SPECIAL INQUIRY

THE HONOURABLE THOMAS BATHURST AC KC

EIGHTH DAY: TUESDAY 21 FEBRUARY 2023

INQUIRY INTO THE CONVICTIONS OF KATHLEEN MEGAN FOLBIGG

AUDIO VISUAL LINK COMMENCED AT 10.06AM

<PETER FLEMING, AFFIRMED(10.07AM)

<EXAMINATION BY MS CALLAN

Q. Could you tell the Inquiry your full name?
A. My name is Peter Fleming.

Q. You're a Professor in what discipline?
A. I'm a Professor of Infantile Developmental Physiology and I'm a Consultant Paediatrician with a background in both Neonatal and Paediatric Intensive care and also a Registered Specialist in Paediatric Respiratory Medicine.

Q. It's the case, Professor Fleming, that you've prepared a report for the purposes of this Inquiry dated 11 October 2022?
A. That's correct.

Q. Do you have a copy of that report with you?
A. I do. I have it in front of me.

Q. For the record, that report is at Exhibit 11-02 commencing at red page 8. There is a brief curriculum vitae attached to that report with appendices that provide further detail of your background, experience and contribution in your areas of specialisation. I don't propose to take you through the details of that, they speak for themselves, but at the beginning of your report under the heading "Introduction", you provide an overview of your specialisation and experience.
A. That's correct, yes.

Q. And that includes over on page 3 of your report, red page 10, this is about line 67, that since 2018 you've been a member of the Core Project Operational Group and Chair of the Professional Advisory Group to the National Child Mortality Database?
A. That's correct.

Q. You describe that database at line 70?
A. Yes.

Q. Which indicates that since 1 April 2019 by force of statute, all child deaths

in England are to be reported to you, or you say “us”, within 48 hours. Who is "us" in that sentence?
A. That's to the National Child Mortality Database. We were established under the regulations of the *Children Act 2004*, which makes it a statutory responsibility of anybody becoming aware of a child death to ensure that our team is notified. We have a secure web-based system that allows that to happen from anywhere in the country. There are 56 teams around the country who are involved in the local investigation of such deaths and our secure web-based notification system is as soon as a local team is told we are informed as well, so we are able to make sure that the local team have the necessary information, support and backup.

Q. To be clear, child deaths means from birth to age 18. Upon receipt of that sort of notification to the database, what role, if any, do you play in reviewing the material gathered during an investigation of such a death?
A. There are three paediatricians who jointly share this and I'm one of those three. We review every death as we hear - every day we review every death that has been notified. It's approximately nine or ten child deaths per day in England with a population of 55 million people. So, we review them. We look at what the information is that we have and where appropriate we will provide support and advice and information to the local team to make sure that the investigations that are carried out are appropriate and effective.

Q. That review occurs as I hear your evidence, at or very shortly after the point of notification? Is that correct?
A. That's correct. And then from then on we monitor on a - on a continuing basis. Anywhere we had an input and we feel there's a need for us to be involved, we will monitor. As new information comes in to the local team we will be there providing, if you like, additional support and backup. And particularly where we're dealing with very rare conditions, we're able because of our national links to ensure that the right investigations are done, whereas local teams may well not have that facility locally, but it allows us to make sure that things are done appropriately and quickly.

Q. Your report of 11 October includes a description of the Joint Agency Response which is a feature of the investigation of unexpected child death in England and has been since 2008?
A. That's correct. There is a system that we developed in the Bristol area in the 1990s, and in 2003, I was a member of a committee set up under the chairmanship of Baroness Helena Kennedy, but the committee agreed that this was the appropriate way for child deaths to be investigated throughout England and it was incorporated in its entirety into the *Children Act*, which at that time was going through parliament.

Q. As you observed, the aim is to investigate the events leading up to and potentially contributing to the death and otherwise review all of the evidence available as to the circumstances of the death, to ensure that appropriate investigation is done.
A. That's correct. That's correct. So, as I said there are 56 teams based in localities mostly dealing with a population of between a half and 2 million

people. And we support the local teams who actually do the groundwork, if you like, or we're there as a national resource to ensure they have the necessary backup. But each of those teams is lead by experienced and senior paediatricians.

Q. And as you tell us, you have personally conducted more than 200 immediate investigations after unexpected child deaths as the medical lead and you've supported, facilitated or overseen such investigations of more than a third of 600 such deaths?
A. That's correct. And in fact, it's part of my role in the National Child Mortality Database. I've been responsible for overseeing the local investigation of around 1,000 more such deaths.

Q. You were originally engaged by the legal representatives for Ms Folbigg and they asked you to address a series of questions and they appear, if you turn to page 6 of your report, red page 13--
A. Yes.

Q. --do you see the questions are there set out. And I'll come to some of the answers you've given to those questions in a moment. Could you turn over the page? It's report page 8, red page 15. The list of documents reviewed?
A. Yes.

Q. That includes all of the report of the 2019 Inquiry, the reports of Professors Horne and Elder and their oral evidence given in that Inquiry, and then the reports of some of the forensic pathologists who have given evidence in the 2019 Inquiry and in this Inquiry. And you also provided a copy of what's described as the forensic pathology tender bundles, submitted to the Inquiry in 2019. Marked in that Inquiry as exhibit H--
A. Yes.

Q. --and in this Inquiry as Exhibit 2-H, just for your information. I propose to take you to just a few number of those documents in addressing some of your answers to these questions. Complementing your lengthy report of 11 October, you prepared an executive summary dated 3 November 2022, didn't you?
A. That's correct.

CALLAN: Your Honour, that's also at tab 11-02. It's page 7-1 through to 7-4 using the red pagination.

JUDICIAL OFFICER: Yes.

CALLAN

Q. Do you have a copy of that report of 3 October there with you, Professor Fleming?
A. I do have a copy of all three of the reports I've prepared.

Q. And the third report, being a report dated 1 January 2023, which is a

response to the report of Dr Cala of 21st December 2022.
A. That's correct.

CALLAN: Your Honour, for the record, that's at Exhibit 11-03.

Q. Professor, I wanted to first address a topic you deal with in your report in terms of physical damage to infants as a result of suffocation. You commence dealing with that topic at page 26 of your October report, red page 33.
A. Yes, I have that.

Q. At the last line on that page, commencing with the words, "the reports from almost all of the experts involved in both the trial and the Inquiry in 2019 note that it is possible to suffocate a young infant by obstructing the upper airways without leaving any marks that might indicate that this has occurred." You go on to say, "this assertion is very commonly made by experts when an externally applied upper airway obstruction is suspected as the potential cause of death of an infant or young child, but the evidence base for this assumption is very hard to identify." The difficulty with the evidence base is that this is not something which can be the subject of experimentation. Do you agree in that respect?
A. No, absolutely. It's - one can’t - one can't conduct an experimental study, but it's - as I said, the assumption is made and it's repeated multiple times in courts around the world, but the evidence for that assumption is very, very hard to tie down. I cannot - I have not been able to find good evidence to support it. And indeed from my personal experience of resuscitating young infants where you put pressure on the face, I find it almost inconceivable that you could suffocate a child with eruptive front teeth without damaging the inside of the mouth. Because often we damage the inside of the mouth by trying to resuscitate them.

Q. Does it depend on the method of suffocation? You've described a method which might be equivalent to actions performed during resuscitation. There are other forms of suffocation.
A. Anything that pushes the lip onto the teeth and anything that disrupts the upper airway would inevitably do that, would risk damaging the inside of the lip. And indeed, one of the reasons we sometimes suspect someone's harmed a child is because we find damage to the inside of the lip.

JUDICIAL OFFICER

Q. Does that depend to some extent on where the teeth are located in the mouth?
A. Where the teeth are? Well, the front teeth always erupt before the back teeth.

CALLAN

Q. At the top of your report, your page 26, red page 33, you refer to work during the 1990s in collaboration with Professor David Southall and Dr Martin Samuels, and you describe there a covert video surveillance operation which

was conducted.
A. Yes.

Q. You weren't personally involved in that video surveillance, but is it the case that you referred cases?
A. Yes, I wasn't involved in the conduct of the video surveillance, but I have seen the recordings that came from it, and the - they're very distressing recordings. I mean, they're - they're horrible. But it's very clear that even in very young babies, when the airway is obstructed even for a few seconds, the child wriggles and struggles extremely vigorously.

Q. One of the articles which you referenced in your report - and, your Honour, this is at tab 15-18(h) - is an article that was published by Dr Samuels and others in relation to this work that was done by way of covert video surveillance.
A. That's correct.

Q. Can I ask if you have a copy of that article with you? If not, I can show you on the screen a reference point that I wanted to ask you about.
A. I don't actually have it printed out in front of me, but I'm quite familiar with it, so I may be able to help.

CALLAN: If I could ask that that article be shown. It's p 163 of the article.

JUDICIAL OFFICER: What page of the bundle?

CALLAN: Red page 384.

Q. Do you see that's the article there, Professor Fleming, and I'm taking you to the second page of the article.
A. Yep.

Q. In the left hand column, just above the heading that reads "Protocol for Instituting CVS", there's a paragraph that commences with the words "Imposed upper airway obstruction".
A. Yes.

Q. "was considered to be the likely diagnosis at the referral of eight of the 14 patients. One of the eight had petechial haemorrhages on the face and neck after a cyanotic episode." And it goes on, "Their paediatricians did not consider, however, they had sufficient evidence to confront the parent confidently and be sure of securing the long-term safety of the child. The remaining six patients were referred for the management of recurrent cyanotic episodes, considered to be due to natural pathophysiology." That description as to what the paediatricians observed, before they referred the cases to this team doing this work, suggest that aside from one of the eight patients with petechial haemorrhage, otherwise seven of the eight didn't display, I'll put it this way, sufficient observable damage for the paediatricians to consider they had sufficient evidence to confront a parent as to the topic of suffocation?
A. That's correct. I mean, the reason they were referred was that there was a

question about whether this was natural or wasn't natural. But I mean the wording of the paper is somewhat misleading in that the only patients who were referred to the Samuels and Southall team, were where this was being considered as a possibility. It didn't mean it was a probability. It was something that was being considered as a possibility, that involuntary obstruction.

Q. Reading this, aside from that one patient with the petechial haemorrhage, it appears that otherwise there was no or insufficient evidence, by way of physical evidence, to permit the paediatrician to consider as a matter of likelihood, there'd been an imposed upper airway obstruction.
A. No, that's right. I mean, these were - there was a concern that these may have been imposed, but there was no hard evidence in the way of - and part of the reason, obviously if we found - if there had been very clear evidence of physical injury, then there wouldn't have been any need to do covert video surveillance. These were the ones where there wasn't any. So, in a sense, they're a selected group in that there was a concern that there may have been imposed airway obstruction, but there wasn't sufficient physical evidence to reach that conclusion.

Q. Do you regard that as providing some support for a view that suffocation of young infants can occur without leaving physical signs or damage?
A. These children didn't die. Sorry, I mean, these were children who'd had a much milder - had - had - potentially had airway obstruction, but had not been - it had only been for a short period of time and there hadn't been - suffered harm. If you go further on in the paper, you'll see that where the episodes were recorded of airway obstruction, there was evidence of changing colour and minor injury to the face, which was visible in several of them immediately afterwards.

Q. But that was in circumstances where the event of suffocation was only permitted to go on for something in the order of 10 to 15 seconds.
A. That's correct.

Q. As opposed to, as I read it, "the 50 to 70 seconds", which is regarded as necessary or required for sufficient hypoxaemia.
A. That's - I mean, the episodes were interrupted as early as possible, having been documented. But even so, most of them did have evidence, when you look at their face immediately afterwards, that was very clear from the videos.

Q. So, what is the point that you're seeking to draw out in observing at the point in time that the children were referred that there had been an event but they didn't die, was it something in that which you wanted to elaborate on in terms of physical damage?
A. Yes. These children were - their circulation continued throughout this period. They did not die. If you have discoloration and damage to the tissues and the circulation stops, then the colour does not return, it doesn't go away. It goes away and it went away after 20 to 30 seconds in these children, because their circulation continued. If the children had died it would not have gone away, it would have still been there, because we don't - there's nothing to clear

that damage to the local circulation if the circulation has stopped. I'm sorry, I've not been very clear perhaps.

Q. No, it's clear, thank you, Professor. This exercise in relation to the video surveillance, it appears that it all involved children who were more than two months only, and I'll come to it in a moment, but you differentiate your view as to the likelihood of damage as between a very young infant of a couple of weeks versus an infant older than that.

CALLAN: If we could have Professor Fleming back on the screen. I have finished with that article.

Q. Professor Fleming, that's dealt with in your report, page 27, red page 34, around line 815. You say you accept it may be possible in infants in the first weeks from birth, you find it hard to accept, however, that three children aged eight, ten and 19 months would have had no physical findings to suggest smothering identified at post-mortem. Is that view based on the presence of erupted anterior teeth only?
A. Predominantly, but also that the children - the vigour with which children fight to protect their airways, in which we saw in the covert videos, is quite extraordinary. They wriggle very, very vigorously. And if the allegation is that someone is smothering them in a fit of rage, the fact of an adult doing something in the conditions of rage and the child fighting vigorously for their life doesn't lead to any injury, I find very hard to believe.

JUDICIAL OFFICER

Q. To what extent is the assumption that it's done in a fit of rage important to that conclusion?
A. Well, that's the assumption made at the trial, as I understand it, that was what was at the trial and that's what I was--

Q. I appreciate that, but I'm just asking you how important is that particular assumption to the conclusions which you've reached?
A. I personally would find it almost impossible to believe that a child with erupted anterior teeth who is having their airway obstructed by something put over their face, would not fight vigorously and injure the inside of their lip. I just find it very hard. I can't say it's impossible, but I would say that my experience of resuscitating children of that age is that it's very hard to do anything without damaging the inside of the lip on the anterior teeth.

Q. But the two children of the youngest age, didn't have those teeth as I recall?
A. Patrick was eight months, Laura was ten months - sorry. Sarah was ten months and Laura was 19 months at the time of their deaths, the youngest, Caleb, was three weeks, and that's why I said I have less certainty about that.

Q. But if you take the fit of rage out of it, do you agree it's possible that - let's just take it one by one - Patrick could have been smothered without there being any overt signs of it?

A. If we're talking about the episode when he was four months old, I don't believe - I believe the evidence, the physical evidence at the time excludes airway obstruction as a cause of that episode. I think there is sufficient evidence in the documentation to say this was not an episode of imposed airway obstruction and I pointed that out in my report. Still I think that the initial episode, which was before he had any teeth, I'm - I cannot believe was an episode of imposed airway obstruction because the physical findings just do not match that.

Q. Which physical findings are you referring to?
A. The fact that he had - he didn't need resuscitation, he did not have a cardiorespiratory arrest. He was cyanosed and responded very quickly to oxygen. He did not have a raised lactate level and the clinical picture in the period afterwards of recurring seizures really is much more compatible with a acute encephalopathic illness rather than an airway obstruction.

Q. In relation to Sarah, again taking out the assumption of fits of rage, do you regard it as possible that she could have been smothered without any traces being left?
A. I always hesitate to say that something is impossible. But I would say I find it very, very hard to accept that that could happen. I can accept it is possible. I couldn't say it's impossible. I'd be very, very surprised.

