Inquiry into Convictions of Kathleen Folbigg

Submissions on behalf of Kathleen Folbigg

PART C - PATRICK

PATRICK

1. In these submissions, any reference to trial transcript will be reference to Exhibit F. All other references to transcript will relate to the evidence given at the Inquiry.

Crown Opening

2. The Crown opened with respect to Patrick's ALTE in the following terms:[1]

> *The Crown case is that this acute asphyxiating event, or ALTE, was caused by an attempted suffocation of him by his mother, Kathleen Folbigg. That is the second charge in the indictment.*
>
> *The lack of oxygen to his brain caused brain damage and a specialist paediatric neurologist will explain to you that such brain damage causes the brain tissue to die. When you get the brain tissue dying, after several days you can get swelling around the dead brain tissue. That swelling, two days later, caused Patrick to have a severe epileptic seizure.*
>
> *A second EEG examination on 23 October, that is, a week after the acute asphyxiating event, showed some abnormality. An EEG examination two weeks after that showed a significant loss of brain matter. The specialist, I anticipate, will explain to you that it was the swelling caused by the death of brain tissue which, over time, caused epilepsy and other brain damage to occur, further brain damage to occur.*
>
> *No cause could be found for the seizures, including any infection of the brain, or any other cause, apart from the hypoxic episode back on 18 October, that is, apart from the acute asphyxiating event that had caused the ambulance to come in the first place.*
>
> *It was later determined that Patrick was blind from the loss of oxygen to his brain and he continued to have epileptic seizures, which were treated with medication.*
>
> (Emphasis added.)

[1] Exh F T 36.11-.41.

3. With respect to Patrick's death, the Crown opened as follows:[2]

I would now like to take you to 13 February 1991. Of course, the ALTE was when he was four and a half months. His death occurred four months later. On the morning of Wednesday, 13 February 1991, Craig left for work at about 7.30 and Kathleen was caring for Patrick at their home.

At about 10am she telephoned her husband at work and screamed to him on the phone, "It's happened again". …

When the ambulance arrived Patrick had blue colouring around his mouth and lips. He was still warm to the touch.

He was taken to the Mater Hospital where he was seen by an emergency medicine specialist and by a paediatric neurologist, but within a short time he was formally pronounced deceased.

The emergency doctor considered that Patrick had suffered a cardiac arrest prior to his arrival in hospital and he was unable to state what had caused the cardiac arrest. The paediatric neurologist noted that Patrick's appearance was consistent with a patient who had suffered asphyxiation.

In the absence of any other explanation, this doctor felt that Patrick could have experienced an epileptic fit which had resulted in the obstruction of his airways. A doctor at the hospital, Dr Wilkinson, issued a death certificate, stating that the cause of death was the obstruction of the airways due to an epileptic fit, but that was a fair assumption on the part of the doctor.

Two pathologists at the hospital conducted the post-mortem examination and one of them will give evidence before you. They concluded that Patrick must have died from a cardiac arrest due to a seizure from unknown causes. There were no abnormalities that they found. There is no actual cause of death that they could specifically locate and they assumed that he had had an epileptic fit which caused a cardiac arrest.

(Emphasis added.)

4. With respect to both episodes, the Crown addressed the coincidence evidence which is addressed in these submissions in detail elsewhere.

[2] Exh F T 36.43 – T 37.38.

Evidence at Trial

5. At trial, there was the confusion between "asphyxia" or "acute asphyxiating event" and "hypoxia". Extensive submissions have already been made on this issue.

6. Further, there was a confusion between "encephalitis" and "encephalopathy". It is conceded that encephalitis was exclude but encephalopathy was not. Again, detailed submissions have already been made on this issue.

7. By the time of trial, many known genetic disorders were excluded by urine tests.[3] However, a genetic cause has not been identified by urine screen. This has been dealt with extensively elsewhere in submissions.

8. However, there was limited evidence regarding the neurology at trial. This may have arisen because of the confusion between "encephalitis" and "encephalopathy" and the evidential gap in the Crown case.

9. In any event, at trial there was limited consideration of any alternative causes of encephalopathy that may explain the ALTE and the death of Patrick. In particular, at trial there was no evidence to address:

   (a) Issues of immunology and infection;

   (b) Any progressive neurological condition that may have caused the symptoms.

10. Additionally, there is evidence from this Inquiry that Patrick probably had a progressive neurological condition that caused the ALTE and caused his death. This will be dealt with elsewhere in these submissions.

11. The issue of some congenital or other disorder was not considered, yet alone excluded, by the Crown at trial. There was no evidence from a neurologist on the cause of the encephalopathy advanced at trial. The highest was Dr Ouvrier who, in his report, excluded encephalitis but did not address the cause of the

[3] Exh H page 38.

encephalopathy. However, this opinion was not advanced to the jury at the trial. It can therefore be put to one side.

12. The potential cause of encephalopathy was treated simplistically by the prosecution, yet the issue was raised in the evidence of Dr Kan.

13. Dr Kan was cross-examined at Exh F T 928:

Q: And if we were to look at the range of causes that could include encephalitis as one of its varied forms?

A: Yes, encephalitis certainly is one of the causes.

Q: Also, for example, if there was a seizure, the result of epilepsy.

