing is Professor Cordner's report which has also got some photos or images at the conclusion of it, yes?
A. Yes.

Q. Other than that, you haven't seen anything else over the last five years, correct?
A. No, that's correct.

Q. You would accept, wouldn't you, that your memory of slides that you saw five years ago is likely to be affected by the fact that you've only seen those images over the past five years?
A. I don't think that necessarily follows, no; and what I can say to you is I recall myself looking at these slides before the television cameras were rolling and being quite impressed, if I can use that word, as to the degree and extent of inflammation, and I recall that feeling quite vividly and I recall feeling to myself that certainly these appearances provide a, as I say, very compelling potential cause of death and that's my recollection, and I have relied upon that memory much more than the images that I've seen in the Brohus article and in Professor Cordner's report.

NO EXAMINATION BY MR JORDAN, MS LOVE, MR HASTINGS AND DR WATERHOUSE

JUDICIAL OFFICER: Thank you very much, Doctor. We are grateful for you to give up your time in the evening to assist in the Inquiry, it's been most helpful and I can only wish you a good evening.

WITNESS: Thank you, your Honour, you too, good afternoon.

<THE WITNESS WITHDREW

AUDIO VISUAL LINK CONCLUDED AT 3.07PM

CALLAN: Your Honour, the next witness is Professor Stephen Cordner, also

to give evidence by AVL. We're just contacting the witness to let him know to join the link.

JUDICIAL OFFICER: All right. There's one matter that I was going to raise. There's a bit of a cross-chat coming I think from the gaol.

CALLAN: Yes.

JUDICIAL OFFICER: That can - it's not, is it?

CALLAN: I gather that may have been coming from the witness' end on that occasion.

JUDICIAL OFFICER: I stand corrected. Do you want me to adjourn? I'm happy either way.

CALLAN: Your Honour, that might be more convenient, yes.

SHORT ADJOURNMENT

AUDIO VISUAL LINK COMMENCED AT 3.10PM

<STEPHEN MOILE CORDNER, AFFIRMED(3.10PM)

<EXAMINATION BY MS CALLAN

Q. Could you state for the record your full name?
A. Stephen Moile Cordner.

Q. It's the case that you're an Emeritus Professor of Forensic Pathology at the Department of Forensic Medicine, Monash University?
A. Yes, Emeritus Professor in the Department of Forensic Medicine at Monash University.

Q. You're presently a consultant to the Victorian Institute of Forensic Medicine. That institute being one where you were the Foundation Director from May 1987 for some extended period of time; is that correct?
A. Well, I'm a retired consultant from the Victorian Institute of Forensic Medicine, and I was the Director, as you mentioned.

Q. The Inquiry has received what we understand to be a current copy of your curriculum vitae as an annexure to a report that you furnished to this Inquiry in December of this year. It might not have been annexed, but it certainly was furnished at about the same time. Do you recall being asked for and providing a current copy of your curriculum vitae towards the end of last year?
A. Yes, I provided a copy. Yes.

Q. Your Honour, that's located at Exhibit 13-02A from red page 13-59 onwards. Professor Cordner, to make things clear for the record, you first prepared a report in relation to the death of the four Folbigg children, which was finalised in April or May 2015 and, your Honour, that is marked Exhibit 2-C and Exhibit 2-Q in this Inquiry. Professor Cordner, having completed that report in 2015, you recall furnishing by way of letter to the 2019 Inquiry a further explanation, if I can put it that way, for the circumstance in which you came to provide photomicrographs of Laura's heart tissue to some of your colleagues at the institute? That letter was dated 8 March 2019 and, for the record, it's at Exhibit 2-R. I'm not asking you to turn up that letter, Professor, but you recall providing that to the 2019 Inquiry?
A. I don't recall. I'm - it wasn't my letter. It was a letter from the Newcastle Public Interest Law Centre to the Coroner. I did have a copy. I don't recall providing a copy to the 2019 Inquiry. I have provided a copy to this Inquiry.

Q. We may be at cross-purposes. I may come back to that. In terms of your work since the 2019 Inquiry in relation to the death of the four Folbigg children, there was a report and opinion relating to the cause of death of Sarah and Laura Folbigg with a comment on the death of Patrick dated 1 March 2022?
A. Yes, I did provide that report. Yes.

Q. Your Honour, that's at Exhibit 13, tab 2. Then, Professor Cordner, you prepared a report and opinion for the 2022 Inquiry into the convictions of Kathleen Megan Folbigg, and the report was dated 13 December 2022?
A. Yes.

Q. Your Honour, that's at Exhibit 13, tab 02A. Can I deal with two specific topics, Professor Cordner, noting that you've provided a significant level of contribution through the 2019 Inquiry and the reports to this Inquiry and you were cross-examined in the 2019 Inquiry, along with several other forensic pathologists. One topic that I wanted to address with you is the topic of smothering or mechanical asphyxia. You dealt with that in your 2015 report, for the record Exhibit 2-C, red page 274 onwards. Professor, in the 2019 Inquiry, you prefaced your evidence on this topic of smothering by observing that diagnosed smothering is very, very unusual, and you use the term "rare". You also agree, I anticipate, that this topic of smothering and the signs it leaves is not capable of experimentation, at least not in humans? Do you agree with that?
A. So, yes, we're certainly not capable of experimentation, and if your first question was is an actual diagnosis of smothering rare in forensic pathology, I, for example, have never made that diagnosis in my career, because I've never been confronted with a case where I've felt I could come to that conclusion.