Q. And would you say the same with Laura, the eldest?
A. Yes. As I say, it's - nobody had enough experience to know what is - under what circumstances it's possible to do this for obvious reasons. But knowing the vigour with which young children protect their airways, I would be extremely surprised if - if that were the case.

Q. And would you be even more surprised if one particular person managed to smother three children without leaving a trace?
A. Absolutely. I would find that extraordinary. That person must have skills that most paediatric anaesthetists don't have in terms of protecting the airway.

Q. Assuming for the purpose of that answer, the children were of the age and physical condition as you've assumed it, of Patrick, Sarah and Laura?
A. I'm really sorry. There's a break in the transmission there. Sorry.

Q. I asked you that question generally. If we can do it specifically in relation to Patrick, Sarah and Laura, your answer would be the same having regard to their physical condition as it was reported to you for the purpose of this report?
A. Yes. I would find it very, very, very hard to believe that somebody could suffocate them by putting something over their face or obstructing their airway and leave absolutely no marks in any of them.

Q. Professor, I have to ask you, are you an outlier in this area? I only ask that because the experts at the trial were almost unanimous that the absence of any signs supported the hypothesis of smothering and in the earlier Inquiry in relation to Ms Folbigg there was no evidence which suggested anything to the contrary.

A. My response to that is that at neither the trial nor the prior previous Inquiry was a paediatric intensivist asked that question. And as a paediatric intensivist, my experience of dealing with airway obstruction is - I've dealt with it a lot and having personally investigated very large numbers of deaths and being the first person on scene, if you like, actually at the point of resuscitation, again I - I may be an outlier but I think without being immodest, I have a greater experience of those areas than the experts who gave evidence at the trial or the previous Inquiry.

Q. Does Dr Berry have expertise in that field?
A. No. Professor Berry was a pathologist, not a clinician.

CALLAN

Q. Professor Fleming, just following on from those questions from his Honour, in the instances as an intensivist that you've responded to child deaths have you ever encountered an instance that you regarded as a suffocation event, that is an imposed upper airway obstruction?
A. Yes. Yes, I have.

Q. In that category were there any which were, if I can describe it as, non natural causes?
A. Yes. I have - I have and, indeed, the - in the instances where I have come across that, the striking finding was usually that there was evidence of injury to the face or to the mucosa, which is what pushed us in the direction of that conclusion.

Q. How do you, if you do, distinguish between injury as a result of resuscitation as opposed to injuries potentially sustained as a result of the suffocation mechanism?
A. It depends on what injuries we're talking about. Certainly you can injure the lips and particularly by pushing the lips onto the gum or the teeth during the resuscitation. That's very common. But the finding, for example, of mucosal tears or petechial haemorrhages don't arise from resuscitation.

Q. In relation specifically to Laura Folbigg, if you assume that there were immediate resuscitation efforts deployed and there is no indication of injuries sustained, does that tell you anything one way or another as to the likelihood that she might have sustained injury during a suffocation?
A. No. Again, it's all I can say is that we sometimes do injure children in the resuscitation, but it's - I couldn't - a child is not wriggling when they're being resuscitated. But I - I couldn't say that that was in favour or against the possibility of injury in the act of suffocation.

Q. You have pointed out the difference between your area of specialisation as a paediatric intensivist and the areas of expertise represented in those who gave evidence at the trial and then the 2019 Inquiry. Do you accept that a forensic pathologist is in a different position to you in terms of making observations and ultimately expressing views as to cause of death and associated signs of cause of death?

A. We're looking at it at a different stage. My investigations and my - so, for example, if I explain. In our largest studies, the big - the national study, the CESDI study in the 1990s where we were investigating the causes, the median time from a child being found dead to me or one of my team being physically present with the child and examining them was less than four hours. Which is very - very - much sooner than the standard time at which any pathologist would be looking at the child. So, we had the advantage of being there very quickly, and that's an important part of our current protocol; to look very quickly and very soon after the event because things do change. So, it's not saying that one's better or worse than the other. It's just that the sooner one can see things, the more reliable the findings are.

Q. Professor, are you aware of a publication titled: "Forensic Pathology". The authors are Saukko and Knight. The extract that we have is from the 2004 version. It's set out in the report prepared by Professor Cordner you've seen.

CALLAN: Your Honour, it's Exhibit 2-C. Red p 277.

EXHIBIT 2-C SHOWN TO COURT

Q. To be clear, this is an extract from that book, Professor Fleming, do you see the words, "the starting point is set out in Saukko and Knight (2004)".
A. Yes.

Q. Is that a text with which you're familiar?
A. I'm not - I can't remember what the particular text was, but the - the content is the sort - is certainly something very familiar to me.

Q. It commences; "In relation to infants… it is essential to appreciate that smothering, whether intentional or accidental, is both rare and difficult to prove." Would you agree with that from the perspective you have as a paediatric intensivist?
A. Yes. Oh, absolutely.

Q. It continues; "The so-called classic signs of asphyxia (ie facial congestion, facial and conjunctival petechiae), for what they are worth, are rarely present in proven suffocation". Do you - from your perspective as a paediatric intensivist, agree with that statement?
A. Yeah, I mean it's - the problem is how do you prove suffocation? This is the difficulty. If the - if the decision about whether this was suffocation or not is based on the observations of the pathologist or whoever's investigating, and then they try to distinguish between the ones they called suffocation and the ones they didn't, then it's a tautology. It's a - it's not a valid argument. If on the other hand you're dealing with, for example, as I have on occasion, a child who's had a plastic bag over their face where there's absolutely no question this was asphyxia, then that's a different question and it does depend on the nature of the original observations and what the circumstances were as to how you interpret them.

Q. Does it also turn on the nature of the form of suspected suffocation?

A. Yeah. No, it's - as a - it is a very difficult question. And I think the point there is it's rare and difficult to prove, and that - you know, there are very rarely instances where one can say absolutely for certain this was suffocation. And the only instances I can think of where I've been absolutely clear in that were instances where a child had a plastic bag over their head, for example. Which is - you know, I have seen, and we've had a number of deaths of young infants with very soft plastic nappy packs over their face. And there, one can be quite clear this is a - this is asphyxia. Imposed asphyxia, if you like, by the outside item. But mostly, it's not that clear.

Q. The statement that I've just read to you says, "are rarely present in proven suffocation". Are you in a position, one way or another, to indicate if you would agree or not with that statement?
A. No, I agree with that. I mean, I think - the point I was getting at is how do you prove suffocation? And that's what I'm not sure about where the - where this question - where this particular statement, what the observations of this individual or this group of individuals were. But I would certainly agree with the concept, and yes, you don't often see those findings in young infants.

Q. To the extent that, for instance, a forensic pathologist who gave evidence in 2003 or indeed at the 2019 Inquiry, used this as a source, that distinguishes their perspective from your contribution to this Inquiry.
A. I'm not quite sure what you're saying. I mean, I have seen--

Q. Well, if they rely on--
A. I have seen--

Q. --this and have made statements to a similar effect, then--
A. Yep.

Q. --do you accept that there was a basis for doing so?
A. I'm not - I'm not - I suppose what I'm trying to say is that I have seen a very large number of dead infants very shortly after they have died, and have investigated the scene and circumstances in which those deaths occurred. And I would agree that facial petechiae are really uncommon, very uncommon, and I don't know what they would - if we saw them, and there was nothing else to suggest suffocation, I don't know what I would - how I would interpret that because they're very uncommon, and nobody has enough understanding of when they might occur to be able to be clear about what might have caused them.

Q. The passage continues, "as intrathoracic petechiae are common in undoubted ‘cot deaths’, these signs cannot therefore be accepted in isolation as evidence of suffocation."
A. That I strongly agree with. You know, the intrathoracic petechiae are very commonly found and they are found in all sorts of circumstances where suffocation just absolutely could not be possible. So, I do not understand what causes them, and that's part of a lot of the research that our - our group has been involved in. But I would agree with that statement.

Q. I've finished with that passage, thank you. In terms of, what I might describe as recognised forms of observable physical damage caused by imposed upper airway obstruction, you've already addressed facial and conjunctival petechiae, which as you observed is really uncommon. Otherwise, is temporary evidence of discolouration to the face from imposed airway obstruction an observable form of physical damage caused by such obstruction?
A. Yes. The point I was making about the temporary colour change that was seen in this Southall and Samuels paper is that this was after a few seconds. You know, 20, 30 seconds maximum. The circulation continued, so the discolouration, the temporary change, was temporary. It went away because the circulation continued. Had the circulation stopped, there would be nothing to get rid of those marks. They would remain.

Q. You'd expect as an observable or recognisable form of damage caused by upper airway obstruction, some discolouration to the face?
A. Well, what we - what was seen in the videos that I looked at was there was temporary discolouration around the face and nose in those - in those infants. And as I was pointing out, that the - the imposed area of obstruction on those infants was for a few seconds, and the circulation continued. When the circulation continues, the discolouration disappears.

Q. That observation as to discolouration is a product of the mechanism of suffocation that was used in those instances?
A. That's correct. I believe so, yes.

Q. If you were talking as you've described for instance, as another mechanism, a plastic bag, that you wouldn't necessarily expect any such discolouration?
A. No. I mean, as I say, it was that there was a pressure on the face and the pressure caused temporary change in colour.

Q. You, as I read your report, place particular emphasis on injury to the insides of either the upper or lower lip or tongue and that relates to the presence of erupted teeth.
A. Yes.

Q. Does that form of injury again depend on the nature or method of suffocation involved?
A. Well, to be honest, I don't know, but then nor does anybody else because the studies can't be done. But if you're applying pressure to the face in some way in order to obstruct the airway, there has to be pressure on the lips or the mouth of some sort to stop air going in and out of the mouth. And I find it very hard to envisage a way you can do that that doesn't push the lips onto the teeth.

Q. What about oronasal blood or blood stained frothy fluid?
A. That's extremely common. It's most common in younger infants. And it's - frothy blood stained fluid at the time the child's found is most common in younger infants, under two or three months. And in the most recent study, that

I - I appended to my third report, we found that that was present in 49% of sudden unexpected deaths of infants in England in the year 2000, 2020, sorry.

Q. Do you distinguish between a finding of frank blood at the external airway versus oronasal secretions, such as what you've described which is common in SIDS.
A. Yes. I mean, I think it's very rare to find frank, overt blood, and if you do, it's one of the things which might suggest there is a - there'd been a physical injury of some sort. But it's - it's very difficult to distinguish frank blood - well, if there's frank, overt bleeding, that's unusual. But some blood, or blood stained fluid is very common, and what you can't be sure of is whether the blood stained fluid was originally just blood which has now got mixed up with other secretions, or whether it was always mixed with the secretions.

Q. Can I move on to the view that you've expressed as to the cause of death in relation to each of the four children--

JUDICIAL OFFICER: Ms Callan, before you do, Professor Fleming has described himself as a Paediatric Intensivist. That doesn't seem to be - the particular area of expertise doesn't seem to be spelt out with great clarity, I wonder if you could clear that up with the Professor.

CALLAN: Yes, certainly.

Q. Professor, in describing yourself as a Paediatric Intensivist, could you assist the Inquiry, perhaps by reference to your curriculum vitae, as to the nature of your clinical experience and expertise?
A. Okay. I was appointed as a Consultant Paediatrician in 1982 and was Head of Paediatric Intensive Care at Bristol Children's Hospital from 1982 to 1996. So, I ran - I was the Senior Clinician running the Children's Intensive Care Unit. As part of my responsibility in that role, I was involved in very many of the resuscitations of children brought in having been found apparently dead in the community, and spent a lot of time working with those children and their families. Over the past - since the late 1990s, I have no longer - I moved out of practice in acute paediatric intensive care and moved into - I remained in neonatal intensive care, but I also established a service for children who are technology dependent, particularly ventilator dependent children at home in the community in the south west of England. I ran that service for many years. But as part of my research commitment over that period of time, I ran the sleep and physiology laboratory in Bristol. I established it in 1978 with a grant from the US government and ran physiological investigations of children who presented with unusual episodes and also carried out quite a large volume of basic physiology research in young infants.

I also established with Professor Berry, the Avon Mortality Study in the early 1980s, which collected information on every child - every infant death in the Avon county area, about 1 million people. Subsequently expanded to the whole of the south west region of 5 million.

Q. I would like to ask you a bit more about that. Up until the point in time that

you commenced that work with Professor Berry in relation to unexpected childhood deaths, is it accurate that your experience running the Children's Intensive Care Unit and otherwise your focus was on children who were alive albeit often very gravely unwell?

JUDICIAL OFFICER

Q. Or capable of being resuscitated?
A. They've come into hospital being resuscitated. In fact, many of them were dead already and they would - you know, they would arrive and I would be, sadly, the person who would confirm that they were dead. But yes, I've been involved in both direct clinical epidemiological and physiological research into unexpected deaths of infants and young children since 1977. If you look at my CV you will see there are several hundred publications in relation to those - those areas of expertise.

CALLAN

Q. The work that you've done with Professor Berry, you describe that in your CV, your Honour at red page 67 in Appendix 1, you say, "From 1980 to 2007, I provided an immediate support and counselling service for all families in the county of Avon, bereaved by sudden death of an infant or child," and you go on to describe working with the families' primary health care system. In that capacity were you involved in any sort of clinical side or the investigation of the circumstances of the death of those children?
A. Yes, that's precisely what I was saying. The 200 deaths that I personally investigated, many of them were from that. If you look at my CV the paper published in the Lancet of 20 years continuous investigation of all unexpected deaths in Avon county, almost all of which I did - I was the lead clinician for. So, yes, I was directly involved in the investigation. I helped identify what investigations were needed, contributed both nationally and internationally to the definitions of what is appropriate investigation.

Q. Professor Fleming, you address in your report your views as to the cause of death of each of the children and you've also spent some time on Patrick's ALTE. You commence setting out your views in that respect at page 18 of your report, red page 25. I did want to ask you a preliminary question though as to what you've done in your report by reference to each of the children. The question that you were asked appears at the top of that page in bold text. Do you see number 3?
A. Yes.

Q. You were asked to assume current cause of death and then you were provided some assumptions to make as to the current cause of death diagnosis of the four children and you were asked, "in light of these diagnoses what role does SIDS risk factors have to play in the deaths of all four children?" You commence your answer to that question at line 528 with the words, "As stated above," and as I read it, your response is that as to SIDS risk factors, is the two sentences there, concluding with the sentence, "For the individual family in the absence of such identifiable risk factors, the use of such

factors is of no value whatsoever." Do you see that there?
A. Yes.

Q. You go on to say, "With regard to the potential causes of the children's deaths, I set out my comments below." Why have you commented on the potential causes of the children's deaths in circumstances where you were not asked to do so?
A. The question that I was asked was to deal with - was related to SIDS risk factors and making those assumptions about deaths. I was not willing to make assumptions about a cause of death without explaining why I came to that conclusion, so that's what I've done. I've explained my interpretation of - I mean, I think firstly, Caleb, SIDS category 2. As I explained elsewhere in my report, the San Diego definition which that comes from is not used in the UK. The system used in the UK is similar but slightly but significantly different in that it looks at a much wider range of - of factors involved, so, I was trying to clarify. And I think the use of the term “sudden infant death syndrome” is often confusing rather than - rather than helpful. So, if a death is unexpected and unexplained, it meets the diagnosis, it meets the definition, provided the relevant investigations have been done, which in fact in none of these four were they done, so that was my reasoning.