A: Yes. That could bring about the changes, provided the seizures were prolonged enough and there was any – and if there was sufficient deprivation of oxygen supply to certain parts of the brain, because of the seizure.

Q: And that seizure doesn't necessarily have to follow from encephalitis. It could follow from some other small part of the brain?

A: That's correct, it doesn't have to.

Q: In other words, quite separately from encephalitis there could have been a seizure here because of some abnormality in some section of the brain?

A: There could have been, yes.

Q: And that abnormality is not necessarily evident by the testing or examinations that you have done?

A: That's true.

...

Q: It [seizure] could have been caused by encephalitis in its varying forms or it could have been caused by a seizure by itself because of an abnormality in the brain?

A: That's correct.

Q: Not apparent from your examination?

A: That's true.

...

Q: And the ultimate cause of death could in fact have been a seizure in itself which has caused an hypoxic event.

A: That was certainly possible.

Q: In the background of a history of seizures?

A: Yes.

14. On re-examination, the Crown sought to clarify this issue:

Q: Doctor, you were also asked by my learned friend questions about whether or not seizures could be caused by an abnormality of the brain. Did you in fact observe any abnormality of the brain which could have caused a seizure in the first place?

A: I could not precisely pinpoint any of the lesions as the primary cause of the seizure, but any of the lesions would have been capable of triggering off seizures.

Q: My friend posed a hypothetical to you that some abnormality of the brain may have led to an initial seizure and then a seizure disorder developed following swelling of the brain. Are you able to say whether or not your findings are equally consistent with another hypothetical, which I will put to you now. If you assume that this child has been deprived of oxygen for some length of time, is it possible that his brain could have swollen and then in turn led to a seizure disorder?

A: Yes, it was possible, yes.

15. In further cross-examination, the question was asked:

Q: Could a seizure cause such prolonged deprivation of oxygen?

A: A seizure could.[4]

16. The problem with this line of examination by the Crown is that it presupposed "*abnormality of the brain*" was structural, the inference being that such an abnormality would be potentially visible. An example would be a mass lesion like a tumour, which was never found.

17. It ignores the possibility of a congenital or other disorder giving rise to a process with respect to the disturbance of respiration, neurotransmitters or cardiac function.

[4] Exh FT 931.

18. The Kan report dated 24 June 1991 was Exhibit AD in which it was found:

    (a) No convincing evidence of neuronal storage disease;

    (b) No convincing evidence of leukodystrophy;

    (c) Canavan's disease was excluded.

19. He went on to observe in his report:

> *In the deeper parts of the cerebrum and in the cerebellar and brain stem nuclei there are neurons showing simple atrophy. They could have resulted from this baby's epileptic seizures.*[5]
>
> *In the leptomeninges there appears to be light lymphoid infiltrate, which is in addition to the small amount of residual haemopoiesis normal to this age group. This could either be non-specific and related to the cortical infarcts or related to treated encephalitis (?assumed or proven).*[6]

20. He then went on to consider alternative features:

    (a) The small amount of linear cortical calcification in the occipital region is part of the laminar cortical necrosis.

    (b) No features of toxoplasmosis;

    (c) No suggestive features of cytomegalovirus infection and the distribution of the lesions is unusual for herpes simplex encephalitis;

    (d) The distribution of lesions is *far more likely to be the result of the episode of cardio-respiratory arrest this baby suffered at about 5 months of age.*[7]

21. Importantly, there was no actual respiratory arrest. Patrick was breathing at all times, but suffering difficulties breathing.

22. Dr Kan did not give an opinion as to what may have caused a cardiorespiratory arrest at five months of age. The Crown postulate was that it was due to smothering. There was no evidence of smothering. The Crown had the

[5] T 565.
[6] Exh E (Trial Exh AD).
[7] Exh E (Trial Exh AD).

obligation to exclude a reasonably available cause of death. In this regard, the respiratory arrest could have been caused by an underlying epileptic or developmental condition that had the potential to cause death, in the nature of Rett or Hunter Syndrome but as yet unidentified. This will be dealt with later in these submissions.

23. Finally, the clinical notes record back arching which can be a sign of cerebral irritation.[8] Torticollis can also be a sign of cerebral irritation.[9]

24. At this point it should be noted a progressive neurological condition had not been explored at trial.

Again I will refer you ladies and gentlemen to the summary of the medical evidence in the prosecution case relating to Patrick's ALTE. You will see there that not only Dr Wilkinson and Dezordi referred to but the other doctors, Professor Herdson, Professor Beal , Dr Beal and Professor Berry were all of the view, and I am referring to item four; that it was very unlikely that the ALTE was caused by an initial seizure because one would expect a history of epilepsy. Patrick did not have a history of epilepsy. The last sentence, "it would be extraordinary if a first epileptic seizure coming out of the blue in an otherwise fit child caused the kind of brain damage which Patrick had suffered. The medical findings were consistent with Patrick's ALTE being caused by an asphyxiating event which resulted in damage to his brain causing epilepsy."

Over the page all the findings were consistent with Patrick having been deliberately smothered and that was attested to by doctor Dezordi, Wilkinson, Carla, Kahn, Herdson, Berry and Beal. In essence what they were saying was this: The defence suggesting to the Crown's experts that maybe this brain damage was caused by a first epileptic fit. They all said you would not expect a first epileptic fit to cause brain damage of this kind. That is not, it would be extraordinary for that to happen. That is the effect of their evidence.