Q. In terms of the state of the literature on this topic, there is, for instance, a study that's reflected in an article that was annexed to - or has been annexed to several of the reports that you've provided over the years in relation to the Folbigg children. That article, the lead author is Burke, in the *American Journal of Forensic Medicine and Pathology* of 2004. Aside from that article, are there other articles that you've identified which reflect the studies or other commentary on the features that might be associated with asphyxia by smothering?
A. Yes. Well, for the research - in my research for the 2019 Inquiry, I could only find two cases of smothering in Victoria and New South Wales in the National Coroner's Information System. So that was other evidence of the rarity of it as a diagnosis and, yes, so if this Inquiry found four or five articles with cases where the pathologist in those articles, authors of those articles, had concluded that intentional smothering had occurred. So it's a relatively small number of articles, and it resulted in around about 20 cases that we could use, or I could use, to provide some information, I believe, to this Inquiry, but I do need to indicate to you that, unfortunately for the Inquiry, I've got one or two of the numbers wrong and that will need to be corrected at some point.

Q. Are you in a--
A. I don't - sorry.

Q. I was going to say are you in a position to do it as you sit there now or would you prefer to do it in writing?
A. I would actually prefer to do it in writing. I'm very happy to do it now. It - just to cut to the chase, it does not alter any of the words that I've used and, in particular, the second paragraph of 4.5 remain the same, with the exception that in the first line "20 deaths" should be 17.

Q. For the Court record, red page 13-18, final paragraph on the page, the number 20 should be 17.
A. Yes, but everything else in that paragraph, which is really the essence of my conclusion does not change.

JUDICIAL OFFICER: In the last paragraph on 13-18?

CALLAN: Yes. It commences with the word, "In the 20 deaths", and that 20 should be 17.

Q. The work that's set out in your report of December 2022, Exhibit 13-02A commencing at - if I can take you there, Professor Cordner, page 9 - that's red page 13-9 - details first a literature search, which as you've described, enabled ultimately a focus on some nine papers with relevant specific case information. Certain papers were then excluded, and over the page what that left was five papers with cases meeting the inclusion criteria, although, as you say, there were some specific cases within those papers excluded?
A. Yes. So that's - however, you're now starting to invite me to point to the correction, so I think you need to ask me whether - you need to tell me whether you want me to do that or whether you're happy to receive that in writing later.

Q. Why don't we do it now so that we don't proceed on an unfair or incorrect basis, Professor?
A. Okay. So, if we start on page 10, line 3, “This left 4 papers”, not 5. Okay?

Q. Yes.
A. Then on page 13, delete the three cases beginning 2.1, 2.2 and 2.3.

Q. Yes?
A. I can clarify why later.

Q. Could you just say now?
A. Yes. That was a case which was referring to asphyxia and smothering, but on reflection it does not specify intentional smothering as opposed to accidental smothering, so given that the focus of these cases was intentional smothering, I shouldn't have included them.

Q. Is that the extent of the?
A. Well, no, it has consequential effects on the numbers on page 14.

Q. So every time we see the words "20 cases" that should be "17"?
A. Well, it - yes and more, more than that, so - and I'm very sorry for this, but yes, in the 17 cases, then the first dot point should be 7 out of 17, not 8 out of 20. Have you got that? Do you want me to keep going.

JUDICIAL OFFICER: Yes. Keep going.

CALLAN: Yes.

Q. Yes please?
A. Second dot point, 7 out of 17, not 9 out of 20. Third dot point 3 out of 17.

Q. Yes?
A. Fourth dot point, 8 out of 17. Fifth dot point, 7 out of 17. Sixth dot point 2

out of 17. Then, in table 2, instead of the first number 8 that you can see in the table, under petechial haemorrhages, that should be 7.

JUDICIAL OFFICER: 7.4%?

CALLAN: Just seven I think and then we get to the percents, your Honour.

JUDICIAL OFFICER: I see.

CALLAN

Q. Yes?
A. Then immediately beneath that the 9 should be 7, and then over the top of the next page, the 5 should be 2, and on the bottom line the 20s should be 17, the number 9 there in the top right-hand corner should be 6.

Q. That is “Unknown whether present or not”, under the column CPR the 9 should be 6?
A. Yes, that's right. Then continuing on down there, line 4, petechiae 8 out of 17 should be 7 out of 14.

Q. Yes?
A. Then the next line of the 7 with petechial haemorrhages, not 8. Then in the following line in the remaining one, not two.

Q. Yes?
A. Then, page 17, in the right-hand corner headed "Intentional smothering" from table 2 above, reading from the number of cases, 17, “Number reported with petechiae or not”, 14.

Q. Yes?
A. “Number (%) with petechiae” 7 out of 14, 50%. “Number reported with facial injuries or not”, 15. “Number (%) with facial injuries”, 8 out of 15, not 1. Then “CPR performed: Yes, No, Not Known”, Yes: 8, No: 3, Not Known: 6.