Q. Your position by reference to the information you had available to you, which I've already referenced and is set out in your report as to Caleb's death, is really stated in that first sentence under his name in bold, that is, "Neither the clinical history nor detailed pathology examination of Caleb established a clear and sufficient explanation for his death, which thus remains unexpected and unexplained."
A. Correct.

Q. And in relation to laryngomalacia, as I read your report, because the post-mortem did not include a histological examination of the larynx, from your perspective that doesn't exclude the diagnosis and you can't take it any further than that?
A. Correct. I mean, the examination of the larynx was superficial. It was less than half a sentence.

Q. Could I take you to your executive summary report of 3 November, red page 7-2? Under the heading "Caleb" you commenced a clinical history strongly suggestive that Caleb had a mild to moderate degree of laryngomalacia. You point out, "The post-mortem examination which did not include examination of the larynx did not exclude this diagnosis." What is the basis for your description of Caleb having mild to moderate degree of laryngomalacia?
A. Well, the comments by the paediatrician, which I quoted in my third report, that when he was lying on his back he had a degree of airway obstruction and - and thoracic recession, particularly when he was lying on his back. Laryngomalacia is very common. It's usually a benign condition, as I've mentioned.

Q. In the absence of an examination of the larynx on post-mortem, you don't

raise it any more than being a possibility. Is that the case?
A. That's the case, yes. I mean, I can't say - I was responding to comments by one of the forensic pathologists, that laryngomalacia was irrelevant and I don't agree with that because I have seen children become quite seriously ill with laryngomalacia and have major - significantly life-threatening events. But it's very uncommon for that to happen. But the description of the larynx at the post-mortem, the post-mortem report, and I quote, says, "Larynx, trachea and bronchi were unremarkable." Now, I don't consider that to be evidence that they've been looked at other than very superficially and therefore I can't comment on whether it was normal or not.

Q. Can I take you to the views that you've expressed in relation to Patrick? I'll take you back to your report of 11 October. It's page 19 of your report, red page 26.
A. Yes.

Q. I'll come to the reasons why you form this view, but in terms of the ALTE, so described, which involved an episode on 18 October 1990, you've expressed the view at the bottom of page 23, red page 30, that you agree with the opinion of Professor Ryan, Paediatric Neurologist, that the features seen in Patrick were more suggestive of an underlying condition rather than a hypoxic ischaemic injury.
A. Yes.

Q. That being your view, I just wanted to work through with you if you could assist us with the considerations which informed that view that you've formed. But first, records before the Inquiry and opinions expressed by a number of others that prior to the event on 18 October 1990, Patrick could be described as a healthy and normally developing baby. Is that your view?
A. Well, there was no - he had not been reported to have suffered from any identified significant problems prior to that, is my understanding. I haven't seen his full primary care records. They were not part of the records that I was supplied with, but I'm not aware of any issues that had been raised before then.

Q. In terms of the clinical and pathology evidence, which from your report it appears leads you to conclude that Patrick did not sustain a severe hypoxic ischaemic injury. You first referenced the fact that no resuscitation was required. What is the relevance of that in terms of informing your view about the possibility that a hypoxic ischaemic injury was sustained?
A. Okay, if - if a child has suffered such an injury to the brain that they develop cortical blindness and all the other neurological problems that Patrick had, that initial episode must have been very severe. When the - at the time of Patrick's presentation, he was noted to be blue, but he was breathing. And the report, both from - that I've seen, the reports from his parents are that he was breathing when they found him. He was struggling, but was breathing. If you've had a sufficiently severe brain injury to lead to permanent long-term serious neurological consequences, that is extremely surprising because the effect of shortage - severe shortage of oxygen, severe acute shortage of oxygen and blood circulation to the brain, is that many vital functions,

particularly breathing, stop. And without resuscitation, the child would have died.

But he didn't need resuscitating. He was, I believe, given mouth-to-mouth, according to his father, at the time, but was breathing at the time. And the report given by his mother to both the - both the ambulance crew and to the emergency duty doctor in the hospital was that he had never been given any significant - any resuscitation. So, it suggests that whatever would have happened to him had - he had an episode of some sort, but it was not one that is likely - would be likely in itself to have caused permanent long-term neurological damage.

Q. The description on presentation of being blue and the administration of oxygen, would they accord with describing Patrick as hypoxic on presentation?
A. Yeah, he was - he was blue on arrival, on the arrival of the ambulance crew. One of the - when you become short of oxygen, the body - when a person becomes short of oxygen, the body shifts the blood supply and the oxygen supply that's available to the two most important organs, the brain and the heart. Everything else can be shut off, but the brain and the heart have to continue to be supplied with oxygen and circulation in order to survive. So, the fact that somebody is blue does not mean that their brain is short of oxygen, it means that their skin is short of oxygen and the body is likely to be using as much of the available oxygen as possible to keep the brain and the heart going. If you recover quickly or your colour improves with just administration of oxygen, that is very much against there having been a very severe shortage of oxygen to the brain. Shortage of the oxygen to the brain leads to a loss of consciousness, a loss of responsiveness and eventually, a cessation of breathing.

Q. Can I ask--
A. But the fact that he was blue; yes, he was hypoxic, he was short of oxygen, but I don't think that was sufficient to have caused brain injury because of the rapidity with which he responded to just being given oxygen.

Q. Can I take you to what I read as a second consideration which informs your ultimate conclusion. It's at page 20 of your report, red page 27. At first consideration as to not requiring resuscitation is dealt with around about point 585, and then at point 590, you observe that Patrick's condition - within a few minutes, his condition improved. You then say, "according to the handwritten notes made by Dr Dezordi, the blood sample taken at the time of admission did not show increased blood lactate level. I just want to be clear about what clinical records you're relying upon for your understanding that the blood sample taken at the time of admission did not show increased blood lactate level. You referred to a document in the forensic pathology bundle, and I'll have that shown on the screen, if I may now.

CALLAN: Your Honour, in our proceedings, it's Exhibit 2-H, red page 3594.

EXHIBIT 2-H SHOWN TO COURT

Q. Professor, do you see that says handwritten “Interim Discharge Letter" under the hand of Dr Dezordi?
A. Yes.

Q. And it indicates, "Date of Admission: 18 October 1990, Date of Discharge: 29 October 1990". This appears, as best as we can identify it, to accord with what's described in your report as being page 87 of the forensic pathology bundle when you look at the PDF equivalent.
A. That's correct, yes. That's correct.

Q. And is that the document that you're referring to as the basis upon which you understood the blood sample taken at admission did not show increased blood lactate levels?
A. Yes, and indeed the following few pages are the original notes written at the time of admission. The next big page is 81 to 84.

Q. Yes, and we can take you through those.
A. But yes, that was the document I'm referring to.

Q. Do you see against the heading "Information Given to Patient", which is a pre-printed aspect of this, but the handwriting appears to contain detailed description by Dr Dezordi of relevant observations of Patrick. Do you see against the word "Serum"?
A. Yep.

Q. Reads: "lactate, ammonia, calcium", is it, "magnesium and glucose all normal".
A. Yes.

Q. And then there's then a reference to the “Urine metabolic screen”.
A. Yeah.

Q. Was that the part of the handwritten note that caused you to understand that blood sample, taken at admission, did not show increased blood lactate?
A. That's correct. I mean, the only - the only record I could find within the records, and these were supplied to me only just a week or so ago, was that a blood lactate was done on the - two days later. But I haven't - I didn't see the results, but my assumption is that a child who's admitted in that condition would have had a blood gas sample taken at the time of admission. The result is not there in the records. I couldn't find it. But blood gas samples routinely have a lactate measurement on them.

Q. You say you’ve quite recently seen the laboratory results from, relevantly, a blood lactate for Patrick dated 20 October, that is two days later?
A. Yes. As I said, it was two days later. It was at 9 o'clock in the morning on the 20th the sample was taken that's recorded in the laboratory records. But that is--

CALLAN: Your Honour, for the record, that's at Exhibit 2-AL, red p 6195.
Q. So, did you assume when Dr Dezordi, in his handwritten note, wrote

Serum: lactate et cetera “all normal", did you assume that that was a reference to a blood sample or test administered at the time of admission?
A. I did. I made that assumption because all the others measurements there, the glucose, calcium, magnesium, there'd be no point in measuring them at any time other than at the time of admission.

Q. I might take you to the lab results.

CALLAN: It's at Exhibit, as I said, your Honour, 2-AL, red p 6195.

EXHIBIT AL SHOWN TO COURT

Q. Is this the document that you're referring to, Professor?
A. Yes, that's right. It was the 9 - the lactate taken at 9 o'clock in the morning on the 20th. That's--

Q. And we can see from the first column that there were bloods taken on 18 October. There's a question mark against the time, "5.59".
A. Yes. I mean, I believed he arrived at the hospital at just before 6 o'clock, so that makes sense.

Q. And that did give rise to tests in respect of, amongst other things, glucose, magnesium, but not lactate. Is that how we should read that column?
A. That's correct. I mean, those - those - as I said, I didn't see this particular document until, I think it was last week. It was supplied to me as part of the previously unsupplied - not supplied documents.

Q. Does that indicate, at least in terms of the blood that was taken on 18 October, that so called extra blood tests for lactate, at least certainly isn't recorded here, in terms of the position on 18 October?
A. That's correct. I mean, my assumption from the note written by the doctor that I was referring, the one that we just looked at, was that blood sample would have been taken routinely. It has been for the last 25, 30 years at least. Routinely, at this setting, you take a blood sample for blood gasses to measure PH, oxygen level and carbon dioxide level, and those samples would routinely also contain - give you information on the lactate. Blood gas analysers give lactate as well. In 1990, not all blood gas analysers gave lactate levels, but in most - most settings, they probably would.

Q. Insofar as that the blood test on 20 October indicated a blood lactate level of 1.6, does that tell you anything about the position as it had been on 18 October?
A. No.

Q. You explain in your report, the significance from your perspective of a lack of elevated blood lactate levels, that is necessarily based on an assumption that the blood lactate levels were normal at the time of admission?
A. That's correct.

Q. So, if you assume they were normal, you set out your view that this is

strong evidence the initial episode was not a severe hypoxic ischaemic insult. Can I ask you if against that assumption there is no evidence of a blood lactate level as at 18 October on admission, does that make your evidence on that subject in a sense neutral, we just do not know?
A. Yes. I can't - as I said, from reading the note I had assumed they were done at the same time because that's the implication from the way it was written. If there was no lactate measure done at the beginning, then that particular argument cannot fly. It's not - I can't answer it one way or the other. What I can say though, is that there is no evidence of renal impairment, kidney damage. If he had a hypoxic ischaemic episode sufficient to cause brain damage, there is always damage to the kidneys as well because the body will sacrifice the kidneys to save the brain and the heart. So you will get injury to the kidneys, you'll get a rising creatinine level and particularly you'll get abnormalities of urinalysis, particularly blood in the urine.

Q. The absence of evidence of any renal impairment, does that tell you anything one way or another as to the blood lactate levels at the time of presentation?
A. No. It's a different issue. If anybody suffers a sufficient hypoxic ischaemic injury to cause brain damage, it will always also damage the kidneys. Indeed, particularly in looking at litigation for injury in children, in young children, if a hypoxic ischaemic episode is reported to have caused brain damage but there was no kidney damage, in many jurisdictions that would be considered to be evidence that the brain injury was not severe because you don't get severe brain injury in the absence of kidney damage from hypoxia ischaemia.

Q. Can I take you back to another observation you make, which is the elevated urinary glucose level and the absence of high blood glucose level.
A. Yes.

Q. You deal with this on page 21 of your report, red page 28. Around about line 625 you say, “That is not easily explicable.” You go on to say, “It’s certainly not suggestive of the predictable consequences of an episode of acute hypoxic ischaemic injury.”
A. That’s correct.

Q. Does that presence in urine and absence in blood exclude the possibility of an acute hypoxic ischaemic injury?
A. It makes it extremely unlikely. When you have an acute hypoxic ischaemic injury, the blood sugar rises, it goes very high to start with, it's a stress reaction. And in the absence of oxygen, which is what you have in a hypoxic ischaemic injury, the brain can't utilise much of that glucose. It utilises some of it and converts it to lactic acid, to lactate, but it can't utilise much of it and therefore the sugar level stays very high. Typically in a child who's had an acute hypoxic ischaemic injury, the blood sugar level is very high. If on the other hand, the initial episode is a seizure, a fit, and there is a continued supply of oxygen to the brain, the brain will metabolise glucose at a vastly accelerated rate compared to normal because of the seizure. The seizure activity is utilising a lot of glucose. It's the primary substrate utilised in seizure activity in the brain. So that even although the initial stress may have led to a

rise in blood sugar, some of which will have escaped into the urine which is why you get glucose in the urine, if the glucose in the blood is being utilised by the brain first off it tells me that the brain is not hypoxic because the brain can't utilise much glucose when it's hypoxic and, secondly, it explains why you've got a falling glucose level in the blood despite having - there's been some evidence that it was previously high in the presence of glucose in the urine.

Q. That point was one that was made in the witness statement of Dr Dezordi back in 2000, where he says, "Interestingly however, there was significant glycosuria in the absence of hyperglycaemia." Is that another way of describing what you've just written?
A. I think he was misinterpreting it actually. He was saying that this was evidence of a hypoxic ischaemic episode and it's actually evidence against it.

CALLAN: Your Honour, for the record, that's a document that's in a forensic tender bundle, Exhibit 2-H, red page 3590.

Q. The fifth factor that you reference in support of your ultimate conclusion that this was not a hypoxic ischaemic insult, is the speed of recovery of Patrick, if I can put it that way? For instance, feeding well within a few hours. What does the rate of recovery indicate one way or another about the likelihood of a hypoxic ischaemic injury having been sustained?
A. The fact that he recovered very quickly, within a very short period of time he was feeding again. If you've suffered sufficient hypoxic ischaemic injury to cause permanent brain damage, you do not start feeding. That's one of the very characteristic features of hypoxic ischaemic injury in young children, is that feeding is the one of the most difficult things to get going after it. They have real trouble getting started to feed and it often takes weeks or months. So, that is very much - on a very acute episode of something happening from which he made what seems to have been a very rapid neurological recovery followed by a further episode of seizure activity later on. That is very - that would be an extremely unusual course of events in someone who has had an acute hypoxic ischaemic injury.

Q. There is evidence before the Inquiry that if you describe this event as the first seizure it was very different from the subsequent seizures or seizure-like presentations in Patrick that occurred until he died. Do you agree with that description of being very different?
A. The descriptions of the seizures are not very detailed in the records that I've seen, so I would find it - you know, the initial episode had a lot of attention because it was the first time it had happened. The descriptions of the seizures at later times were often quite brief, "another seizure". He'd had a seizure and it was relatively little detail about what exactly happened. So, I'd be hard pushed to say this was different because I'm - the level of detail given about them at different points is very different.

Q. The sixth reason that you give for your view as to this not being a hypoxic ischaemic injury, is by reference to histopathology, that is the post-mortem histology report.

CALLAN: I ask that that be put on the screen. It's Exhibit 2-H at page 3559.

Q. Just to confirm, is that the report that you had regard to?
A. Yes. I mean, I was referring both to the - to the CT scan and to that report. I suppose that one of - one of the important things is that since 1991, there have been great advances in understanding the histological features of hypoxic ischaemic injury in young infants and I did put some references in with my report to explain that. This report shows evidence of old infarction glyosis and laminar necrosis in the parieto occipital area. The laminar necrosis is a characteristic finding in the relatively superficial part of the cerebral cortex and characteristically it's seen after severe hypoglycaemia, a shortage of blood glucose. It may also occur in children who have had prolonged and severe seizure activity, which is not the finding that you typically find after a single very acute initial episode of hypoxic ischaemic injury.