Let us look at what the accused said about Patrick's ALTE. We go now to her interview in 1999 with Detective Sergeant Ryan. And again, she happened to be going to the toilet. This is not a case where she said, I heard him gasping in his bedroom and I ran out to see what was wrong with him. No, no.

[8] Dr Dezordi Exh F T 482.49-483.04.
[9] Prof Ryan Exh AJ.

25. At this point, it should be noted she heard him with laboured breathing.

She happened to be going to the toilet, just like he (sic) was with Caleb. Isn't it a coincidence, ladies and gentlemen, or is it a coincidence, that in the two minutes or so between unconsciousness and death that Patrick was in, during his ALTE, that she just happened to get up to go to the toilet and happened to go into his room and discover him and raise the alarm?

26. At this point, it should be noted there is no evidence he stopped breathing. He remained pink before and after the administration of oxygen.[10]

Crown Address

CROWN PROSECUTOR: … Dr Wilkinson explained why it was that he was epileptic seizures only started two or three days later. Because it was only after several days that you had the swelling and scarring from this dead brain tissue that caused the epileptic seizures to start: He was never able to determine what had caused the original starvation from oxygen. He excluded encephalitis. He did a vast array of tests for encephalitis because one of the very rare but possible causes of such brain damage is encephalitis, caused by herpes simplex virus or some other virus. He didn't have meningitis. Extensive testing they did excluded any form of encephalitis. They did lumbar punctures extensively, didn't have a fever. When he came in they did blood tests and scans and they did EEG's (sic). All of that enabled Dr Wilkinson to say that this child did not have encephalitis. We were not able to determine what it was that had caused his brain damage. … There was no evidence of trauma, no evidence of meningitis. Blood tests disproved any severe infection.

His neurological examination on the first day was normal. No signs of pneumonia. Lots of tests for viruses that were done which were all negative. However, Patrick had a high level of glucose in his urine which suggested a fairly catastrophic event such as an asphyxiating event or a prolonged seizure. Dr Dezordi and Wilkinson were separately trying to work out what it was that this boy had and explored all types of possibilities to work out what he might have had and try and teeth (sic) out what he might have had, but in the end they were not able to determine what he had. …[11]

The post-mortem examination [on Patrick] was done by Dr Singh-Khaira. He gave evidence that he found no abnormality in the respiratory cardiovascular or any other of the body systems. He sought the opinion of Dr Kan who was a neuropathologist. We presume he is a pathologist that looks at brain and nerve tissue; that Dr Kan's opinion excluded effective causes of death, metabolic causes of death, genetic disorders, and that the changes in the brain from the past episode, the ALTE, appeared to have been caused by some event which is just a hypoxic event in the past. There was only signs of old

[10] Exh H page 79.
[11] T 1318-1319.

damage to the brain, consistent with having been done four or five months earlier. Dr Kan and Dr Singh-Khaira were unable to find any cause of death.[12]

27. These submissions require some clarification.

    (a) Dr Wilkinson was a paediatrician, not a neurologist;

    (b) In short the Crown prosecutor's submission at T 1235 is an overstatement of Dr Kan's report.

28. During his summing up, the Crown handed up a schedule which became MFI 40 and relied on MRI 39.

29. In his address, the Crown prosecutor drew attention to the likelihood of Kathleen Folbigg checking on Patrick in the two minutes between cessation of breathing and death. His address is as follows:[13]

    … We were not able to determine what it was that had caused his brain damage. Of course Dr Wilkinson again saw Patrick when Patrick died. But I'll come to that in a little while.

    We go to doctor Dezordi's evidence. Doctor Dezordi's evidence was that when oxygen was being administered in the hospital, the doctor noticed that Patrick was improving. He actually came to the conclusion that Patrick was improving without the need for oxygen. This lead (sic) Dr Dezordi to conclude that Patrick did not have any pathology, that means anything wrong with his lungs, chest or airways. Neurologically when he was admitted to the emergency section he appeared to be a normal child, nothing was obstructing his airway, no sign of long term or general or acute illness. There was no evidence of trauma, no sign of meningitis. Blood tests disproved any severe infection.

30. At this point it should be noted Patrick remained pink at all times, with no evidence of cyanosis, which one would expect with hypoxia.

    His neurological examination on the first day was normal. No signs of pneumonia. Lots of tests for viruses were done which were all negative. However, Patrick had a high level of glucose in his urine which suggested a fairly catastrophic event such as an asphyxiating event or a prolonged seizure. Dr Dezordi and Wilkinson were separately trying to work out what it was this boy had and explored all types of possibilities to

[12] T 1325.
[13] Exh F T 1319.04—T 1321.29.

work out what he might have had and try and teeth (sic) out what he might have had, but in the end they were not able to determine what he had.

Realise what was happening and raise the alarm? Let us look at question 149 in her interview. "At that stage it was close to only waking up, sort of once a night. " This is Patrick.