Q. Thank you Professor.
A. Now we're getting close. Over the page, last line of the first paragraph, 7 out of 14, 50%, not 8 out of 17. Then third line, 8 out of 15 not known. Fourth line, 8 out of 15, which is 52%, not 60%.

Q. Yes.
A. I believe that is the totality of the corrections and I apologise for them.

JUDICIAL OFFICER: Not at all, thank you, Professor.

CALLAN: Thank you, Professor. We will proceed and I am grateful for that, because I don't want us to be going off on the wrong basis in terms of your questions, and I imagine that Ms Horvath, that might have implications for her as well, your Honour.

Q. After you've concluded your evidence this afternoon, Professor Cordner, we might seek to ensure that those changes are correctly reflected in an updated form of your report so that there is no--
A. Sure.

Q. --lack of clarity and correctness as to the position. In terms of the circumstance in which you came to cause this literature review to be undertaken, would it be fair to say that it was because of the lack of numbers around the topic of whether intentional smothering leaves physical signs in the information that's relevantly available?
A. Well an underlying theme of the forensic pathology discussion, a dominant theme really, is that smothering, so it is said, can leave no signs, and the absence of signs in really the four Folbigg infants, with a footnote of discussion about Sarah, it seemed to me came to be regarded as Longhurst is neither here nor there, and that didn't really accord with my sense, if you like. So, nobody, no individual has the experience of cases of smothering to be able to delve into, so we needed to go to the literature. We had gone as far as the experience in Victoria and New South Wales but that had only produced a couple of cases, which by the way did show petechiae and injuries, and then so we found a number of case reports where observations had been made about the presence or absence, so in other words absence was specifically mentioned as well as presence specifically mentioned in each case, and so we got the 17 cases that are set out in the table there. These are cases where the pathologists involved have concluded smothering largely but not exclusively buttressed, if you like, perhaps I shouldn't use the word buttressed, but in very many of these cases there were confessions.

Q. The outcome of the analysis that you set out in your report by reference to the literature review appears at page 15 of your report, red page 1315?
A. Yes, so the cases are set out in the table, is that what you're asking?

Q. Yes, and then your commentary in the paragraph immediately thereafter. Recognising the numbers are not large, they are, you say, "sufficient in my view for Saukko and Knight 2004 to revise their comment, quoted above", and that's a comment that appears back at page 8 of your report?
A. Yes, although I think it is interesting, I think it shifts the dial a little bit for forensic pathologists who have now got something to shoot at if they want to say something different.

Q. So that is rather than per Saukko and Knight's statement that, "The so-called classic signs of asphyxia, … for what they are worth, are rarely present in proven suffocation", you say at page 13 of your report, the numbers per the literature review you've conducted show that in around half the published cases of intentional smothering where there is specific reference to presence of absence of signs injuries are present?
A. Petechiae and/or facial injuries are present, yes.

Q. The position remains, is this right, that from your perspective as a forensic pathologist that intentional suffocation can leave no signs?

A. Well, the answer to that is yes. Of the 12 cases where the presence of petechiae or injuries was mentioned, or their absence was mentioned, in 12 of those cases 9 of them had petechiae and/or injuries, so it's not the case that they're rare, that the signs are rare. Indeed, it is the case that in - now, these - I don't pretend that these numbers rise to statistical - some sort of high level of statistical significance, but I do believe that they are numbers that have to be taken notice of, based as they are on case reports in reputable journals. So I think it's reasonable to say that in cases where the conclusion has been that the child under two was smothered and petechiae and facial injuries are specifically noted as present or absent, in three quarters of those cases one or both are present, and that has to be put up against each of the four cases, each of the four children of Mrs Folbigg, had no such findings, in parentheses, a footnote in relation to Sarah, who had two tiny abrasions on her lip or chin, which we know nothing about in terms of their appearance, their age, whether anybody saw them or not. So the conclusion at the end of the Inquiry was that these injuries amounted to nothing and all the pathologists at the Inquiry agreed that they could be related to resuscitation. So with that proviso, no injuries to the four Folbigg children.

Q. Can I take you to page 18 of your December 2022 report. It's red page 13-18.
A. Sorry, which page?

Q. Page 18. The third sentence, which commences with the words, "Thus, 3/12" or 25%. That's the necessary corollary of the evidence you just gave about the 75%, isn't it? That is 25% represents the best estimate we have of quantifying what it means when someone says that suffocation is very easy to do on a young child, a baby, without leaving any signs?
A. Yes, that's right.

Q. This further analysis that you've done is reflected in your December 2022 report. Do you regard that as strengthening the weight that you had already given to what you described as a significant negative finding, being the lack of facial injuries, as on the topic of the smothering of the four Folbigg children?
A. Yes, I do.