The technical brain histological findings after a single severe initial episode of hypoxic ischaemic injury, is damage to the basal ganglia and the thalami, which are not noted to be abnormal here but normal, and in the CT scan were also not shown to be abnormal. So, these are features which are not typical of a child who's suffered damage from a single past episode. I note the opinion of the person who wrote this report, that they thought this was part of a severe episode of cardiorespiratory arrest the baby suffered. But actually he never suffered a period of cardiorespiratory arrest. That was a mistake. He hadn't had a period of cardiorespiratory arrest. If he had, the findings would have been different to this anyway. And I think that really reflects the improvement in understanding of brain histopathology over the past 30 years.

Q. And that consideration is dealt with in your report, page 23, red page 30, where you refer to the lack of any significant identified abnormality in the basal ganglia or the thalami, you put it as being more suggestive of a progressive neurodevelopmental disorder.
A. Yes.

Q. The use of that term “more suggestive” indicates, as I read it, you don't exclude the possibility of a hypoxic ischaemic injury, but you prefer this--
A. No.

Q. --having regard to what you see in the histopathology report?
A. Yes. In medicine things are very seldom clearly black or white. There are patterns of injury which are more suggestive of one thing than another and the pattern of injury in Patrick, was far more suggestive of a progressive encephalopathic process with severe and persisting seizure activity than it was of an acute hypoxic ischaemic episode several months earlier. I can't tell you it's impossible that there was an acute hypoxic ischaemic episode. That's not - and we don't have enough knowledge to be able to do that. But it was - the pattern is certainly much more suggestive of a progressive encephalopathic process.

CALLAN: Your Honour, for the record, this a reference from the report of Dr Cala at tab 13-04, red page 55.

Q. Dr Cala has suggested that the brain examination showed no features of an underlying metabolic encephalopathy or of any other abnormality of brain development where epilepsy may be a feature--

JUDICIAL OFFICER: Sorry, what page?

CALLAN: This is red p 55, Exhibit 13-04. Top of the page.

Q. Professor Fleming, do you agree or disagree with the suggestion that the brain examination showed no features of an underlying metabolic encephalopathy?
A. No. I think it's - it's far more suggestive of that than of an acute hypoxic ischaemic episode. Sadly, I've had the opportunity to be present at the examination of the brains of children who've had - a lot of children who've had major hypoxic ischaemic injuries. That's part of what we do in paediatric intensive care, is to try and care for said children. But this - the appearances that were seen, that were described in Patrick, were not those that I would have expected to see in that - in that setting.

Q. Can I go back to some studies that were undertaken during Patrick's life. There was an EEG administered on the day of admission on 18 October which was reported as being normal. Professor Ryan, in her report to the 2019 Inquiry - your Honour, it's at Exhibit 2-AJ - observed that had Patrick sustained severe hypoxic ischaemic insult on the morning of 18 October, it was difficult to imagine the EEG would have been entirely normal. Do you have a view in that respect?
A. No, I completely agree with Professor Ryan. I didn't draw that out in my report, but certainly one of the things we routinely do now in children who've had what may have been an acute hypoxic ischaemic episode is to monitor the EEG. And you'll note from my CV, I've published quite a lot on that subject. But actually the EEG is a very useful way of monitoring what's going on, and normally an EEG within a few hours of an episode like that makes hypoxic ischaemic injury very unlikely.

Q. The only other topic that I wanted to raise with you is as to Laura and myocarditis. You deal with evidence, and your view as to her cause of death from page 24 of your report, red page 31, and over the page, page 25, red page 32, line 735, you address the position as it relates to acute myocarditis - did you inspect any slides or otherwise have regard to primary evidence in terms of forming a view as to the extent of myocarditis in Laura?
A. No. I would always defer. In interpreting the slides, I would defer to the expert histopathologists. But my understanding was that there was an agreement amongst all histopathologists that there was a degree of viral, or probably viral myocarditis. The severity is differently assessed by different pathologists, but nevertheless, I've not seen a report by any of them that says there was no evidence of myocarditis.

Q. You used the term "acute" myocarditis, you agree there's a--
A. Acute just means it's just - it's not been there long. Acute - acute means something that's happened very recently rather than something that's been

there a long time.

Q. If you proceed on a description of that myocarditis as being "patchy and mild", does that affect what you go on to say about the possibility of myocarditis as a cause of death?
A. No. So, it's - children - the degree of myocarditis as assessed histologically is not a good indicator as to whether there will - it will trigger an acute cardiac arrhythmia, an abnormality of cardiac conduction. And indeed, as I mentioned, I have on several occasions come across very sudden collapse and death of young children where there is - where there was a presence of myocarditis which was not assessed as being severe or chronic, and which where the death was almost certainly due to an acute cardiac arrhythmia. I have - a particular instance I dealt with relatively recently where a child, a three-year old, was running around the kitchen, and obviously not for a respiratory infection, but was running around the kitchen playing with her father, suddenly fell on a heap on the floor and died, and that was a child who had acute myocarditis. And almost certainly this was an arrhythmia causing the death.

CALLAN: Thank you, Professor Fleming. Those are my questions. I note that it's past midnight at your end in the United Kingdom, and I'm grateful for your continued attention.

JUDICIAL OFFICER: I rather thought it was 10 to 11, but I'm certainly also grateful.

NO EXAMINATION BY DR WOODS

<EXAMINATION BY MS HORVATH

Q. Professor, can you hear, possibly even see me?
A. I can hear you, and I think I can see you at the back, yes.

Q. Excellent.
A. Okay, you're focused on now.

Q. I appear for Dr Cala in this Inquiry. The first thing I wanted to ask you about is, Ms Callan read an extract to you from Knight's Forensic Pathology, the third edition, and asked you whether you agreed or disagreed with certain aspects. I wanted to ask you some similar questions. It's from the fourth edition, although it follows precisely from where Ms Callan was asking you questions. So, you recall that Ms Callan asked you whether you agreed or disagreed with the proposition that the so-called classic signs of asphyxia are rarely present in proven suffocation, and my recollection is you agreed with that, yes?
A. Yes, but with the proviso that proving suffocation is extremely difficult.

Q. The text goes on to say that pressure marks on the face can rarely be distinguished from post-mortem postural changes. Is that something with which you agree, and I don't mean to be disrespectful, or is it something that you don't have experience with?

A. Actually, I would say I probably have more experience than anybody - than almost anybody of that. Because I see the babies very soon after death, and one of the issues is that things change over the period after death. But certainly, if there's been a pressure effect, as I was explaining, that persists for a while. And if you see the child very soon after death, you can still see those pressure effects. So, yes, there are - there may be post-mortem artefacts because almost always after a child has been found, they are placed in a position lying on their back. Nobody ever puts a dead child down on their front, so that any changes that may have reflected the position in which they were found, may disappear by the time that the pathologist sees the child, because changes in colouration and tissue pigment, tissue colours, change after death. Unless the child has been in a particular position for more than two or three hours after death, when those changes become fixed, they will move and all they will reflect is the position the child has been in after the resuscitation has stopped.

Q. You also gave evidence that you found it hard, and I'm summarising so tell me if you disagree, you found it hard to say how children the age of Laura or Sarah could be smothered without there being any evidence, for example, in their mouth. Is that a fair summary of what you said?
A. That's correct, yes.

Q. You're aware, I take it, that for example, in plastic bag suffocation, unless the bag is present, there generally is no forensic evidence of the suffocation?
A. That depends on the age of the child, but yes, I have - and sadly, I have actually been a person involved in trying to resuscitate more than one child after a plastic bag incident. So, yes, I'm very aware of that.

Q. Using the plastic bag as an example, that's a circumstance where there may be no signs whatsoever of the asphyxial cause of death.
A. There are no signs of asphyxial cause of death in general terms. I mean, there are - unless there's been imposed airway obstruction where you see physical marks from it, internally there is nothing that can tell you this child died from imposed asphyxia or - because asphyxia is the state of death. Everyone dies from asphyxia. When the heart stops beating, the tissues become hypoxic and it's - I'm not going to - you can't conflate imposed airway obstruction, for whatever - by whatever means, and the word "asphyxia".

Q. I'll make it clearer. Would you agree with the statement that plastic bag suffocation can be rapid and leave no signs whatsoever?
A. I believe that's correct. I've never seen it in an older child, but I've seen it in young infants who've accidentally had plastic bags over their face.

Q. In relation to some other evidence that you gave, you said that blood stained fluid is common around a child's nose or mouth when they are found dead with presumed SIDS; correct?
A. Yes. Children who are found unexpectedly, young infants particularly, and it's most - the younger the infant, the higher the rate of finding blood stained fluid. The difficulty, as I was explaining, is that even although it's blood stained fluid which you see at the end, part of that, there could have been fresh blood

there which is mixed with fluid that comes out of the nose. So, you can't over - it's difficult to interpret the distinction between blood stained fluid and fresh blood, unless there's an obvious injury.

Q. And you would expect, I take it, that if there was a recording of blood and froth around a child's mouth, that that may or may not be the same as blood stained fluid; correct?
A. It depends who's making the description, yeah.

Q. If it is the case that there is blood around a child's mouth, then that is something that would need to be investigated; correct?
A. The commonest setting in which I've seen - I have seen children with blood around their mouth, are children who've died of acute respiratory distress in the neonatal intensive care unit. It's very - the younger the infant, the more common it is to see blood around the mouth and nose. So, I - I - it depends on the - if there's no evidence of an injury, then it's - I would not draw any conclusions from it. As I said, it's extremely common.

Q. But you would understand, I take it, that the blood has to come from somewhere--
A. Yes.

Q. --and a forensic pathologist's job is to work out from where it's come. Correct?
A. The forensic pathologist - well, the blood will almost always have been wiped away by the time the forensic pathologist sees the baby. But yes, part of the job of trying to understand what went on is to see where it's come from. And it's - as I said, it is so common, and very rarely do you find evidence of where it's come from.

Q. You referred to - and I don't think I need to take you back to it - but the report which talked about 49% of children having blood on their face, either at time of death or post-resuscitation. Yes?
A. At the point they were found.

Q. In relation to those children, I think the same report says that 52% of the children were found co-sleeping. Correct?
A. Yes.

Q. And I think 48%, the co-sleeping was described as hazardous co-sleeping. Correct?
A. Correct.

Q. And that, of course, would be a reason why a child may well be found with blood on their face. Correct?
A. No. Wrong. No, that is not correct. That is not correct.

Q. So there's no prospect during hazardous co-sleeping, for example, of an inadvertent injury to the child which gives rise to blood on their face?
A. There is a - there is a possibility, but we did not find - I mean, the striking

thing about blood around the face is that it was most commonly seen in children who were found laying on their back with their face uncovered. That's the typical finding. That's precisely the finding reported for Caleb, and that's what we found in the - both the CESDI study and the recent study we've just published.

Q. One other matter that was raised with you by Ms Callan is the question of myocarditis. You expressed the opinion that the degree of it histologically is not a good indicator of whether or not it has caused a person's death. That was your evidence a moment ago. Correct?
A. Yes. If there's an acute episode which is a cardiac arrhythmia, rather than a cardiac failure, then it doesn't require florid or extensive myocarditis. I've seen this in - the children I've seen it in have had relatively mild to moderate myocarditis.

Q. I was going to ask you the basis for that opinion that was proffered. Is that from your experience or is this something that you have journal articles which are supportive of this opinion?
A. I think I put one journal article in with my report, but yes, my personal experience is that - as the case I just described earlier - I have been involved in the investigation of unexpected deaths in children on several occasions that have turned out to be acute myocarditis. Presumed arrhythmic deaths, because there's no evidence of cardiac failure or chronic changes in the heart.

Q. Is it your evidence that in those cases that you're describing the myocarditis was mild, as opposed to florid?
A. It was - it depends where you look. As I say, there is a huge range of opinion amongst the forensic pathologists, and I'm not going to get into who's right or wrong, because that's not my field. But they all agreed there was evidence of probable viral myocarditis, and depending on which bits they were looking at, that could be more or less severe. But you know, there was evidence of myocarditis, and some of them, some of the reports suggest that's quite extensive and severe; others, notably Dr Cala, suggests it's less severe. But I wouldn't get - it's not part of my job to interpret the differences between pathologists.

JUDICIAL OFFICER: Ms Horvath, how long are you going to be?

HORVATH: I'm almost finished, your Honour.

JORDAN: Your Honour, I expect one question. It may be a matter we can deal with in submissions.

JUDICIAL OFFICER

Q. I was wrong before, Professor, it is 1 o'clock over there. I apologise both to you and to Ms Callan. Do you mind going for a few more minutes?
A. No, that's fine.

HORVATH

Q. Professor, I wasn't asking you to determine whether the myocarditis seen in Laura was florid or mild. I was just trying to ask you about your evidence that the degree of histological findings is not a good indicator of whether or not the myocarditis has been the cause of a person's death. I think that was your evidence. Correct?
A. My evidence was that you don't - if we're talking about an acute arrhythmic death, which is very different to a cardiac failure death, then you don't need - it doesn't require much virus to affect the conduction system, and cause an acute arrhythmic death.

Q. Is this a matter on which you would defer to the opinions of the expert cardiologists who have given evidence in this Inquiry?
A. It depends if they've had experience of children dying of acute viral myocarditis, and particularly of acute arrhythmia. But certainly my experience, and from my reading of the reported literature, the viral load necessary to cause arrhythmia is not the same as the viral load necessary to cause cardiac failure. And it may be much less.

<EXAMINATION BY MR JORDAN

Q. Dr Fleming, can you hear me?
A. I can.

Q. I appear for the Director of Public Prosecutions. I'm very grateful for your patience. I only have one question, and that is this: to the extent that you refer to the genetic variant affecting the two girls, do you defer to the evidence of other witnesses with specialised expertise in relation to that genetic variant?
A. Do I? Yes, I do. I mean, as you know from my CV I've published extensively on genetic variants in relation to unexpected deaths in infants and children, but I would certainly defer to the expertise of people like Peter Schwartz, who have spent a lifetime investigating that.

Q. And also the other witnesses with relevant expertise?
A. Yes. I think that if - I'm less aware of the expertise of all the other witnesses, but Professor Schwartz I have huge respect for.

JUDICIAL OFFICER: Ms Callan?

CALLAN: Nothing arising.

NO EXAMINATION BY MS LOVE, MR HASTINGS AND DR WATERHOUSE

<THE WITNESS WITHDREW

AUDIO VISUAL LINK CONCLUDED AT 12.01PM
Your Honour, we resume at 2.30 today with the evidence of Dr Monique Ryan.

JUDICIAL OFFICER: Thank you. The Inquiry will adjourn till 2.30.

LUNCHEON ADJOURNMENT

Is Ms Von Reisner present in Court?

CALLAN: Yes, she is, your Honour.

VON REISNER: Yes.

JUDICIAL OFFICER: You have an application, I think. Ms Von Reisner, I've read your application. It seems to me quite indistinguishable from the application you made earlier in this Inquiry which I refused and gave reasons. Can you come up to the bar? I was saying that this application seems quite indistinguishable from the application you made earlier which I refused and in respect of which, I gave reasons for my refusal. Is there any difference to the application?