> *"So he used to feed around at twelve, one o'clock in the morning mark. I had done that, if I am remembering correctly, I had done that feed and everything went well. He went back to bed and so did I. Again it was a case of me finding myself awake for some reason or other and I've gotten up and thought, well, I need to go to the toilet. I'll go now and I'll check him on the way past because I had to go past to get to the toilet, when I stopped at the door to check on him, because again I passed his bed. He was sleeping in across the door, so easy access. I sort of listening for breathing and noticed it was labored (sic). It was as if he was trying very hard to draw a breath and it was, so I just immediately flung on a light and we've gone into action from there. He was lethargic, not really responsive. The eyes were shut and he was just really trying to take a breath."*

31. It should be noted this is an illogical submission.

Again I ask rhetorically; why not lift him up? How could any parent not lift up a child in those circumstances unless they were responsible for the condition that the child was in? We then go to question 172. "Was there anything unusual about that feeding? No, no. It went well. He was quite ... An hour that particular morning."

And I submit to you ladies and gentlemen that that was why she attempted to smother him that morning. She was exasperated with this child, lost her temper with this child who would not go to sleep when he was meant to go to sleep. She was going to get him to go to sleep if it was the last thing she did. She just totally lost her cool, lost her control and attempted to smother him.

32. It should be noted that if Ms Folbigg lost control in a murderous rage, she would likely have inflicted some injury on Patrick. Further, this is inconsistent with the evidence of Craig Folbigg who noticed nothing untoward regarding her behaviour proximate to this event.

Question 186 she says "he used to only take 15, 20 minutes to feed ... And I could go back to bed."

In other words, I was entitled to my sleep, I couldn't go to sleep if they were awake, so they had to go to sleep. Then she said she was up again at three o'clock, told Dr Dezordi that. Then we go to question 189. She says again, "I didn't look at the clock, I couldn't tell you what time it was. I decided I am awake, I may as well get up and go to the toilet ... he didn't sound like he was breathing properly."

She admitted question 445. That it may well have been Craig that picked Patrick up. It was put to her in a question that Craig had said that he would pick Patrick up and she agreed that that may have been the case.

Ladies and gentlemen, what a coincidence she happened to wake up in the approximate two minutes between the cessation of breathing and death. Between her hearing a high pitched cry and the few seconds later that it took for Craig to get into that room, he was unconscious and gurgling for breath. Between, say, one am the previous feed and six am the next morning, there were three hundred minutes; that she happened to get up to go to the toilet in two minutes or so that he was gasping for air. Is that mere coincidence? The Crown says it's not. If you combine that with the fact that Craig had only just gone back to work three days earlier, if you combine it with the fact that she failed to pick up Patrick, there is only one conclusion that you could reasonably come to, we submit, that is that she caused the acute life threatening event that Patrick suffered when he was four months old.

Judge's Summing Up

33. The trial judge summed up in the following manner:

That lumbar puncture was clear, which suggested no disease process. I think at that stage Dr Dezordi was ready to exclude meningitis as a cause of the symptoms from which Patrick was then suffering. … He excluded infection as a cause of the seizures. He concluded that the cause was unknown. He was ultimately of the view that the seizures did not cause the abnormality; it was the other way round. (62-63)

There was a discharge summary when Patrick was sent home, which included the expression "probably viral encephalitis". You will remember Dr Dezordi was asked about this by Mr Zahra. He described it as "a working possibility" that he had in mind at the time. He was then a specialist of the experience of, I think, about two years. He has now had more than ten years (sic) experience and his view now would be quite different. Whereas then he thought that an explanation of the symptoms that Patrick had suffered was possibly encephalitis he would not now have that opinion and his expression of the chances of that, of encephalitis being the cause, was "almost not possible". That is the way he gave his evidence to you. (T 63)

Dr Wilkinson was the other clinician who attended upon Patrick. He thought that the changes which were seen in the brain could have occurred after seizures, encephalitis, or interference with the oxygen supply. That is an expression taken from a letter which he wrote, much later, after Patrick died, and after he had presumably had seen the post-mortem reports and examination results. (T 63-64)

You will remember he was asked you about that letter. It is exhibit 6. You have a copy of it. Dr Wilkinson is not now of that view. He is of the view that the most likely cause was asphyxia. (T 64)

If you look at MFI 39, you will see the opinions collated there of Professor Herdson, Professor Berry and Dr Beal. They are of the opinion that the symptoms were not

caused by seizures. They are of the view that a first seizure in a series of epileptic seizures would not cause brain damage of the kind that was ultimately found after Patrick's brain was examined after death. (T 64)

The second reason was that they would expect there to be a history of epilepsy and, of course, there was no history of epilepsy. If this was an epileptic seizure, which took place on the occasion of the second count in the indictment in October 1990 it was the first epileptic seizure. (T 64)

Addressing you on the medical evidence, the Crown asks you to accept the present day opinions of Dr Wilkinson and Dr Dezordi and dismiss as a reasonable possibility encephalitis or epilepsy as causing the ALTE. … (T 64)

34. It should be noted that a progressive condition was not raised.

Just confining submissions to the medical evidence for the moment, Mr Zahra points you to the letter of Dr Wilkinson and to at least the initial opinion, when he was on the job, of Dr Dezordi. They were at that time prepared to go into writing on at least the possibility that the effects which had resulted to Patrick came from epilepsy or encephalitis. … (T 65)

Mr Zahra says that insofar as you rely on the opinions of Professor Berry or Dr Beal you must bear in mind that they must, and they said they had to, defer to the clinicians about the condition of Patrick. The conclusion that the symptoms were consistent with seizures or encephalitis was dependent, in part, on the report of Dr Kan, the pathologist. Dr Zahra relies on his evidence. (T 65-66)

35. This is the extent of the summing up. There was no reference at all to the clinical finding on autopsy of encephalopathy. There was no reference to a progressive disease or the emergency physicians' opinions that Patrick had suffered a cardiac arrest.