Q. Can I turn to the topic of Laura's myocarditis which you describe in your 2015 report as widespread and at least moderate in degree. Your Honour, that's Exhibit 2-C, red pages 303 and 306. I just want to be as clear as we can about descriptors, Professor Cordner. Do you describe Laura's myocarditis, as you saw it in the slides you inspected, as florid?
A. Sorry, can you point me to where I've - I've got my--

Q. Certainly. In your 2015 report--
A. Yeah.

Q. --at page 77 of the report, at red page 303, Exhibit 2-C, do you see at the top of the page?
A. Page 77, did you say?

Q. Yes, top of the page.
A. I can't recall whether I've used the word "florid". I wouldn't run away from the word "florid". I mean, writing the same, "widespread and moderate", so it's obvious. It hits you in the eye. There's no question about it. It's definite. It was in every section of every one of the slides I looked at, seven slides, I think, so it, sort of - I mean, you can't possibly avoid it or miss it or - so it depends, I suppose, on what you mean by "florid".

Q. I'll come to that, but I did just want to give you an opportunity to respond to that as a descriptor, but I recognise it all depends on what one might mean. Over at page 80 of your 2015 report, red page 306. Just above that heading which commences, "Conclusion about myocarditis", do you see the paragraph above that, you say, "The actual degree of myocarditis present was, in my opinion, substantially more than mild, and at least of moderate severity".
A. Yes. Can you - I'm sorry, can you just - where exactly are we? "Conclusion". What page is that?

JUDICIAL OFFICER

Q. Page 80 of your report.

CALLAN

Q. It's page 80.
A. Page 80. Yes. Got - thank you.

Q. There, again, you use a descriptor "moderate".
A. Yes.

Q. In how you've chosen to describe it, do you regard that term "moderate" as being different to describing the myocarditis as florid?
A. Well, it is, really, isn't it. So I think I'm - you know, but--

JUDICIAL OFFICER

Q. Is there any technical significance in the word "florid"?
A. There's no technical definition. These are descriptive terms used by pathologists to describe the intensity of an appearance.

Q. Is "florid" a well-accepted term of description in the profession?
A. Yes, it is. Yes.

Q. If you'd regarded it as florid, would you have said so?
A. Yes.

CALLAN

Q. In terms of the slides that you saw, which were the - as I read, the basis upon which you express your view and your descriptions of the myocarditis, those--

A. Perhaps - could I just interject just slightly. Whether it's florid or moderate, really, is neither here nor there in its consequences.

JUDICIAL OFFICER

Q. That's what I was seeking to ascertain.
A. From the point of view of a pathologist, it's not really a strong indicator of greater or lesser likelihood of sudden, unexpected death.

CALLAN

Q. Professor, the slides that you inspected, where were you when you inspected the slides?
A. Sorry, what was that?

Q. Where were you?
A. I looked at these slides at the Institute of Forensic Medicine in Sydney.

Q. Did you take those slides away with you, back to your employment at the Victorian institute?
A. Look, I can't remember. It - I have to say that that's possible, and with that possibility in mind, I have looked, but I certainly can't find. So - and I can't clearly remember whether I took them away. So if I didn't take them away, I must have photographed them at the Institute of Forensic Medicine in Sydney.

Q. The slides that you inspected at the Institute of Forensic Medicine in Sydney, do you know if they were the same slides that Dr Cala looked at for the purposes of the post-mortem?
A. I would think almost certainly not. I would think those slides had been cut and recut from the block. I can explain what that means, if you want, but I suspect that these are slides that are a little bit removed, I wouldn't know how far, from the slides of the heart that Dr Cala would have looked at at the time of the autopsy.

Q. You inspected the slides in, was it 2014 or 2015, in the context of preparing your 2015 report?
A. I have got a note about that somewhere. If that's important, I can track that back, but I certainly had to travel to Sydney, which I did think was totally unnecessary, but--

Q. You did so, in any event. I'm just wanting to identify why you consider that it's almost certainly not the slides that Dr Cala looked at for the purposes of his post-mortem. He, it seems from the records, undertook the post-mortem of Laura in March 1999 on the day she died and the days after that. It's some 15 years later that you're looking at the slides. Is that part of the reason why you say it's almost certainly not what you looked at?
A. Well, I think they probably did cut another set for me and stain them again so that they were freshly stained so that they looked as good as possible. That is one reason. Second reason is clearly this was a major, you know, very important death investigation, and I imagine slides were shared with other

pathologists in the institute and perhaps beyond. It's a truism that slides get lost, so more slides would've been cut and there might've been some special stains made. Dr Cala himself probably asked for more and deeper cuts. I don't know that but he might've, wouldn't be surprising. So, I think there are quite a few reasons why it's almost certain that I am not looking at the same slides as Dr Cala looked at, and, you know, that is always sitting there in the background when there is some slight disagreement between pathologists as to what they're looking at that they may not actually be looking at the same material.

JUDICIAL OFFICER

Q. Is that potentially a reason for the disagreement between you and Dr Cala?
A. It could be a contributor, yes.

Q. If Dr Cala had looked at the original slides, if I could use that word crudely, why in those circumstances shouldn't I give greater weight to the views that he's expressed as distinct from the view you expressed?
A. I'm sorry, I didn't quite hear that.

Q. If you accept that the difference between you and Dr Cala could relate to the difference in what you observed - in the slides you saw, your view may well have been different had you seen what Dr Cala saw at autopsy?
A. I don't - and I think we agreed that there was myocarditis in every slide, and he might be saying that it was of a lesser degree than I was saying, and the fact that we're looking at different slides is a possible reason contributing to that difference.