VON REISNER: Your Honour, difference is only one, that I don't ask in this application to be joined or given opportunity to be a party to this Inquiry. I am filing this application and the Attorney General expressly made a decision on the department website which stating “any person who considers that they have information which may assist the Inquiry should contact the solicitor assisting the Inquiry as follows.”

JUDICIAL OFFICER: Yes, I know that. That was a general call for information to be provided. If you have any information or any documents which you say would be of assistance to the Inquiry, you should give them to the solicitors for the Inquiry so they can consider it. You don't need an order to do that. You simply need to supply it.

VON REISNER: Your Honour, usually this kind of matters are better to make by official application which would be recorded in the proceedings.

JUDICIAL OFFICER: This is an official application, and in refusing your application which I'm about to do, I'm indicating to you that you can supply any information you have to the solicitors assisting me in this Inquiry. Now, that's about as official as it can get.

VON REISNER: Your Honour, does it mean that under *Royal Commission Act* of the Commonwealth, an estate, as a mirror image of Commonwealth *Royal Commission Act*, no any person have right to rise up questions to expert witnesses of any kind?

JUDICIAL OFFICER: No, it doesn't - the Act doesn't mean that. The Act empowers me to give people who have an interest in the Inquiry, in the technical sense, a right to appear or a right to ask questions. You don't have any interest over and above any other member of the public. In those circumstances, I'm not only not entitled not to have you here, I'm bound not to give you leave. So, Ms Von Reisner, I went through this with you on the last occasion. I've given reasons. Those reasons remain the same and I don't propose to make the orders.

All I will say is for the reasons that I gave in relation to your previous

application, I refuse Order 1, Order 2, Order 4 and Order 5 in your application. In relation to Order 3, to the extent that you have written records which may be relevant to this Inquiry, you are entitled to supply them to the solicitors assisting the Inquiry.

VON REISNER: Thank you.

SHORT ADJOURNMENT

Yes, Ms Callan.

CALLAN: Your Honour, before we commence with the evidence of Dr Ryan, can I ask that the application made on behalf of Ms Von Reisner be marked MFI 9?

JUDICIAL OFFICER: Thank you.

MFI #9 APPLICATION MADE ON BEHALF OF MS KOIDU VON REISNER

CALLAN: And in relation to material which may be dealt with within Dr Ryan's evidence, can I tender a current copy of a curriculum vitae? The proposal is that it would be inserted at the back of Exhibit 12-02, commencing on red p 22.

JUDICIAL OFFICER: Thank you.

EXHIBIT #12-02 SUPPLEMENTED BY THE ADDITION OF A CURRENT CURRICULUM VITAE OF DR MONIQUE RYAN, ADMITTED WITHOUT OBJECTION

CALLAN: There are a number of academic articles to be added to the tender bundle. The only one relevant to Dr Ryan's evidence is an article which we tender, and it should be inserted at Exhibit 15, tab 267, which is titled "Hypoxic-ischaemic encephalopathy after near miss sudden infant death syndrome".

EXHIBIT #15 TAB 267 SUPPLEMENTED BY THE ADDITION OF AN ARTICLE BY DR MONIQUE RYAN ENTITLED "HYPOXIC-ISCHAEMIC ENCEPHALOPATHY AFTER NEAR MISS SUDDEN INFANT DEATH SYNDROME" INSERTED AT TAB 267, ADMITTED WITHOUT OBJECTION

I now turn to Ms Roy.

AUDIO VISUAL LINK COMMENCED AT 2.34PM

<MONIQUE MARIE RYAN, AFFIRMED(2.34PM)

<EXAMINATION BY MS ROY

Q. Dr Ryan, for the record can you give us your complete name?
A. It's Monique Marie Ryan.

Q. And your place of work?
A. I work at the electoral office for the electoral seat of Kooyong in Melbourne. I previously worked at the Royal Children's Hospital.

Q. Dr Ryan, strictly speaking you're a Professor but you prefer Doctor?
A. That's right, yes.

Q. You have a medical degree from Melbourne University?
A. I do. I have a medical degree from Melbourne University and a Masters of Medicine from the University of Sydney, and I'm a Fellow of the Royal College of Physicians.

Q. Prior to becoming a member of the Australian Parliament you were Director of the Department of Neurology at the Royal Children's Hospital in Melbourne?
A. That's correct.

Q. Also an Honorary Clinical Professor at the Faculty of Medicine at the University of Melbourne?
A. Yes.

Q. And an Adjunct Clinical Professor in the School of Clinical Sciences at Monash University?
A. That's correct.

Q. At our request you provided an updated CV dated 11 February 2023 which for the record has just been added to the tender bundle at Exhibit 12-02, red page 22.
A. Thank you.

Q. Doctor, can you tell us in your own words what are the areas of specialised knowledge and expertise that you draw upon to express the opinions you have in your report?
A. Well, I'm an experienced Paediatric Neurologist, having completed my training in Melbourne, Sydney and Boston. I worked as a Senior Clinical Neurologist in Sydney and at the Royal Children's in Melbourne for about 15 years up until late December 2021. And in that context I was a Clinician involved in acute and out-patient care of thousands of children I guess with paediatric neurological conditions, including acute care of children with hypoxic, ischaemic and other brain injuries.

Q. Would that include being involved in care from the point of initial presentation to an emergency department?
A. Yes, absolutely. So I would've been on call for many weeks of each year

and in that context would've been called for acute advice on children presenting to the emergency department at the Royal Children's or elsewhere with neurological conditions including acute brain injuries, epilepsy, infectious encephalopathies and other sorts of conditions as well, and then I had on call duties which included the care of children on the wards, within the ICU of the hospitals where I worked for acute complications of neurological conditions.

Q. Would you continue involvement in patient care over the course of a patient's disease, from the initial or key presentation through the progression of their disease over some years?
A. Yes. So I'm sure I would look after children from the time that they presented until the time that they graduated to adult care, usually at some time between 16 and 18 years of age, or unless they died earlier than that from their - complication of their condition.

Q. Your first involvement in the Folbigg matter, if I understand correctly, was when you prepared a report at the request of Ms Folbigg's legal representatives in relation to the 2019 Inquiry, is that correct?
A. That's correct.

Q. For the record that report appears at Exhibit 2-AJ. You then gave oral evidence to the Inquiry on 17 April 2019. That appears at Exhibit 4, page 588 to page 618 which is transcript page 581 to 611. You were briefed with the transcript from the Inquiry for the purposes of preparing your report to this Inquiry, is that right?
A. I was.

Q. You've satisfied that that transcript was a true and correct record of the evidence that you gave to that Inquiry?
A. I am.

Q. You were asked in your report for this Inquiry if there were any matters on which you wished to elaborate which you've done in writing in pages 14 to 15 of your report to this Inquiry, is that right?
A. That's right.

Q. For the record your report to this Inquiry appears at Exhibit 12-02 at red page 9, and that was a report that was prepared at the request of the representatives of Ms Folbigg?
A. That's right.

Q. Dated 23 September 2022?
A. Yes.

Q. Doctor, before we step through specific aspects of your report and your evidence, I'd like to step through your overall conclusions and so I'll put a series of propositions to you for your agreement or clarification. First, is it correct that it's your view that the features of Patrick's initial presentation and his clinical course are inconsistent with the hypoxic ischaemic brain injury on 18 October 1990?

A. I think that that's the less likely proposition and, as I've stated in my previous reports, I think it's more likely that there was another - he was suffering from another neurological condition. Would you like me to elaborate why?

Q. I'll step through the why in a moment, I just wanted to get this at a high level to begin with. You were going on to say your primary view is that the most plausible explanation for the entirety of Patrick's medical course and death is an unidentified neurogenetic disorder? Is that right?
A. That's right. That's right. I think it more likely than not that he has an as yet uncharacterised epileptic encephalopathy that resulted in progressive neurological dysfunction and in his death.

Q. Do you exclude the possibility that Patrick sustained a hypoxic ischaemic brain injury on 18 October 1990?
A. I don't exclude that. It's possible that he did. I don't think that his course though was consistent with that, that one would expect with a child who'd had a discrete single hypoxic ischaemic encephalopathy and developed persisting neurological disease as the result of that. So it's possible that part of his clinical scenario related to a hypoxic ischaemic encephalopathy sustained on that date, but I don't think that that's the only thing that was going on, his course was inconsistent with that.

Q. If you were to assume that there was a hypoxic ischaemic injury on 18 October, is it possible that was caused by a seizure?
A. It is possible that hypoxic ischaemic brain injuries sustained on the date of 18 October was in the context of the seizure, yes.

JUDICIAL OFFICER

Q. That's assuming there was such an injury.
A. Yes.

ROY

Q. In that case, that is assuming that there was hypoxic injury and it was caused by a seizure, is it correct that his progression would still be atypical for a hypoxic event?
A. That is correct, yes.

Q. Finally, coming to Patrick's death, is it possible to exclude Sudden Unexplained Death in Epilepsy, also known as SUDEP, or other epilepsy-related events as the cause of Patrick's death on 13 February 1991?
A. I think it's more likely than not, that his death on 13 February 1991 was related to a seizure, which would then, you know, would be consistent with SUDEP, or Sudden Unexpected Death in Epilepsy. He had at that time an uncontrolled seizure disorder, was refractory to medication, and it's more likely than not that that's what caused his death on that date.

Q. I'd like to step you through now, chronologically if possible, some of the key

features of Patrick's medical history. First, prior to his ALTE, you highlight a few matters of note from his early life. There were reports of torticollis from 2 months of age. Is that right?
A. That's right.

Q. And reports of back arching and irritability at 5 months of age, which at that time, were described as, "always does this".
A. That's correct.

Q. Is it correct that these are often seen in well babies?
A. It's - it's certainly true to say that otherwise well and normal babies can have torticollis. Otherwise well and normal babies wouldn't often have persistent back arching. That would be a bit unusual. The reason why I drew attention to them in this instance was that in isolation, you might not read too much into them, but in a child presenting with an undiagnosed or atypical neurological condition, torticollis and persistent back arching could be suggestive of specific diagnoses.

Q. So, to be clear, they're suspicious for, but not diagnostic of a potential underlying neurological disorder.
A. Potentially, yes. If they were real - it was really hard to note from the notes how significant or how persistent the back arching was. It was sort of mentioned in passing. Same for the torticollis; it wasn't really clear how much of a problem it was clinically at the time.

Q. Apart from those observations, do you consider the records are otherwise consistent with a well baby?
A. Yes. I mean, the records are, up until 18 October, are relatively brief. He'd actually undergone more investigations than most because of the family history of what was felt to be SIDS. So, he had actually probably had more monitoring and more observation up until that time than a baby usually would. But everything else in the record up until that point was unremarkable.

Q. If Patrick had been experiencing seizures prior to 18 October, would you expect this would have been noticed by his parents?
A. Sometimes when babies have seizures, they can be relatively subtle, or they can be a bit atypical, and it might be that parents don't know - don't recognise them as such. But usually, they would recognise that the babies were having unusual events, even if they didn't recognise them to be seizures. That wasn't the case in this instance. Neither parent had remarked on any atypical, unusual events. So, I think it unlikely that he was experiencing any seizures before 18 October.

Q. Do you consider 18 October likely to have been his first seizure?
A. I do. I think that the - the episode on 18 October was probably his first seizure, yeah.

Q. Coming to that event, he was found at home, floppy, abnormally responsive and making minimal respiratory effort. Is that right?
A. That's right.

Q. But he was noted to be breathing?
A. Yes.

Q. Dr Ryan, this may not have been in the material that was supplied to you, but I'd like to describe Mr Folbigg's response to Patrick, and ask you to consider it. He described observing Patrick to be breathing, and then says he put his mouth over Patrick's mouth and nose to deliver breaths. From that description, would it be appropriate to describe Patrick as having been resuscitated by Craig Folbigg?
A. I guess so. I mean, it would depend on, you know, how many breaths he gave him and how forceful they were and things like that. It's hard to know. But, yes. But clearly, his respiratory effort was impaired, to the extent that his father needed to provide some sort of support. So, yes, I suppose you could call that resuscitation.

Q. Just to be clear, in case it wasn't clear, I said that he described Patrick as "breathing".
A. Right. Well, I mean - sometimes - if someone's had a seizure or any other sort of neurological event, sometimes they might be breathing but, you know, more shallowly than usual, less effectively than usual. It was clear from the notes that, contemporaneously that the baby was a bit bluish around the lips, I think, at some point or cyanosed, so his respiratory effort, I think, was not normal when his parents found him. Clearly that was the case. They were very concerned about it. Hence, I guess, his dad feeling that he needed to resuscitate him or provide him with some sort of support, and that's an appropriate thing to do, even if someone's breathing. If they're breathing shallowly or ineffectively, that would be a reasonable thing to provide to the baby.

ROY: If we could bring up Exhibit 2-AL, red page 6181.

EXHIBIT 2-AL SHOWN TO WITNESS

Q. Do you recognise these as the notes of the attending paediatrician at Patrick's admission?
A. Yes, I do.

Q. There's an indication in the middle of the page, "seen by mum at 3am because heard him coughing. At 4.30, mum heard him gasping. Was blue around the lips." You've just mentioned that. What does that indicate?
A. Well, that - it would indicate that he's - is either not breathing, although it sounds like he was breathing shallowly. I think the next step - the next line says, "minimal respiratory effort", so he's breathing, but he's making some respiratory effort, but it's not normal. And as a result of that he's got some - what we would call, you know, perioral cyanosis, which is just a clever way of saying that he was blue around the lips, really. So his respiratory effort was abnormal.

Q. Does that indicate that he was, at that stage, likely hypoxic?
A. He could well have been hypoxic at that point. The diagnosis of hypoxia is

something which is usually contingent on measurement of the blood oxygen level, but if someone is clearly cyanotic that - they may well be. They're more probably more likely than not to have been somewhat hypoxic at that stage.

Q. The EMTs arrived about 20 minutes later and administered oxygen, and took Patrick to the hospital. Over the page, so we're still in Exhibit 2-AL, now at red page 6182. At the top of the page there is a report that "Initially T 35". What do you take that to be?
A. That is temperature was 35, measured rectally – per rectum.

Q. Is that hypothermic?
A. It is, so his body temperature was abnormally low.

Q. Is that indicative of anything in particular?
A. Well, that he was – he was unwell. Quite unwell, but not specifically why.

Q. He was also peripherally cyanosed?
A. Yeah, so he wasn't – he was obviously fairly unwell. So his PR, his pulse rate, is 160, which is elevated. His respiratory rate, RR of 60 is elevated. He's clearly quite unwell at that point. They describe him as responding only to painful stimuli and that his pupils were dilated. But it's interesting that his oximetry – the blood oxygen measurement, oximetry – subcutaneously via an oximetry meter, was 88% in room air. If he was critically unwell, one would have expected that he wouldn't have been in room air, they would have been administering oxygen at that point. And I guess – it appears that once they picked up on that low blood oxygen they administered oxygen via a Hudson mask. So he was – he was quite unwell at that point. But I guess, kind of not critically so, in that they didn't feel the need to immediately administer oxygen or to provide active resuscitation.

Q. And so if the EMTs had been administering oxygen en route to the hospital, the inference from having taking a blood oxygen reading on room air is that at some point they stopped doing that?
A. That's right, yes.