Evidence at Inquiry - Neurologists

36. Since this Inquiry, further information has become available on these issues as set out elsewhere in these submissions.

37. In this context, it is submitted on behalf of Kathleen Folbigg that the medical circumstance of Patrick alone would give rise to a considerable doubt as to whether the Crown proved its case with respect to that individual charge. In the event of such a doubt, then the charge relating to Patrick alone ought be

the subject of a report that recommends referral to the Court of Criminal Appeal for consideration.

38. With respect to Patrick, his ALTE was likely caused by the same condition that caused his death. At trial, it was the evidence that genetic factors had been excluded. The evidence of genetic factors at trial was confusing because it did not differentiate between familial disorders (which might explain the deaths of all four children) and individual genetic disorders which could give rise to a sudden death of any one of them individually, but especially Patrick.

39. The following submissions relate to Patrick only. It does not relate to the other children. It specifically relates to Patrick's ALTE.

40. It is important to note Dr Dezordi opined Patrick's condition was not likely to be due to a respiratory condition because when he was being attended upon by ambulance officers, he remained pink even when he was not administered a high concentration of oxygen.[14] The corollary of this is that had he suffered a respiratory condition that compromised his oxygen saturation, his skin would not have been pink. This observation is inconsistent with smothering. The ALTE is more likely to be some other cause that was causing difficulty breathing and incomplete oxygen saturation.

41. The evidence of back arching is important when seen in context of the other presenting symptoms. As Dr Dezordi noted, back arching could be the result of cerebral irritation. Dr Colley may be correct that it can be a common symptom. The same can be said of Dr Colley's observation of torticollis. Patrick was a seriously unwell child on 18 October and Prof Fahey would not commit to him being a normal and healthy child before that date.[15] That he was previously perceived to be well and healthy is not to the point.[16] He had torticollis and a history of arching his back.

[14] Exh H page 75.
[15] See T 586.33-.46.
[16] Counsel Assisting submissions Part 3 at [4].

42. Prof Ryan accepted it was possible Patrick suffered a single hypoxic ischaemic episode on 18 October 1990.[17] A clear EEG at the time of admission does not preclude seizure activity.[18] This is not the end of this issue. The question is what caused the hypoxia and whether it was total or relative hypoxia.

43. There is no doubting Prof Ryan's qualifications as a paediatric neurologist.

44. The submission of Counsel Assisting neglect to address critical information provided by Prof Ryan (with which Prof Fahey did not disagree) that genetic disorders can only be identified in about one third of cases of severe neurological dysfunction:[19]

> *WITNESS RYAN: The, the issue with the genetic testing is that, even in the very best of hands and with the very best genetics, geneticists using the most up to date databases that in children with unexplained genetic disorders and, and only undertake Whole Exome Sequencing or Whole Genome Sequencing we identify a causative mutation or a genetic cause for their presentation in only about a third of cases. So in, in instances where it's very, very clear that there's a, an underlying genetic disorder by virtue of the family history or the clinical presentation, we, we still are unable to identify the genetic cause of that in the majority of instances and my concern is that this is one of those instances in which there, there might be an underlying genetic problem but we haven't been able to identify it with the knowledge that we have in 2019.*
>
> *FURNESS SC: I don't understand, Professor, why you're assuming that there is an unexplained genetic disorder; can you help me there?*
>
> *WITNESS RYAN: I think that the, the - in my report I laid out a number of things about Patrick's presentation and course which I felt were atypical, things about, the, the specific things which I can go through if, if you'd like me to do that which didn't go along with the expected trajectory of a child who has a, a, an acute hypoxic ischaemic insult at the age of four and a half months and then has neurological residua of that insult. There were other thing that, there were things at the time and subsequent to that initial presentation which appeared to me to be atypical of the course of, of a child having sustained that sort of insult and so my question was whether he, in fact, had a different condition, a different diagnosis and, in children with progressive neurological disorders in the first 12 to 18 months of life, a genetic cause would be one of the top two or three things that you would consider in that instance. You would consider things like infection, but they were excluded. You would consider those metabolic disorders which are easily excluded but they were excluded as best they could do at the time, and then you would consider things like genetic disorders as well, and so that's how I came to that suggestion.*

[17] T 587.
[18] Exh F T 453.55.
[19] T 853.48-T 584.467.

FURNESS SC: And the genetic disorders that you considered have been the subject of testing and the result of that testing is that those genetic disorders were not found to be present in Patrick; do you understand that?