CALLAN

Q. In a sense to go back to what emerged in questions his Honour was asking you about the difference between moderate and florid, do you regard the difference in descriptions as between Dr Cala who tended to describe what he saw in the slides as patchy or mild compared to your description as bearing on the view that you have expressed as to myocarditis being a potential cause of death for Laura?

HORVATH: Your Honour, I object to that question. It's inconsistent with the autopsy report and the evidence in the 2019 Inquiry. If the attribution to Dr Cala has changed to be consistent with the autopsy report, I wouldn't have any difficulty.

JUDICIAL OFFICER: Are you happy to rephrase it?

CALLAN: Yes.

JUDICIAL OFFICER: though I must say, I thought there was a measure of agreement on this topic.

CALLAN: I had understood that but I am trying to ascertain whether any of the

difference in these descriptors has been given over the years as material.

JUDICIAL OFFICER: Yes.

WITNESS: I personally don't think it's material in terms of the consequences and my debate, if there, and there was debate with Dr Cala, was about the reasons he relied upon for a conclusion that it was unlikely that Laura's sudden death could be explained by the myocarditis.

JUDICIAL OFFICER: Yes.

CALLAN: Yes. I'll move on.

Q. Professor Cordner, do you recall in 2018 being involved in the provision of slides of Laura's heart tissue to Dr Matthew Orde in Canada?
A. Okay. Now that you mention that, I do recall that.

Q. Do you recall whether the slides that you caused to be provided to him were the same ones that you examined?
A. They probably were.

Q. Why do you say that, probably were?
A. Well, I wouldn't have had any others.

Q. Why did you send them to Dr Orde?
A. He was interested to have a look, he'd been asked his opinion about the case by a television program and it seemed reasonable to me to share them with him. He is a colleague I knew and know and respect, so I did not see any reason not to.

Q. The fact that you were in a position to provide those slides to Dr Orde, does that indicate that you did take slides away with you after inspecting them at the Sydney Institute of Forensic Medicine?
A. It could be that or it could be that they subsequently sent them to me from New South Wales, so I can't - but that's neither here nor there, I think they are likely the slides that I saw in Sydney.

CALLAN: Your Honour, I note the time, I have one short further topic.

JUDICIAL OFFICER: Yes, go.

CALLAN: Thank you.

Q. Professor Cordner--
A. Perhaps I'll just say one thing, it took - I don't think I would've taken, unless it was provided to me or given to me or--

JUDICIAL OFFICER: I don't think Ms Callan was suggesting anything to the contrary, Professor.

CALLAN

Q. I am sorry, I meant no implication by that, Professor Cordner, other than that they were slides provided to you or which you had access to to enable you to provide copies to Dr Orde.
A. Yeah. Thank you.

Q. You're aware, aren't you, Professor Cordner, of an article that was published in the *European Society of Cardiology Europace Journal* in 2021 titled "Infanticide vs. inherited cardiac arrhythmias"?
A. Yes.

CALLAN: Your Honour, that's at Exhibit 15, tab 2.

Q. Do you have a copy of that article with you, Professor, we can put it up on the screen?
A. Look - please do.

CALLAN: If we could have that up on the screen, it's red page 19, page 449 of the article, and if we could zoom in on the acknowledgment section.

Q. Do you see there, Professor Cordner in the acknowledgements there is an indication of being grateful to Stephen Corner for providing the cardiac photomicrographs?
A. Yes.

Q. They are the photomicrographs that appear if we turn back to the article at red page 13?
A. Yes. I would've provided them with a number and they would have selected whatever they selected.

Q. That was part of my question, do you recall providing more than the four that appear there?
A. I probably did but I can't recall, I'm pretty sure I would've given them more than four. They probably picked four and then I would've written - I think I wrote what is the description of figure 1.

Q. Do you recall, were you involved in the selection of the four that appeared in the article?
A. Well, I would've been involved in the selection in the sense that I picked them I suppose to be sent to the article and then there probably was some correspondence saying well we'd like one, two, three and four, could you please describe them. So I did, I think it's important to also say that, as I'm sure you're aware, these are small sections of the slides, they are not whole slides in themselves.

JUDICIAL OFFICER

Q. You don't suggest those, or do you suggest they show any selection bias?
A. I'm sorry, your Honour.

Q. Do you consider these slides paint an accurate picture of what you see when you have the totality of the slides that you had in your possession?
A. I think they are a good representation. I mean there's only really I mean the small number of slides there, there's four and two of them are very high power. So, I do think that's a reasonable representation of the moderate nature, at least moderate nature of the myocarditis.

CALLAN

Q. If we could go back a page, red page 12, page 442 of the article, it's the right-hand column, top quarter? Do you see, Professor Cordner the right-hand column commences, “Of 10 years", and if you read it through, there's some information about child one, child two, child three and then child four? It reads child--
A. Yes, and I certainly didn't provide that sort of information, I'm pretty sure.

Q. The reference to child four having died at 18 months old, two days after being treated with paracetamol and pseudoephedrine, that reference to pseudoephedrine, that's wrong, isn't it?
A. Yes and there's the word florid, is that what you're pointing to?

Q. Then, yes, it uses the word florid, that didn't come from you?
A. Well, I'm not saying it didn't come from me, but I mean I haven't got any direct memory of that. So, yeah, I'm not quite sure what your question is.