Q. And you consider that indicative of the fact that he had recovered somewhat if they felt safe enough to do that?
A. That's exactly right, yes.

Q. You've also noted the absence of a blood gas reading. Is that right?
A. Yes. If a child's critically unwell, they're not breathing effectively or they need active resuscitation, then usually you would provide oxygen. You might feel the need to provide cardiopulmonary resuscitation or bag and mask ventilation. If the child is in extremis you would intubate them and ventilate them, and you would regularly obtain a blood gas on presentation, which requires an arterial blood stab. But you do that to ascertain how unwell the child was, and you would get a significant amount of information from that measurement. But it wasn't done in this case, and the fact that it wasn't done suggests that the baby wasn't felt to be critically unwell.

Q. So you actually take something from the absence of the blood gas?
A. I do. I do. So it's hard to imagine that you would have a baby who's critically unwell, who didn't need more resuscitation than was required in this instance, and didn't require a blood gas measurement. Or wasn't felt to require a blood gas measurement.

Q. You've explained some of the reasons why the 88% blood oxygen reading might have been unreliable, and you did that as well at the last Inquiry. Would that include the hypothermia?
A. Yes. So if a baby is hypothermic, or if they're what we call peripherally shut down, that they're not - the circulation to the extremities is not ideal; then a blood oxygen measurement recorded from the extremities might not be reliable. And so if you saw blood oxygen of 88% you might think that it was a false reading; it's hard to know. Again, though, if you're really concerned about that, then the more definitive measure would be measurement of an arterial blood gas.

Q. Assuming that the reading of 88% was verified by a blood gas, would that in any way change your opinion?
A. Well, it would show that the baby, Patrick, was at that point somewhat probably hypoxic. It would have been also useful to have that information because one of the other parameters that are measured on a blood gas routinely are acidosis of the - metabolic acidosis, or respiratory acidosis; the carbon dioxide level in the blood, which is a marker of the efficiency of gas exchange, but also the lactate level in the blood, which is a measure of metabolic stress. So a blood gas would have been really informative at this point in time.

Q. But specifically as to 88% blood oxygen, if that was confirmed would that change your view as to whether this was likely a hypoxic insult?
A. Well - well, firstly, you don't measure actually - you don't actually measure the saturation level on a blood gas; you measure the partial pressure of oxygen. So it's a slightly different measure that you would obtain from a blood gas measurement. But no, it wouldn't significantly change my mind at this point in time; I think it would reflect that when the baby presented to the hospital the blood oxygen, or the - if you identify the degree of low oxygenation in the blood, it just reflects the blood - the effectiveness of oxygen exchange at that point in time. So I don't think it would change my opinion at all, no.

Q. Patrick was also described on the page that's on the screen - he's described as lethargic. This is on the second line at the top of the page, the second line of handwriting. First of all, what does that mean - lethargic? What's the medical meaning?
A. It would mean less responsive than usual - is probably the most - the best interpretation of that. I--

Q. Then on the next line it says--
A. So conscious--

Q. --conscious?

A. So, yes, obviously you have to be conscious, eyes would be awake but less responsive to touch and to voice than usual.

Q. Is it then inconsistent to record on the next line "responding only to painful stimuli", is that inconsistent with lethargic?
A. No, well, no, my take on that would be that the baby's eyes were open, that he appeared awake but that he was less responsive than usual and in this instance they'd - the fact that he's responsive, only took painful stimuli gives you I guess an idea of how relatively unresponsive he was. Like he was really not on board. He was - how best to say, I can imagine it myself but that he was not quite, you know, I guess diminished - well, just diminished responsiveness really, it's just a measure of that. So, you know, if you tickle a foot and the baby withdraws, then you would expect that they were in a better condition than if you really had to give them painful stimuli to the extremities to get them to withdraw. So, you're trying to work out how - I guess how much you have to bother the baby to get them to respond in an appropriate way and responding only to painful stimuli is really reflective of the baby really not being very well.

Q. I take it that would not be a coma?
A. Would not be?

Q. A coma?
A. A coma, no, no. So with a coma the baby's unconscious, the eyes would be closed and the baby would be unresponsive.

Q. Subsequently still on that page, it notes, "After about 15 minutes became much more alert and pink in air"?
A. Yes.

Q. What does that indicate?
A. That he recovered pretty quickly really. So, within 15 minutes of what was clearly an event where he was at least moderately unwell on presentation, then he sort of bounced back reasonably quickly, that it sounds that within 15 minutes he was looking reasonably well. Although there was this arching of the back which is not a normal pattern of movement for a small baby, and, as we've already mentioned, it says he always does this, which is, you know, it is a bit unusual and I think somewhat concerning in that context. Babies don't normally back arch.

JUDICIAL OFFICER

Q. Is there any significance in the arching of the back at this particular point of time, immediately after he's been brought into the hospital, in a not very good condition?
A. Well, I think you'd - I would be concerned at that point that the baby has something going on from a neurological point of view and a baby who's been brought in who's really lethargic, he's not responding - he's not very responsive, only really sort of to maximal sort of sensory input, and then he's behaving, his pattern of movements is unusual or abnormal, I would be

concerned that the baby had a neurological problem of some sort.

ROY: We can take that down.

Q. Can you tell us, Doctor, in your own words now, what were the features of Patrick's initial presentation that you considered inconsistent with what you would expect following a hypoxic insult that sufficient to cause brain injury?
A. Well, usually if a baby's had a severe brain injury related to breathing problems, what we call a hypoxic ischaemic brain injury, first, for it to be severe enough to cause the subsequent problems that Patrick experienced, I would've expected that he wouldn't have recovered as quickly as he did from that insult. The fact that within 15 minutes he was more responsive and he was behaving more normally, would be inconsistent with a severe hypoxic brain injury. Also, things like his breathing pattern and his heart rate returned to normal. Usually if a baby's had a severe hypoxic ischaemic brain injury, they'll need significant resuscitation, often ventilation, they'll need oxygen supplied on an ongoing basis. Then what we start to see is we'll see things like abnormal blood tests, so a level of low blood oxygen sufficient to cause a significant brain injury, what usually causes injury to other end organs as well. The liver, the kidneys and the heart were usually both signs of having had a hypoxic injury as well and that would be reflected on by chemistry but also on things like abnormally low blood pressures and things like that. So, the blood tests and things that were done initially, which were limited, were relatively benign and it appeared that the baby picked up pretty quickly, to the extent that it was possible to transfer him to a normal ward, he wasn't sent to intensive care and he didn't require significant resuscitation at that point. All of which really argues against an acute severe hypoxic ischaemic brain injury at that time. It just - it doesn't fall together, just it's not how these things present and progress.

Q. You note he also did not require inotropic medications, what does that mean?
A. Inotropic medications are medications to increase the blood pressure. So if the heart muscle has been subjected to a hypoxic ischaemic injury, if there's been a problem with blood supply to the heart muscle, the heart muscle doesn't contract well and so patients will usually have difficulty in maintaining the blood pressure, and so they'll usually require medication so we call inotropes things like adrenaline or noradrenaline or dopamine to increase that blood pressure, and after a significant hypoxic ischaemic brain injury you might well expect that the patient would need inotropes for several days.

Q. You also noted he had a normal EEG, is that significant?
A. So later in the day the baby underwent an EEG, which was normal, and I think it was an awake and sleep EEG, it was, you know, a good study. That would really - again, if you'd had a significant hypoxic ischaemic brain injury it would be very unusual to expect, for the baby to have a normal EEG at the time. His head ultrasound was also normal that day, I would read less into that because ultrasonography of the head is relatively insensitive as a measure of these sorts of things. The other thing is the other measures that were taken at the time were his blood sugar was normal, generally any significant stress to the body will result in elevation of the blood sugar, which was not identified

when Patrick was brought to the hospital. He did have some sugar in his urine, what we call glycosuria which would reflect - which could reflect a brief hypoxic ischaemic insult or a significant stress or a seizure. But whatever it was, it was not sufficient to result in persistent elevation of the blood sugar for any period of time, because it was normal when he got to the emergency department. So, he might have had a stressful event. It sounds like he did. But it wasn't sufficient to result in persisting elevation of the blood sugar. All of which kind of argues against a severe hypoxic ischaemic brain injury. It's just not a presentation that you would - well, it's not consistent with that sort of injury.

Q. Would sugar in the urine, together with an absence of sugar in the blood, indicate that blood sugar levels had risen in response to a stressful event, but been burned up by the brain during a seizure?
A. It's - well, if you have a stressful event, your body releases adrenaline, and that's why the blood sugar goes up as part of that pathway, and it's true that the metabolic demands of the brain are increased during a seizure. But if it was a significant seizure of any duration - yeah, more often than not, the blood sugar would still have been elevated at the time of presentation to the emergency department. But the fact that the blood sugar was - sorry, that the sugar was present in the urine, the glycosuria was present, suggests that there was a stressful event and, I think, clearly he was really quite unwell for a period of time. But the fact that the blood sugar was normal on presentation, I don't think it's been burnt up by the brain, so much as that he just had a transient elevation in result to a transient stress.

Q. You say in your report, including consistently with what you've just said here, this is Exhibit 12, tab 2, red page 14, that “had he been subjected to a severe hypoxic-ischaemic insult at home, …it would be expected that he would be obtunded (persistently unresponsive), that he would not feed well, and that he would have had evidence of hypoxic injury to the kidneys, liver, heart and bone marrow.” I think you've said in your evidence this afternoon that there were limited tests in relation to those other organ functions. Is that right?
A. Yep. The tests that were done after he presented, blood tests were all normal. They showed really no evidence of liver injury, kidney injury or bone marrow injury, which would - you would normally expect to see in someone - in a baby who's had a significant hypoxic insult. But yes, also, there was a, I think, a follow up; a couple of follow up urine dipsticks which were done in the following days and they didn't show blood or protein which you would expect if there'd been a hypoxic ischaemic insult to the kidneys. Also, he continued to produce urine and had a normal number of wet nappies and things, which is not always the case. If the baby's had a significant hypoxic ischaemic insult to the kidneys, they usually would - their urine output would be decreased. But also, yes, you would expect him to be, I said obtunded, which is very, very sleepy. It's sort of halfway between, I guess, lethargic and comatose; extremely - extremely - difficult to rouse. And you wouldn't expect him to feed normally. You know, babies who've had that sort of insult don't feed normally for, you know, days to weeks, if ever, and so the fact that he was felt to be feeding reasonably well, at least in the first 24 hours after this episode, again sort of argues against him having had an acute severe hypoxic ischaemic

insult.

ROY: Can we please have Exhibit 2-AL, red page 6195 on the screen? Can we zoom in to the top half of the results?

Q. This is a series of lab results from his initial presentation in his first few days of admission. Drawing your attention to the lactate level, which is, I think, the third result from the bottom. There's no result under the first two columns, and then under the third column is "1.6". Do you see that?
A. I do.

Q. That's referrable, if you follow the column up to "FLOX". Can you help us with what FLOX means?
A. I think it's fluoride oxalate. It's a sort of tube that the blood - that lactate levels are taken in.

Q. Is there anything informative about a blood lactate level two days after the initial presentation?
A. Look, if - if - if there'd been a dreadfully severe hypoxic ischaemic brain injury, the blood lactate might be a little bit elevated after two days, but really that's due to significance in the context of understanding the acute event that Patrick experienced. However, if he'd had - there are a number of genetic and neurometabolic disorders that result in persistent lactate elevation, such that what this is, which is essentially a random lactate taken in between an acute episode, might be really elevated, and that would draw attention to the likelihood that he had one of those metabolic disorders. I would call a lactate level of 1.6 in a baby essentially normal, maybe a tiny bit elevated. But it just means that I don't think it's helpful in understanding what caused the initial event that caused his presentation on 18 October, but it does tell us that in between episodes, or between seizures, he had an essentially normal blood lactate, and that rules out a number of different conditions.

Q. Metabolic disorders?
A. Metabolic disorders, yes.

Q. So, you've already described his initial recovery period, feeding well. Later that day, the medical notes describe, "pallor, sweatiness, persistent crying and irritability", and overnight being "unsettled and irritable. Given a sedative." And then the following day, 19 October, "experiencing multiple multi-focal seizures, requiring treatment with several anti-convulsants." You've already told us you consider his presentation atypical of hypoxic brain injury, and what you would ordinarily expect in that regard. There's a 1989 academic article that was discussed in your oral evidence to the 2019 Inquiry, between you and Professor Fahey, concerning the presentation of near miss SIDS cases; and I'll come to that in a moment. But putting the case as described in that one paper to one side; are you otherwise aware of cases in which an infant or child suffers a hypoxic brain injury and initially recovers well and appears normal, and then declines in the 12 to 48 hour period afterwards?
A. Sometimes when babies - very small babies at birth have had a hypoxic ischaemic brain injury they can look relatively normal before a period of

apparent deterioration related to brain swelling and things like that. But it comes down to what the relatively normal looks like, because if you examine them well, you can identify neurological abnormalities. But it's not a - it's not a common picture, really. Certainly not in babies who've had a severe brain injury. So when we talk about newborn babies, we say that, you know, if they've had a very mild sort of insult then they might be a bit hyperalert and jittery for a day or two and then come right. But they won't be normal, and then deteriorate significantly. I'm not sure if that is clear.

Q. It is, Professor, thank you. Doctor. Coming now to the article I mentioned, it's just been added to the tender bundle. It's Exhibit 15-267, if we could have it on screen; red page 3807. The article is from the "Archives of Disease in Childhood", 1989, edition 64. The title is "Hypoxic-ischaemic encephalopathy after near miss sudden infant death syndrome", and the lead author is Constantinou.
A. That's her.

Q. This article describes 14 infants between 1982 and 1985, between 3 and 26 weeks of age, who presented with severe hypoxic episodes as a result of what was described then as near miss sudden infant death syndrome. You're familiar with this article?
A. I am, yes.

Q. Is it fair to say that this was relied on by Dr Fahey in the 2019 Inquiry as recording cases in which a presumed hypoxic brain injury in an infant was followed by a period of apparent recovery, before significant decline?
A. That's true, yes. That's fair.

Q. If I could take you to a few aspects of that article. Beginning in the second column on the first page, the second paragraph under "Patients and methods" - "All subjects fulfilled the accepted criteria for the diagnosis of near miss SIDS. They had been found pale, cyanosed, limp or not breathing, and had required vigorous stimulation or cardiopulmonary resuscitation because death was thought to be imminent." Would Patrick fall into that category?
A. Well, it's a bit hard to say. I wasn't aware before today that his father had felt the need to give him what was essentially like mouth to mouth. And I suppose you could call that pulmonary resuscitation. I can't really read into the notes, the contemporaneous notes, about whether his death was thought to be imminent. Obviously he was really quite unwell; he was cyanosed and limp, and breathing poorly when he was first found.

JUDICIAL OFFICER: Did the ambulance officers give him any resuscitation?

ROY: No. There's no record of that. They administered oxygen.

Q. Then further down the column, a new paragraph: "Evidence of a hypoxic insult during admission consisted of metabolic acidosis". There was no evidence of that here. Is that right?
A. Well, it would have been - were it present, it would have been identified on the blood gas. So that would have been diagnosed on the blood gas that was

not undertaken.

Q. "Cardiovascular instability", specifically hypotension?
A. Which was - was not present.

Q. "Acute renal failure"?
A. Not present.

Q. "Ischaemic colitis"?
A. Not present.

Q. Then "Acute neurological dysfunction", which they define as "deep coma or convulsions". Was that present on admission?
A. It was not, no.