WITNESS RYAN: I do understand that the testing that has been undertaken has included, as best we can in 2019, those known genetic causes of those presentations, but if I, for example, took patients - I, I, I suggested a number of alternative possible diagnoses in my report. One of them, just for, as an example, is a condition known as alternating hemiplegia of childhood in which children have developmental delay, fluctuating movement disorder, fluctuating feeding issues and changes in their tone over time. If we took all children with that clinical diagnosis in 2019 and subjected them to genetic testing, we would not find a genetic cause in all of those patients and that's because there are unknown genetic causes of that presentation at that time - at this time. So the genetic tests that we have, I guess, I'm suggesting is, it, it does not identify all of the neurological - the cause of all of the genetic neurological disorders that we see in infancy.

(Emphasis added.)

45. This being the case, these passages demonstrate the clear lack of scientific understanding of the course of such conditions. It demonstrates that in the vast majority of cases, the cause is unknown. This entirely undermines the opinion of Prof Fahey that because no pathogenic genetic mutation was found after genetic testing, all recognised genetic conditions are now excluded as the cause of Patrick's ALTE and death.

46. Both Professors Fahey and Ryan agreed that one third of neurological conditions are capable of identification by whole exome or whole genome testing.[20] Prof Fahey hoped that medicine would start to push it over 40 per cent.

47. There are two issues with this evidence[21] and the submission of Counsel Assisting.[22]

48. The first is the use of the words "pathogenic genetic conditions". There was a debate between the geneticists regarding the pathogenicity of Hunter Syndrome.

[20] T 606.39 - T 607.4.
[21] Exhibit AK report of A/Prof Michael Fahey pages 4 and 16; and T 588.24.
[22] Part 3 paragraph 54).

49. Dr Kirk thought it was highly unlikely to have caused the death of Patrick but his opinion was qualified by the lack of further testing. This does not exclude a digenetic trigger for the onset of symptoms or a combination of a genetic cause and exogenous stressor, like infection or fever.[23] Patrick was running a fever on the night before his death. In other words, the Hunter Syndrome of itself may not have been pathogenic of itself but in combination with other things, it could have become pathogenic. Prof de Vinuesa had a similar experience with four deaths in one family about which she gave evidence.

50. Returning to the ALTE, the addition to the debate had between the neurologists regarding genetic causes of Patrick's ALTE and subsequent presentation, a disagreement developed between Prof Ryan and Prof Fahey as to whether Patrick had suffered hypoxia or from a hypoxic insult on 18 October 1990.

51. Professor Fahey opined that "*the face value is that Patrick was hypoxic at that time*": being upon his presentation to hospital on 18 October 1990.[24] He went on to opine that Patrick had suffered hypoxia "at some stage"[25] and reiterated his acceptance of oxygen saturation levels recorded in the clinical records "on face value"[26] whilst acknowledging "*the potential for spurious results*".[27] The controversy is not resolved by the evidence at trial, that approach accepting the results at face value without reasoning[28] nor exploration.

52. Whilst Professors Fahey and Ryan disagreed with respect to accepting the oxygen saturation levels as a reliable indicator of hypoxia, Prof Fahey conceded that Prof Ryan had, nonetheless, taken the face value presentation of oxygen saturation level of 88 per cent into account when formulating her opinion.[29]

53. Professors Fahey and Ryan disagreed on the adoption of scientific literature regarding drowning to ground opinions concerning whether Patrick had

[23] Skinner T 511.50 – T 512.26.
[24] T 590.27-.30.
[25] T 590.34.
[26] T 590.28.
[27] T 598.15.
[28] Exhibit F, T 452.10-13 per Dr Dezordi.
[29] T 588.47-589.7.

suffered hypoxia. That controversy was not adequately resolved though Prof Fahey indicated that he had limited his use of that data to the evolution of EEG in a given person following hypoxia.[30] That qualification did not appear in his report and did not adequately address Prof Ryan's concerns.[31]

54. Dr Colley's intervention into the debate[32] added nothing of relevance to resolve the disagreement between the neurologists and was dealt with by Prof Ryan.[33]

55. Prof Ryan's point regarding end organ injury[34] was not addressed by Prof Fahey save to reference the grammar of the study referenced.[35]

56. That study[36] is prefaced by the commentary (citations omitted):

The vast number of publications on near miss SIDS suggests that infants either die or recover rapidly following such an event. At most, subtle neurological abnormalities occur. Only a single report suggests that significant neurological dysfunction may occur.

57. The conclusion of the paper, in light of its findings, comments that:

It remains surprising, however, that reports of significant neurological deficits are near miss SIDS are infrequent.

58. The paper itself related a study of infants admitted to the Royal Alexandra Hospital for the period 1 June 1982 to 30 September 1985. It noted that:

... the study identifies a selected group of infants who were resuscitated from a 'near miss' event who showed clinical evidence of hypoxic derangement of at least one, and often many, organ systems.

59. Prof Fahey sought to draw the distinction, referencing this portion of the study, in his evidence:[37]

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[30] T 597.14-25.
[31] T 597.22-26.
[32] T 598.
[33] T 598.50 - 599.10.
[34] T 599.17-560.4.
[35] T 560.9-16.
[36] Constantinou, J. E., Gillis, J., Ouvrier, R. A., & Rahilly, P. M. (1989). Hypoxic-ischaemic encephalopathy after near miss sudden infant death syndrome. Archives of Disease in Childhood, 64(5), 703–708.
[37] See T 560.9-16.