Q. Did you critically review the entirety of this article before it was published?
A. Did I receive the entirety of the article? I'm not sure that I did. At the beginning they asked if I would like to be an author and involved, and I said, no, I didn't think that was right because this is very much out of my field, and at that point I might've even - I think they were offering me authorship because they knew that I could provide the photographs, and I didn't think, you know, I was going to be contributing enough at all to be an author and it was a field that was quite some distance from mine.

Q. Professor Cordner, did Professor Vinuesa ask you to critically review the manuscript before it was submitted?
A. Okay. Well, so if she's saying she sent it to me, well, of course, she did, and perhaps the only contribution I could make was in that sort of area where you've shown me.

Q. Do you recall being asked by Professor Vinuesa to critically review the manuscript?
A. Look, I can well - I can imagine she did. I can't actually - well--

JUDICIAL OFFICER

Q. Did you critically review the manuscript?
A. She was - we was - she was certainly communicating with me at the time the article was being generated. I honestly can't specifically recall that she asked me to critically review the entire manuscript, but I'm not going to say she

didn't. So that's where it stands.

CALLAN

Q. If you did critically review the manuscript, can you explain why the error appears there suggesting that child 4 had been treated with pseudoephedrine?
A. Yeah, I can't.

JUDICIAL OFFICER

Q. Professor, whether or not you were asked--
A. I can't explain. I don't know.

Q. --to critically review the manuscript, do you recall in fact doing so?
A. Do I recall what, sorry, your Honour?

Q. Do you recall in fact critically reviewing it?
A. I don't recall critically reviewing the whole article, no.

CALLAN

Q. Given your level of familiarity with the circumstances of each of the four children, is it likely that you overlooked that error in describing child 4 as having been treated with pseudoephedrine?
A. That's possible.

Q. Possible?
A. Well, you know, yes. That's possible I could have made that sort of mistake if I was reviewing it.

<EXAMINATION BY DR WOODS

Q. Professor, you were asked some questions about the autopsy slides, which were seen by Dr Cala, who, of course, conducted the autopsy, and the slides that were later produced, and his Honour asked you a question about the comparison of the usefulness to this Inquiry of what Dr Cala originally saw and the slides that you asked to be cut. Could you assist us as to that? Is one better than the other?
A. Yes, I think I can. The degree of myocarditis, I think, and, understandably in some minds, has come to be equated with the likelihood that Laura actually died of myocarditis. So you can understand somebody thinking if it's more severe myocarditis, the greater likelihood of a sudden, unexpected death.

Q. Can I just ask you this. We know little at the Bar table about how these slides are created. You used the expression "new slides being cut". Cut from what?
A. Okay. So at the autopsy, a 10-cent-size of tissue, perhaps half a centimetre thick, is removed from the organ, in this case the heart, and placed in a small plastic cradle, a small plastic container called a cassette. That cassette is put in formalin and stays there for a day or so, and in the histology

laboratory, that cassette is, with the tissue in it, goes through a form of processing, which leads to the tissue being embedded in wax, so that there's a 20-cent-size piece of wax, in the middle of which is the tissue. That enables a very thin slice of the tissue, five to seven microns thick, so thin that is then placed on a glass slide and the tissue adheres to the slide and then the tissue is colourised, and then a cover slip is placed over the tissue to protect it. So you've got colourised tissue on a glass slide which can be looked at through a microscope.

Q. Do you say that even years afterwards another very thin slice can be taken from that wax cassette or--
A. That's right, and that's why those cassettes are in a - probably a credited setting, are kept for years and years in case they need to be looked at again.

Q. When you were at the institute in Sydney obtaining that material, were all the proper procedures followed to do that? For you to obtain the slice?
A. I'm sure that - I'm not quite sure what--

JUDICIAL OFFICER: Dr Cordner could only assume it to be the case, couldn't he?

WOODS: Yes. I won't pursue that.

Q. The only other thing I want to deal with, Professor Cordner, is that in the course of the statistics that you produced, and which you've corrected, giving rise to the statistics that you've been talking about as to petechiae and possible injury, I noticed that there was a reference in a number of the reports that you wrote up to the mother confessing, presumably to authorities. Did you see that in a number of them?
A. Could I what, sorry?

Q. Did you see that in a number of the reports that were examined?
A. Yes, there was reference to confession in quite a number. Yes.

Q. A few days ago my learned instructing solicitor contacted you, did she not, to ask you to prepare a list, if you could, of instances in which confession was mentioned. Do you recall that?
A. Yes.

Q. If his Honour wishes to be assisted in this way, would you be prepared to send along to the Inquiry a document which deals with the same material that you've already referred to, but lists off the instances in which, as you analyse it, there appears to have been a confession to authorities?

JUDICIAL OFFICER: Why would that assist me, Dr Woods? Firstly, is it going to show any more than what's in Professor Cordner's reports and, secondly, the significance of a confession, I would rather have thought, is that people can be confident that the death was caused by a deliberate suffocation.

WOODS: Your Honour, these are cases of suffocation that we're talking

about, and the point I want to put forward is that there's a - in the case of Mrs Folbigg, that she's persistently said for 20 years that she didn't do it and, by contrast, these statistics may indicate a pattern of those few people who have been guilty of smothering have mostly confessed to it, by contrast to her situation.