Q. I just note while we're there that that continues to define deep coma. It says, "Deep coma was diagnosed when the infant did not open his eyes, …made no sound, and made no purposeful response to localise or resist painful stimuli".
A. So our understanding is that the baby was lethargic, and responsive only to painful stimuli. …(not transcribable)… by which I would take that he was conscious. Less responsive than normal, but was responsive to painful stimuli.

Q. It sounds as though Patrick would not have qualified - under this article's definitions - as a hypoxic presentation?
A. Not a severe hypoxic-ischaemic near miss SIDS, no.

Q. Of the 14 infants in this study, is it correct that half lived and half died within about 60 hours of first presentation?
A. That's correct.

Q. On the second page of the article, which is red page 3808, the right-hand column, the second full paragraph begins: "After resuscitation at home all seven who lived had established spontaneous respirations by the time that they arrived in the hospital emergency department. Resuscitation had consisted of vigorous stimulation, mouth to mouth resuscitation or cardiac massage." Again, that's conceivably consistent with what Mr Folbigg described. Is that right?
A. That's true.

Q. The right-hand column continues down towards the bottom of that same paragraph. "In two infants (cases 2 and 4) acute neurological dysfunction was the only evidence of an hypoxic injury." Do you see that?
A. Where's that?

Q. Down the column on the right-hand side of the page. It is the third from the bottom, point 7. It's the third from the bottom sentence in that paragraph, above "Acute neurological course".
A. I see it, yes. Yes. Cases two and four, yes.

Q. Pausing there, the inference there is that for the purposes of this study the earlier definition we looked at of deep coma and convulsions was considered enough to diagnose hypoxic injury. Do you agree with that?
A. Yes.

Q. In your view is that enough to diagnose hypoxic injury?
A. No, it's not, because just deep coma in and of itself would not have been sufficient to - it's a clinical - it's a description and the child is comatose, they're deeply unconscious, it doesn't - you can't extrapolate from that as to the cause, whether it was hypoxic ischaemic brain injury, it could be something else, it could be what we call a postical state where you've had a really big seizure but you will often be comatose after that. It could relate to something like an intoxication, you know, there's lots of different things that can cause someone to go into a deep coma and hypoxic ischaemic brain injury is only one of them.

Q. So on one view would it be possible that these two infants were inappropriately included in the study of hypoxic brain injury?
A. Well, I think it says in that first - on page one in the bottom right paragraph that we just had a look at that all of the children had that on presentation, and going on to that, if you look at this paper, if you look at the next page down from the one that we've got upon the screen there, page 705 which is the next page down, they give a bit more further information and they say that out of those two infants, and so that's cases two and four who just had acute neurological dysfunction, it does appear that case two had - if you just zoom in on that table at the top, case two had cardiovascular instability and in fact case four developed an ischaemic colitis. So, they had evidence of - it's not really an isolated acute neurological dysfunction, one had evidence of cardiac involvement and one had evidence of an ischaemic colitis. So, I think that it's a little bit misleading that, where they say that it's now only evidence of a hypoxic injury.

Q. Continuing back on page 3808, at the bottom of the page in the second column and over onto page 3809:

> "Five of the infants … showed a biphasic evolution of neurologic dysfunction characterised by an initial period of near normality and subsequent neurological deterioration. These five infants were conscious and rousable within one hour of resuscitation. They were awake, alert and active, or irritable or lethargic with only minor abnormalities of tone and deep tendon reflexes... This striking interval of near normality continued until the onset of seizures from 2-51 hours … after the event."

To an extent that's consistent with Patrick's course?
A. Well, in that he was initially really quite unwell, and then he had a period of what seemed like near normality, and then a neurological deterioration, yes.

Q. This article goes on to describe that every one of the seven infants who lived went on to fall into a deep coma?
A. Yes, that's right, and that's shown in table 2 which is just above what you've

got on the screen there.

Q. That's not consistent with Patrick's presentation?
A. No, and for that reason, Michael Fahey put a lot of store in this paper when we discussed this at the last hearing, but to me the course of the infants described in this paper, which is, I have to say, really unusual and most atypical, is not consistent with what Patrick had and I think that's why I had discounted it when I prepared my original reports. He wasn't terribly unwell in extremis at the time of presentation, with an apparently normal sort of period and then going into a deep coma, that just wasn't what he did at all. His course was quite different to that.

Q. From what you've just said, this article is generally reporting upon an atypical presentation following presumed hypoxic brain injury?
A. Yes, and so the paper was published in 1989, I think as the authors felt that they had identified a sort of an unusual clinical phenomenon, you know, an unusual temporal pattern of response to a near miss SIDS event, and so they published this paper and, you know, it's - I guess it's of some interest but it's probably worth noting that that was 1989, which is 34 years ago I think, and there's been - yeah, 34 years, and - I'm not aware of any reports since that time of anything like this sort of clinical scenario. So I don't think that with the passage of time people have recognised this as a clinical phenomenon, something that does happen repeatedly. I think it, you know, and for that reason I think it can be discounted because, you know, it's really just an isolated small series published 34 years ago.

Q. Have you checked to see if there's been any other publications more recently than this reporting similar phenomenon?
A. I have. So, you know, I have gone back and looked to see if any other centres have reported similar sorts of things at any point in time. It wasn't a pattern of response to hypoxic ischaemic encephalopathy that I ever saw in my own career, and I haven't been able to find any reports from other centres where that sort of pattern has been recognised subsequently.

Q. Another feature of this paper, we don't need to bring it back up, but in all of the infants who lived, some degree of cortical blindness was noted?
A. That's right, yes.

Q. I understand your evidence that's also atypical of hypoxic injury?
A. It can be part of - if you've had a very significant severe global brain injury, which is with all parts of the brain being effected by severe hypoxic ischaemia, which would be consistent with what's described in these children that they were all extremely unwell at presentation and they all went through a period of coma, then cortical blindness, which reflects injury to the parts of the brain that subserve vision, would be pretty common. But that's not in isolation. It's in association with other neurological deficits, reflecting very severe neurological injuries, so; quadriparesis; cortical blindness; severe development delay, and refractory seizures would usually, kind of, travel together as part of what you see in someone who's had a very, very severe global brain injury.

Q. Which is not what you've seen in Patrick?
A. No. So, with Patrick, it did appear that he had brain injury that was affecting his vision. From the notes, his medical history, after 18 October but before his death, it appeared that he had what seemed to be fluctuating visual loss. At times, he was felt to have quite significant visual loss. At other times, it was felt to be less severe. But he certainly didn't have this very severe pattern of injury with spastic quadriparesis and severe - severe - developmental delay that was identified in the seven children in this series.

Q. Coming back to Patrick's ALTE, assuming Patrick's presentation was occasioned by a seizure on 18 October 1990, would it be different to the rest of the seizures that Patrick was later observed to have experienced?
A. Yeah, so the - Patrick had a number of episodes of seizures and he had some episodes where it wasn't clear if they were actually seizures or not. There was some question about some of the episodes that he had, but the very first one, if that first one was a seizure, it was associated with significant respiratory impairment. He was blue. He was not breathing effectively when he was first identified, to the extent, obviously, where his dad felt that he needed to give him mouth-to-mouth resuscitation. He doesn't seem to have had seizures subsequently that resulted in that degree of respiratory impairment.

So, the seizures that he was described as having, subsequently were what we call "clonic", so jerking of the extremities, effecting different parts of his body at different times, which we would describe - a neurologist would describe as multi-focal. And some of them it sounds like may have been - none of them were particularly prolonged and none of them were associated with significant respiratory impairment, so if it - if that first episode was a seizure, it wasn't similar to the ones that he had subsequently.

But he also had really unusual episodes, I should say, which - where he had upward eye deviation. His eyes were sort of deviated upwards for a period of time and he was very stiff, and at the time, it was suggested that they look like oculogyric crises, which is a - is a different phenomenon; seizure-like phenomenon, but not an epileptic phenomenon, and oculogyric crises are very unusual in small babies. And it wasn't really clear from the contemporaneous notes whether those episodes were seizures or not. If they were seizures, then that - it's a very unusual form of seizure for a baby to have.

Q. Are oculogyric crises something that's ordinarily seen in babies or adults following hypoxic brain injury?
A. No. Oculogyric crises would not be something you'd see after a hypoxic ischaemic brain injury normally. Usually they generate - they're generated by the basal ganglia, deep centres in the brain that are important for the control of movement. They sometimes happen in response to medications that affect the basal ganglia, or the - so the - sorts of medicines you give to Parkinson's and things like that, or you can also see them in certain metabolic disorders.

Q. Assuming that the initial presentation was a seizure, does the difference between that initial presentation and the subsequent seizures cause you to

doubt whether or not this was an undiagnosed neurogenetic disorder.
A. No, I'd say not. So, children with neurogenetic disorders, and I guess with Patrick, I sort of felt most likely that he had an epileptic disorder of some sort, what we would call an epileptic encephalopathy, and because it happened in early childhood; infantile epileptic encephalopathy. Very often you can have more than one seizure type. The seizures can look different. We call that semiology, it's what you do during a seizure, so you can have seizures with varying semiology. Sometimes with these conditions, seizures vary with other body parameters. So, for example, very often the first seizure will occur or other seizures might occur in the context of a temperature, and if you have a body temperature or you're otherwise unwell, then you might be more likely to have a very long or very severe seizure. It seemed that the - if that first episode was a seizure, it was a fairly nasty, significant one, but he didn't go on to have that sort of seizure again. There was some notes at the time that he was a bit unwell at the time with a mild respiratory illness and that would be consistent. Often babies with a - who present with an infantile epileptic encephalopathy will first present at the time of some other intercurrent illness, often one associated with fever.

Q. Before we leave Patrick's ALTE, in your experience, is it possible to experience hypoxia, sufficient to occasion a hypoxic ischaemic injury, without requiring resuscitation, to survive that event and not require resuscitation?
A. It's - it's - it would be possible to have a significant hypoxic ischaemic event and to be able to recover without resuscitation on - at the time. In terms that - I mean, you know, for example, if you think about a baby who's rolled over and perhaps obstructed their airway in bed, and then managed to extract themselves from that and rolled, perhaps, back off onto their back or whatever on to their side and stop that obstruction, and so recover in that respect. But had the hypoxic ischaemic insult been significant enough to cause significant brain injury, then the baby, I would have felt, would require resuscitation, would be sick enough that they would need more help than Patrick had received - than Patrick required.

Q. Coming now to the details of Patrick's death and his autopsy. I understand that SUDEP, Sudden Unexplained Death in Epilepsy, is by definition, only occurs over the age of 12 months. Is there any reason for that age cut off, in terms of the mechanisms of death?
A. I think - the concept of SUDEP is kind of an artificial construct. It's a description of - I guess you could call it a syndrome. But basically, you know, it's - SUDEP is just - it is what it is. It's people who die suddenly and unexpectedly in the context of known epilepsy. So from that point of view you can have SUDEP in the first year of life, just as much as you can have it subsequent to that. I think this definition of it occurring only over the age of two is kind of an academic one; is for classification. Certainly babies with early infantile encephalopathies have a very - unfortunately, those conditions are associated with a significant mortality, at least kind of 15% in the first year of life. And very often those deaths occur either in the context of an epileptic seizure, or a respiratory illness, or a pneumonia or aspiration, or something like that; which might often be occasioned by an epileptic seizure. So I don't think that unexpected death related to an epileptic seizure - it can happen at any

time. It doesn't - it's not limited to those more than 12 months of age.

Q. The previous Inquiry heard numbers of the rates of death, SUDEP in infancy, ranging from one in 45,000 to one in 10,000. Do I understand your experience to be that in infantile epileptic encephalopathy, the rate is as high as 15% in the first year of life?
A. Yes. So some colleagues and I actually published a paper a couple of years ago, which found that in early infantile encephalopathies mortality, I think, was 16% in the first year of life. And in some conditions, in fact, in some discrete genetic, well-described, recognised genetic epilepsies, it's much higher than that even. Unfortunately.

Q. Coming to the issue of genetics. In your view, has the whole exome sequencing that occurred in this case excluded the possibility of a genetic cause for Patrick's disorder?
A. If we look at whole genome sequencing, or whole exome sequencing, and we apply that to babies with undiagnosed severe progressive neurological disorders, we consistently find - and when I say we, I mean paediatric neurologists and geneticists world-wide - find a diagnosis in about 33% of cases. So about a third of cases. Which means that in two-thirds of cases we don't. And that's a lot better than it was in 1990, where these techniques weren't available at all. But it still speaks of the fact that in the majority of cases we don't find the cause, even though we can be pretty confident that it's a genetic disorder of some kind. And that just reflects limits in our understanding of the genetics of these conditions. New conditions are found all the time, which is why we often, in kids who are undiagnosed, go back and re-examine the data every three to five years, to - in the light of what's been described in the interim. But the fact that a definitive abnormality wasn't found on Patrick's genetic testing does not, to me, infer that it wasn't a genetic disorder.

Q. Ordinarily, assuming a child with Patrick's presentation had lived, is there an ordinary timeframe for diagnosing the precise cause of a disorder?
A. Well, if he was born today and we did whole genome sequencing, which is the best testing known to us at this point in time, and we did that today, it would be negative. And then we'd just kind of have to wait and see. And we would re-examine the data every three to five years. We'd look at clinical papers and see if anyone with a consistent pattern or gene--

Q. I'm sorry, Doctor, I think we're at cross-purposes. I didn't mean to confine you to genetic diagnosis. In terms of otherwise known disorders, how long would it ordinarily take to arrive at a diagnosis for a child who presented? Is there no such thing as ordinary?
A. No, it's hard to say. Cause it can sometimes, you know, the first test you do, gives you the answer and sometimes you do all the tests, as, you know, he could have been subjected to all the tests and we might not find an answer.

Q. I'll put more plainly what I'm really asking. Is four months a long enough time, is there enough data here, to arrive at a conclusion as to what was going for Patrick?

A. Well, I think unfortunately he died without a definitive diagnosis. And I think that - is that what you mean? I mean, I think, unfortunately, these sorts of things happen, and I've certainly had many patients where I've made a diagnosis years after they've died, because we've gone back and done further testing, or re-examined tissue or things like that. Very often we don't have sufficient time during babies' lifetimes to make a specific diagnosis.

Q. That would be the case with Patrick?
A. Yes.

Q. In terms of infants that are at particularly high risk for death in connection with epilepsy; you would include young age?
A. So babies who are particularly at high risk for sudden death in children with epilepsy would be associated with long - babies who have really long seizures; babies who have seizures associated with respiratory impairment, so apnoea or impaired breathing. Being refractory to medications. So he was tried on a number of different medications with limited success, and so that's certainly a risk factor for SUDEP. And being very young, yeah.

Q. You said seizures associated with apnoea.
A. Yeah.

Q. Does that presume that the first presentation was a seizure, that was apnoeic?
A. I'm not presuming that, but I think it's quite possible, that if that first episode was a seizure, it was probably associated with some apnoea and impaired breathing.

Q. Other than that first seizure, the other seizures were not associated with apnoea. Is that right?
A. Not as far as I can tell.