WITNESS FAHEY: So I wondered about this issue and I read the Constantinou paper back and forth as recently as this morning, thinking about this, they've got an inclusion criteria of, "or" so it's not a "and" liver failure "and" kidney failure, is my first point, and one of the ors is neurological impairment presenting with seizures, so they included people just like Patrick and the other support for that is Professor Ouvrier who gave evidence in the initial hearing, is the co-author on that paper and makes the point that Patrick was the very sort of person that they would've included in his series.

60. The study, when property considered, described "*at least one, and often many, organ systems*" showed hypoxic derangement following a hypoxic event or insult. This detail was not considered by Prof Fahey and it contradicts his opinion. This study is consistent with Prof Ryan's views that it is more likely ("often"), had Patrick suffered hypoxic insult, that there would be hypoxic derangement in other organ systems, and, "*Patrick was not presenting like a child who had sustained a significant hypoxic-ischaemic insult*.".[38] Patrick did not have any evidence of dysfunction in other organ studies.

61. Certainly, Prof Fahey did not give evidence that liver, kidney or other end organ injury was not a potential consequence of hypoxia.

62. Ultimately, Prof Ryan commented that:

... if a child is unwell enough to present after an acute hypoxic ischaemic insight, you'd expect that additional blood tests would be done, other than showing the one, there was a urine test that showed glycosuria but the other blood tests were essentially normal, he didn't have a whole battery of things done but the limited testing ...

63. This point was not taken up by any of the expert witness, save Prof Ryan. Dr Colley's general comments suggested that this was exactly the approach that would likely have been taken following such an event:

When a catastrophic event happens, obviously mothers, families and doctors try to go back into the history and say is there anything, anything possibly that could give us some clues, and so you often have an overzealous natural wanting to find something beforehand ...[39]

[38] Exhibit AJ page 15.
[39] T 591.40-45.

64. The failure to administer more than a urine test when combined with the presentation of Patrick as being "*pink*"[40] and the observation of Dr Dezordi that Patrick was "*getting better spontaneously...he was improving despite, or without, the actual need for oxygen*"[41] are key indicators that the true treating medical practitioners on the day in question considered that nature of Patrick's condition on 18 October 1990 was not hypoxia otherwise there would have been a different clinical management strategy revealed in the treating records. There was not and this is powerful evidence telling against hypoxia and the opinion espoused by Prof Fahey.

65. Whilst Prof Ryan was not provided with Dr Dezordi's evidence from trial for the preparation of her report, the contents of that evidence, far from contradicting her opinion, supported her hypotheses.

66. Prof Fahey, whilst willing to accept select evidence from clinical notes (such as oxygen saturation levels) on face value, was not willing to accept other evidence on face value, such as the ophthalmological results.[42] Prof Fahey's reasoning was to question the observations of medical practitioners at the time and those that reported to same. Those witnesses were not cross examined in this Inquiry nor at trial.

67. Prof Fahey also cavilled with the use of the term "progressive" and preferred "evolving".[43] Counsel Assisting did not re-examine as to the gravamen of the distinction and, frankly, nothing turns on this.

68. Prof Fahey did not dismiss Prof Ryan's opinions nor Prof Ryan his. The submission by Counsel Assisting then that Prof Ryan's opinion should be rejected[44] is without basis. It is an available scientific opinion based on the progression of symptoms, and the reasons are well set out in her report. There was no detailed cross-examination that challenged the observation she made

[40] Exhibit F, T 448.10-12.
[41] Exhibit F, T 448.2-7.
[42] T 609.30-48.
[43] T 608.50 and T 609.5.
[44] Crown Submissions, Chapter 7, Part 3, paragraph 72.

regarding the progression of symptoms over several months. The base clinical assumption is that after a hypoxic episode, the cerebral impairment is immediate and cerebral impairment improved with time. The key issue in Patrick's case is not had he suffered from hypoxia. There is powerful evidence telling against a hypoxic episode in the terms advanced by the Crown at trial. It is not the potential for late onset epilepsy (which was suggested by the drowning studies) but the gradual deterioration over time when the ordinary course should be gradual improvement.

69. Prof Fahey thought that Prof Ryan's postulate that the ALTE may have been caused by a seizure was "less likely"[45] but, at no stage during his evidence, excluded that possibility nor derided its reasonableness. A seizure could cause transient relative hypoxia.

70. Certainly, Professors Hilton, Duflou and Cordner all agreed that Patrick's ALTE could have been caused by an epileptic seizure.[46] No mention of this evidence is made in Counsel Assisting's submissions who preferred to cite the evidence of Dr Cala alone, of four forensic pathologists, on this point.[47]

71. Ultimately, Prof Fahey selected evidence he was willing to accept on face value, the effect of which was to ignore the deteriorating and inconsistent presentation of Patrick post 18 October 1990 and, in particular the opthalmological observations and physiotherapist observations.[48] This deterioration was inconsistent with a hypoxic event.

72. On the other hand, Prof Ryan's acceptance or otherwise of the oxygen saturation levels as being 'on face value' evidence of hypoxia makes no material difference to her opinion given the presentation of Patrick following that date. Indeed, Prof Fahey conceded Prof Ryan had taken same into account.[49]

[45] T 593.33-46.
[46] T .270.17 to T 271.13.
[47] Paragraph 44 of Chapter 7 of Counsel Assisting's submissions.
[48] T 609.43-48.
[49] See footnote 13.