JUDICIAL OFFICER: I don't mind if someone wants to extract for me from Professor Cordners' report how many of them are confessions. It'd probably save me adding them up, but I can't see it's going to do much greater benefit than that.

WOODS: Your Honour, with respect, I understand that's your Honour's present position, but I would wish to make that argument.

JUDICIAL OFFICER: I understand the argument, but all I'm saying to you is that why do I need another document when I can add up to about 6 or 12 myself.

WOODS: Your Honour, I don't seek that they - can I ask Professor Cordner a question?

JUDICIAL OFFICER: Yes.

WOODS

Q. Professor, from the material that you looked at so far, do there appear to be other cases in the materials you've looked at which would supplement or add to those where the word "confession" appears in the already submitted material?
A. Yes, there were. So there were a few cases - well, a number, really, where there was information about the circumstances which referred to confession, but didn't fit the inclusion criteria for inclusion in my report. So it was a different analysis and it was actually quite interesting to see the, sort of, dominance, really, of confession in the concluded - cases concluded to be intentional suffocation.

Q. If my learned friend at the Bar table and her associates contact you about it, could you provide that report?
A. Sure.

Q. If I can just add something to what I was asking you before. The slides that you had cut in the way that you described, would you regard them as being, in your experience as a long-standing Senior Forensic Pathologist, of the normal kind that would be obtained?

JUDICIAL OFFICER: I don't understand that question.

WITNESS: I'm sorry, I didn't get the last part of the question.

WOODS: Your Honour.

JUDICIAL OFFICER: You can ask it, I just don't understand it. I'm not going to prevent you from asking it.

WOODS: Very well.

JUDICIAL OFFICER: I don't understand what's the normal kind of slide to be obtained.

WOODS

Q. Very well. In terms of the value of proving anything, are your slides, the slides you looked at, less significant or important than the slides that Dr Cala looked at?

CALLAN: I object.

HORVATH: I object.

JUDICIAL OFFICER: I reject it.

WOODS: Very well. I've got no further questions.

<EXAMINATION BY MS HORVATH

Q. Professor Cordner, I appear for Dr Cala. Can you hear and see me?
A. I can, but I can't see your head.

Q. Let me know if you can't hear me at all. Can I ask you, going back briefly to the pictures in the Brohus article. Your evidence was that those were representative of what you saw on the slides you viewed; correct?
A. I thought they were reasonably representative, yes.

Q. On those pictures, there is evidence of cell damage, myocyte necrosis; correct?
A. I believe so, yes.

Q. If it's the case that Dr Cala says that on the slides he's got, four of the seven don't have any evidence of myocyte necrosis, would that suggest that the slides you're looking at are somewhat different?
A. No, absolutely.

Q. Is it the case that when new slides are cut from a particular tissue block, they're very thin, so you would assume that they would be reasonably close and similar to the previous slides that were cut, yes?
A. Well, that's the basic assumption, but we all know they're at least five to seven microns removed, and quite often more. There's - multiple slices are cut in order to get a good one.

Q. If multiple sets of slides were created between the ones that Dr Cala looked at in 1999 and the ones you looked at in 2014, that would, presumably,

mean that they're even more different; correct?
A. Yes.

Q. You would expect, wouldn't you, that in a case which has been to a trial where there were a number of forensic pathologists who gave evidence at that trial that multiple sets of slides would have been cut between the set that were cut for Dr Cala and the ones that were cut for you; correct?
A. Yes.

Q. Going back to a question that his Honour asked, that would, no doubt - or could, no doubt, explain why you and Dr Cala take a slightly different view about the severity of the myocarditis?
A. Yes.

Q. In terms of the relevance of the severity, there has been some evidence before this Inquiry from a Professor Wilde where he said, and this was in his report at Exhibit 9-02, that, "In the case of extensive myocarditis the heart is susceptible to cardiac arrhythmias. This is because myocarditis may create 'corridors of surviving tissue in the heart' which will facilitate the occurrence of arrhythmias". Is that something that is consistent with your experience?
A. Look, I'm not disagreeing with that, but I do think that isolated foci or isolated areas of myocarditis, as opposed to defuse areas spread throughout the entire heart, can also be associated with sudden and unexpected death, particularly if those foci are near the, sort of, conduction system or the electrical wiring above the heart, so I wouldn't say that is the only type of myocarditis that is associated with sudden unexpected death.

Q. In relation to the slides you looked at, and you're not able to say, I take it, whether they were or were not located near the conduction system of the heart?
A. Well, I'm not conscious that they were, and I do think that increasingly now in infant deaths in dissecting the heart, pathologists would be more alert to including a section that includes the conduction system and the atrioventricular node, it's called.

Q. It's the case, isn't it, that myocarditis, as an incidental finding in child death, is reasonably common, as compared to cause of death. Do you agree with that?
A. Well, if myocarditis is reasonably common as an incidental finding, then you'd have to use similar words in relation to its frequency as a cause of sudden unexpected natural death, because the incidences are about the same.