Q. Are you aware, Doctor, of any studies or guidance which suggests that swollen airways would be an expected post-mortem finding of a SUDEP death?
A. Well, I guess - it's a little bit sort of outside my area of expertise but I guess if you had obstructed airways during a seizure, that could contribute to a baby's death and that might be associated with swelling of the airways, but it's not a specific finding associated with SUDEP as far as I know.

Q. Coming to the post-mortem findings, can we please have Exhibit 2-H, red page 3559 and zoom into the text in the centre of the page? You're familiar with this report?
A. I am, yes.

Q. This is the report of the brain biopsy, Patrick's brain biopsy?
A. Yes. It's not really a biopsy, it's sort of, they examine the whole brain, pathologically.

Q. About the middle of that first paragraph it says, "In the deeper parts of the

cerebrum and in the cerebellar and brain stem nuclei there are neurones showing simple atrophy. They could have resulted from this baby's epileptic seizures", do you agree with that?
A. Yes.

Q. Continues down the bottom, "I believe that the small amount of linear cortical calcification in the occipital region is just part of the laminar cortical necrosis", and then continues in connection with that, "far more likely to be the result of the episode of cardio-respiratory arrest this baby suffered at about 5 months of age"?
A. Yes.

Q. I take it - I mean this is, you would agree this is a mistake, there was no cardiorespiratory arrest?
A. I guess so. I think any pathologist would agree that the quality of their interpretation of what's in front of them is often limited if they're not given the right information. As a sort of garbage in, garbage out sort of situation. So, yes, so I don't believe that on the evidence that we have that Patrick had a cardiorespiratory arrest and so the pathologist seems to have been misinformed.

Q. Is this otherwise, would you agree, this is otherwise consistent with a brain injury of some kind?
A. Yes. There's definitely brain injury. What I guess I would comment on is that most of the changes, old infarcts or gliosis which is like scarring effectively in the occipital lobes at the back of the brain, they're important for vision, but also some in the left frontal and I think both parietal lobes as well, and it was reported that they were most consistent with strokes. So strokes are areas of brain injury related to potentially hypoxic ischaemic insult or sometimes to other sorts of insults as well. But there wasn't, what wasn't identified was pathology in the basal ganglia and the thalami which are deep nuclei in the brain which are usually pretty much always affected in acute severe hypoxic ischaemic brain injury and that was not identified on the imaging contemporaneously, on the CT scans that were done contemporaneously, or on the post-mortem, which would argue against an acute severe hypoxic brain injury being the cause of Patrick's subsequent course.

Q. Can I take you to Professor Fleming's report in connection with that, Exhibit 11, tab 2 at red page 30, if we could have that on the screen? Reading from just above line 680, if we could zoom in. I'll give you a moment to read that, I won't read it out, beginning from, "The lack of any significant identified abnormality"?
A. Yes. I agree with that. The fact that there didn't seem to be any involvement of the basal ganglia or the thalami on those CT scans that were done contemporaneously, or at the post-mortem really argues against it being a hypoxic ischaemic brain injury as such. And the infarction - in the right occipital region, if they could go along with the hypoxic ischaemic injury, but you wouldn't expect to see those without involvement of the basal ganglia and the thalami. Just it's a really inconsistent brain injury. I was going to say I agree with what he says about the acute episodes of hypoxic ischaemic injury

of severe but brief, you know, they take up the basal ganglia and thalami essentially, so it's just a really atypical picture and the picture that we've seen in Patrick would be very atypical of an acute severe brain injury like that.

Q. Can I ask you to read the balance of the paragraph and indicate whether there are any matters you disagree with?
A. Okay. Yes, no, I agree with that.

Q. Can I take you next to Dr Cala's report which is Exhibit 13, tab 4, red page 55 which we'll put on the screen. At the top paragraph of that page Dr Cala says:

> "The brain was retained and sent for neuropathological examination which disclosed very extensive hypoxic brain damage. Brain examination showed no features of an underlying metabolic encephalopathy or any other abnormality of brain development where epilepsy may be a feature."

First of all, do you agree with what he said there?
A. Well - no, I do not.

Q. Can you explain what you disagree with?
A. Well, there are hundreds of different metabolic encephalopathies, so different metabolic conditions that can affect the way that the brain functions, and they have their own patterns of associated neuroradiological changes. I'll just give you one example. There's a couple of conditions which are involved in a particular pathway. They're very small. They're genetic disorders that can cause - they can - they cause changes in the brain that look exactly like strokes, and in fact they both affect the occipital lobes preferentially. So, if you took the brain of a baby who'd succumbed to these particular conditions, they would have multiple areas in the occipital lobes that look like strokes, but they are metabolic disorders and genetic disorders that are progressive and devastating. Now, the ones that we know about were excluded by Patrick's whole genome sequencing, so they were the sorts of conditions that I - which led me to suggest in the first instance that he should have that sort of testing, because I thought, well, he could have sulfite oxidase deficiency or this other condition, molybdenum cofactor deficiency. I'll spell them if you need me to. But, you know, they can look exactly like a stroke-like episode, so I don't think it's reasonable to say that he had no features of anything that could be consistent with a metabolic encephalopathy because I don't think that the neuropathological changes are that specific.

Q. Could I take you then to your report which is Exhibit 12, tab 2, and on red page 15 you say about halfway down the page that the changes, referring to the post-mortem of the brain, "were potentially consistent with old strokes (previous hypoxic-ischaemic injury)." Can you explain that?
A. Yeah, so those sorts of changes, as that last gentleman reported, look like the sorts of things that you can see with strokes, and the most common cause of strokes is hypoxic ischaemic injury. And I probably could have worded that better because you can also have strokes associated with metabolic

disorders. All a stroke is is that there's an energy failure in the brain which results in tissue injury. Most often, that's because of a problem with blood supply and oxygen supply to the brain, but it occurs as a result of a metabolic problem where there's an issue with energy metabolism in the brain. And so, the information that we have from that post-mortem examination is - yeah, it's consistent with old strokes with areas of brain injury which are focal and localised. But I probably could have worded that better. It doesn't necessarily reflect hypoxic ischaemic injury.

Q. Are you hypothesising that Patrick had experienced strokes at some point in his life?
A. It - well, it depends on - well, when I say a stroke, I mean brain injury and scarring, loss of brain tissue associated with an insult, which - which could be - yeah, so I do think he has. I mean, it sounds like those areas that he's got a change on his - that were identified on the post-mortem are strokes. But the issue is, are they strokes related to brain injury from a hypoxic ischaemic insult, or from some other sort of injury? That's what I don't think we know for sure.

Q. What other sorts of injuries could occasion that damage, consistent with epilepsy?
A. Well, I guess - so, in the context of someone who had epilepsy, it could be that that very first episode that he had on 18 October 1990 was a seizure, between which, he had some hypoxia ischaemia and had some brain injury, but for the reasons that I've described in some detail, that seems unlikely. Other - other possible explanations are that he had an underlying genetic disorder that resulted in stroke-like episodes to - in damage to the brain. Not as a result of hypoxia ischaemia, but as a result of energy failure, and we see these metabolic strokes in a range of genetic disorders, many of which are associated with seizures. And they often, kind of, travel together. So, those two conditions I mentioned earlier, sulfite oxidase deficiency and molybdenum and cofactor deficiency, are actually metabolic conditions that effect the body's metabolism of uric acid, but they result in stroke-like episodes in the brain. And so over time, babies with those disorders develop progressive strokes. And, you know, Patrick's post-mortem findings, if you took them in isolation, would be consistent with those disorders. As I said, they were excluded on his whole genome sequencing, but that's not to say that there's not other disorders like that that we don't yet know about, we can't yet diagnose, that could cause the same problem.

Q. But have you seen it before in a case that was not confirmed to be one of the known genetic pathogenic variants?
A. I have, yes. And so, you know, unfortunately, you know, I'm old enough to have seen lots of babies where we saw this sort of atypical sort of a course. We couldn't explain the findings that we saw at the time. Often, unfortunately those babies have passed, but we've gone back a year, two years, five years later and in the light of new knowledge, have been able to make a diagnosis. And I have seen these sorts of changes on post-mortem examination and not been able to explain them contemporaneously, but examiners have been able to do that with increasing medical knowledge some

years later.

Q. If Patrick was having stroke-like episodes as a result of energy failure, which I think was your expression, would that be something that you would expect his parents or other carers to notice?
A. Well, I - you would expect that you would see something, and--

Q. What would it look like? What would you expect it to look like?
A. --what you might see would be something that resembled a seizure or a seizure-like episode. So, the oculogyric crises, you know, something like that, you might see some unusual episodes, really depending on where in the - where in the brain the energy failure is happening. So, if you think about the occipital lobes, they subserve vision, so a stroke-like episode or an episode of metabolic energy failure in that part of the brain would present with visual loss which might fluctuate a bit. So, it would kind of be consistent with what we - what was described in Patrick, which was unusual episodes with odd eye movements, and what was felt to be fluctuating vision.

Q. My last question, Doctor, in your evidence at the last Inquiry you had said - this is at transcript 605 - "Everyone agrees a brain injury was sustained at that time; 18 October." Can I ask what you meant by brain injury in that context?
A. I think it was pretty clear that something happened on 18 October that was fairly profound, and that before that date Patrick Folbigg was felt to be - was felt to be essentially normal. There are a couple of things about him that were perhaps a little unusual, but he was felt to be normal. And subsequent to that date he was not neurologically normal. He very quickly evolved a refractory seizure disorder, fluctuating visual loss and developmental delay. So there had to have been some sort of brain injury at or around 18 October. The question is exactly what that was. I don't feel that his presentation at that time, and his subsequent course were consistent with him having experienced a severe - or profound, it would have to be - profound hypoxic-ischaemic brain injury on that date, that then accounted for his subsequent seizure disorder and developmental delay. But I think that it's more likely that he had an underlying condition that first manifested on that date, that first manifested severely enough that it was recognisable that he had a problem, and more likely than not, you know, with something like a seizure that - on the early morning of 18 October caused his presentation.

Q. Is Patrick's course, including his death, consistent or inconsistent with other cases that you've had over the course of your career?
A. Unfortunately, Patrick's course is consistent with other cases that I've seen in the past, which is the - you know, you have a baby who's felt to be normal in the first few months of life. They present with - often with a severe seizure, sometimes a really prolonged seizure, or a first seizure; and then after that fairly rapidly there's a change with an evolution, development of an increasingly often refractory seizure disorder, which is associated with development of permanent - usually neurological - deficits, which is accompanied by a failure to make normal developmental gains. So evolving developmental delay. And unfortunately in such cases babies often do really

poorly. And unfortunately they often succumb to those illnesses. So it is something - it's a clinical presentation I've seen many times in my career. It's easier when you make a diagnosis, and you can understand why it's happened, and you explain that to the parents, and have some expectation of how things are going to progress. It's always harder when you're unable to make a definitive diagnosis.

NO EXAMINATION BY DR WOODS

<EXAMINATION BY MS HORVATH

Q. Doctor, I think it was referred to earlier - it was, page 45 of the forensic bundle, being the letter from Dr Kan. Do you have that available? Or can it be brought up, perhaps?
A. I'd be grateful if it could be brought up.

Q. The second sentence, which I think you referred to before; "The major changes in this extensively sectioned brain are old infarcts", et cetera. That sentence, the description of the old infarcts and gliosis; that is referring to something which is also consistent, is it not, with a hypoxic brain injury?
A. It could be, yes. So, yeah, so as I said, so strokes can happen as a result of hypoxic ischaemia, or they can occur for other reasons as well. But potentially consistent with hypoxic ischaemia.

Q. I don't want to cut out half of the sentence. Looking at the remainder of the sentence as well; that is, "the form of old laminar necrosis which, in keeping with the macroscopic finding, is most severe in the"?
A. Parieto-occipital.

Q. That description, I take it, is also consistent with a hypoxic brain injury?
A. It could be. But - potentially, yeah. But the pattern of injuries would be really unusual for an acute - as I said before - to have a hypoxic-ischaemic brain injury, a sudden one, that just affected the parieto-occipital region. And usually you'd either have - it would be global, so the whole brain affected, or if it was acute and severe then it would affect the basal ganglia and the thalami. And there's - there's not a lot of - there's no great mention made of those parts of the brain in the post-mortem; they're not really sort of focused on. So presumably they weren't felt to be abnormal.

HORVATH: Thank you, your Honour.

ROY: Your Honour, just for the benefit of the record, on screen was Exhibit 2-H, red page 3559.

JUDICIAL OFFICER: Thank you. Mr Jordan.

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

CALLAN: Once Dr Ryan has terminated the link, we have some documents to

tender.

<THE WITNESS WITHDREW

AUDIO VISUAL LINK CONCLUDED AT 3.58PM

Your Honour, we have obtained some articles that were referred to in the reports of Dr Orde and Professor Cordner, so the table of articles which has been included at the end of their reports has been updated. Can I hand up a tender copy of those updated tables, which is to replace the existing table; in respect of Dr Orde at Exhibit 13-06, commencing at red page 128 and a tender copy of an updated table of articles referred to by Professor Cordner which is to go at the end of Exhibit 13-02A at red page 1358, and complementing that, your Honour, we have some tender copy of articles to be inserted. The first is an extract from the fourth edition of Knight's Forensic Pathology which relates specifically and by way of update to the section that I took Professor Fleming to this morning as to suffocation and asphyxia. The photographs that are included in that part of the text have been blocked out but otherwise that's the complete copy of that chapter for which we are grateful. It's proposed that that would be inserted at Exhibit 15, tab 127A.

Then, your Honour, there is an article, "Petechiae of the Baby's Skin as Differentiation Symptom of Infanticide Versus SIDS", to be inserted at Exhibit 15, tab 264. An article which is "Chapter 19: Respiratory Tract & Mediastinum", from Houin and others - it's referred to in I think it's Dr Orde's report, to be inserted at Exhibit 15, tab 265. Finally, an article in relation to myocarditis, "The Dallas Criteria", also referred to in Dr Orde's report, to be inserted at Exhibit 15, tab 266. Your Honour, we also received a supplementary report from Professor Schwartz of 20 February 2023 addressing the questions that those assisting your Honour had raised with him in terms of further data from the CALM Register, if that could be received and marked Exhibit 8-08.

Finally, your Honour, I have a one-page document that we are proposing would be tendered and marked Exhibit 14-08A, which is titled "Summary of Phenergan Prescription and Toxicology Screening Results". In short, your Honour, there was an analytical toxicology report of 12 April 1999 in respect of Laura Folbigg which appears at Exhibit 14, tab 8. Those assisting the Inquiry wrote to the Ministry of Health enquiring as to whether the screen tests listed in the analytical toxicology report would include screening that would detect Phenergan in circumstances where it's apparent from some progress notes that Laura Folbigg was prescribed Phenergan about eight weeks before her death, and this note records a response from the Ministry for Health advising that the testing performed as reflected in the analytical toxicology report was capable of detecting promethazine which is the active ingredient of Phenergan and its metabolites; and a review of the analytical data does not record the presence of promethazine. So that avenue of enquiry has been pursued to its end point.

There is a tender copy, your Honour, we have hard copies for the parties and it

will be included on the SharePoint file to which the parties have access. I note, your Honour, it's proposed the Inquiry would resume tomorrow morning at 9am with the evidence of Professor Abrams.

JUDICIAL OFFICER: Thank you. The Inquiry will adjourn until 9am tomorrow morning.

ADJOURNED PART HEARD TO WEDNESDAY 22 FEBRUARY 2023 AT 9AM