Q. You're aware, I take it, of Professor Byard's study in relation to, I think, almost 5,000 autopsies of children and babies over 35 years, which was reported in 1992?
A. Well, you'll have to remind me of that.

Q. Where he referred to 32 cases of myocarditis that had been discovered at autopsy of the 5,000 autopsies, and found that about half of them were

incidental findings, and the other half were causative of the death. Is that--
A. Yes, well, that was the point I was making that you just mentioned that myocarditis was quite a frequent finding in - as incidental to other causes of death, and I've just simply said that if you were going to say that it was reasonably common in - as an incidental finding in other causes of death, you would also have to say it was reasonably common as a cause of death. I only mention that because the rarity of myocarditis as a cause of death was something that exercised the first Inquiry.

Q. Yes, and I think there was debate about uncommon and rare, and I'm not going back to it, but you would agree, wouldn't you then, that your experience I take it is consistent with that reported by Professor Byard that about 50% of findings of myocarditis are incidental findings as opposed to cause of death?
A. Well, there are variations on that conclusion, so I can give you a reference if you're interested, there's a couple of references which are similar but not the same as the experience you've just described, and the particular reference is "Myocarditis and SIDS", this was a Danish study from way back in 2002, published in APMIS, volume 110, page 469-480, which showed that 69 out of 410 infants dying suddenly and unexpectedly had myocarditis and 2 out of 27 who died violently had myocarditis. There's a lot of interest in that paper from my point of view. That relatively high number of infants in a population of infants dying suddenly and unexpectedly, that relevantly high number that died of myocarditis might have something to do with the fact that they had a very specific dissection method that made sure they included representative samples of the electrical wiring of the heart and in those areas they found areas of isolated myocarditis and there's no question that they reasonably ascribed death due to myocarditis. So, just goes to show the importance I think of the sampling.

HORVATH: Your Honour, I'm not quite sure how long your Honour is intending to sit.

JUDICIAL OFFICER: How long will you be?

HORVATH: I was about to go to a different topic and I'll be, I suspect, at least 20 minutes.

JUDICIAL OFFICER: No, I have to adjourn by quarter to 5 at the latest. So, Professor, unfortunately I don't think we can finish your evidence today. Could I have those assisting me make some enquiries as to when you're otherwise available?

CALLAN: Yes, we understand Professor Cordner is not available first thing tomorrow morning, and in those circumstances, we need to review the witness schedule, noting that otherwise it was proposed to hear from Dr Cala, but that shouldn't occur until we've finished with Professor Cordner's evidence. So, can we leave it on this basis? Your Honour, that we will communicate with Professor Cordner about if we can find a convenient time during the balance of tomorrow for him to complete his evidence, and the consequence--

JUDICIAL OFFICER: That's going to be difficult, isn't it?

CALLAN: Well, there is something in the order of three hours that's been allocated to each of Mr Sheehan and Dr Eagle. I'll speak to my colleagues about whether or not that's generous or not in terms of the time estimates. If we can fit, for instance Professor Cordner in tomorrow afternoon--

JUDICIAL OFFICER: The possibility may be - I'm sorry, Professor, if you don't mind listening to this - the possibility may be, I don't know if Mr Sheehan can be moved to a little earlier.

CALLAN: We'll certainly make these enquiries, your Honour, and we'll let the parties know.

JUDICIAL OFFICER: And you'll discuss it with Professor Cordner.

CALLAN: Of course, yes.

JUDICIAL OFFICER: Mr Jordan, I understand you've got an objection to part of Dr Garstang's evidence, is that right?

JORDAN: Yes, that's right, your Honour.

JUDICIAL OFFICER: How long do you think that'll take to argue?

JORDAN: I can put the objection very briefly.

JUDICIAL OFFICER: I'm not asking you to do it now, but can we deal with it at 8.30 tomorrow morning?

JORDAN: I don't think what I have to say won't take anything like half an hour.

JUDICIAL OFFICER: We'll deal with it at 8.45 tomorrow morning then.

JORDAN: Yes, I mean even 15 minutes is a generous estimate.

CALLAN: Your Honour, perhaps if we could let Professor Cordner as it were terminate the link.

JUDICIAL OFFICER: Yes. Yes. I'm sorry, Professor, we don't need to keep you hanging on, so you can terminate the link.

<THE WITNESS WITHDREW

AUDIO VISUAL LINK CONCLUDED AT 4.33PM

CALLAN: And we'll make contact with him about the time for him to resume his evidence. It may be of assistance for instance for the legal representatives for Ms Folbigg to hear the nature of Mr Jordan's objection and whether or not it can be determined.

JUDICIAL OFFICER: Yes.

JORDAN: Do you want me to say it now?

JUDICIAL OFFICER: Yes.

JORDAN: Essentially it is this, and I do, as Dr Garstang is clearly a paediatrician and the essential basis for the objection, which really starts at paragraph 8.4 of her report and goes to the end of the report--

JUDICIAL OFFICER: Look, I think I'll hear this tomorrow morning, Mr Jordan.

JORDAN: Yes your Honour.

JUDICIAL OFFICER: We'll adjourn until 8.45 on the basis that will take 15 minutes.

ADJOURNED PART HEARD TO THURSDAY 23 FEBRUARY 2023 AT 8.45AM