73. In this regard, it is tolerably clear Prof Ryan undertook a retrospective analysis of Patrick's ALTE, ie taking into account the presentation of Patrick after his ALTE to arrive at a postulate for its cause, whilst Prof Fahey appears to have undertaken a prospective analysis by assuming ALTE was as a consequence of a hypoxic insult and, consequentially, draws inferences from that assumption as to the clinical presentation that followed thereafter.

74. Whilst Counsel Assisting references an asphyxiating event in submissions,[50] there is no evidence of such an event having occurred. Patrick was breathing when discovered by his parents but was having obvious impairment for a prolonged period. He remained pink at all times. The issue discussed and analysed by Professors Fahey and Ryan related to a potential hypoxic event and evolution of Patrick's medical presentation post 18 October 1990. The evidence is silent as to the cause of the hypoxic event on 18 October 1990, if it be that.

75. The evidence there may have been a natural cause for Patrick's ALTE comes from Prof Ryan in her analysis of the medical picture presented by Patrick post 18 October 1990.[51] Prof Ryan maintained this opinion when giving evidence in the Inquiry. That opinion should not be rejected as urged by Counsel Assisting, it was not rejected by Prof Ryan's peer, Prof Fahey. Under the circumstances, the balance of Counsel Assisting's submissions regarding Patrick's death need not be considered, they being premised on only one view being open to the judicial officer. This is far from the case. Prof Ryan's opinions should be accepted by the judicial officer as consistent with the scientific literature, logical, applicable to the specific circumstances of Patrick and unrejected by her peer with whom she gave evidence. It remains a valid medical opinion.

[50] Paragraph 75 of Chapter 7 of Counsel Assisting's submissions.
[51] Exhibit AJ page 15: " This suggests that he had a fluctuating picture- potentially more consistent with a metabolic or other encephalopathy- rather than a fixed neurologic deficit related to a static hypoxic-ischaemic injury sustained some months earlier."

Immunology and Infection

76. The evidence at trial established the following:

    (a) That Patrick had a fever the night before he died;[52]

    (b) Fever is a symptom or sign of infection;

    (c) On autopsy, there was inflammation consistent with infection;

    (d) On autopsy, there were bacteria isolated in tissues;

    (e) There was congestion in the lungs and x-ray suggested his lungs were consistent with bronchiolitis;[53]

    (f) Post-mortem blood cultures grew mixed bacteria.[54]

77. The issue of contamination has been raised elsewhere in these submissions, but Patrick was showing clinical signs of infection on the night before his death. Infection can cause cardiac arrhythmia. This was not addressed at trial.

78. Christopher Walker opined Patrick had sustained a cardiac arrest prior to this arrival in hospital.[55] This is evidence that supports a finding Patrick suffered from an arrhythmia that caused his death. The infection could have combined with an underlying genetic variant that triggered his death. Submissions have been made on this issue elsewhere.

79. In any event, we submit this is an alternative natural cause of death.

80. The autopsy report read:

    Clinical diagnosis

    1. *Encephalopathic disorder leading to intractable seizures. The underlying cause of encephalopathy not determined on investigation.*

[52] Exh H page 43.
[53] Exh H page 38.
[54] Exh H page 47.
[55] Exh H page 66.

2. *Asystolic cardiac arrest at home leading to death.*[56]

81. Dr Beal opined at trial with respect of the death of Patrick:

> *Children who have epileptic fits at that young age usually have a disease process that causes the fit, and you usually can find that either on EEG or at autopsy. Things like Canarvons; there are number of disease process (sic) that present in that young age group as fits. It is not like febrile convulsions which present later, and they can happen in perfectly normal children. But in a child presenting at that young age with fits, you would usually find another disease process and I won't go into all the ones you would look for.*[57]

Ms Folbigg's Submissions

82. In the autopsy report, Patrick's cause of death was characterised as undetermined. As such, Dr Duflou advised it is difficult~~y~~ to ascribe a cause of death with any certainty. There were no signs of abuse, no history consistent with Munchausen by Proxy, no signs of smothering.

83. At the time of death, Patrick was seriously unwell due to his encephalopathy. He had a history of epilepsy.

84. There are a couple of points to be made here:

(a) No expert suggested that at the time Kathleen Folbigg attended on Patrick, he had stopped breathing. Her clear and unchallenged evidence was that he was breathing but his breathing was laboured.

(b) If he had stopped breathing (which is not the case) there was no resuscitation intervention by Kathleen Folbigg or Craig Folbigg that would explain why it was he recommended breathing (which was observed by the ambulance officers on arrival);

(c) Kathleen Folbigg's account was that as she passed the room she could hear laboured breathing. This was consistent with all of the evidence of the ambulance officers and Dr Dezordi;

[56] Exh V page 1, Inquiry Exh E.
[57] T 1139.40-.50.

(d) It was clear the Crown was using coincidence reasoning to resolve the cause of the ALTE in the absence of any physical sign of smothering. If Ms Folbigg was in a blind range, the likelihood is there would have been some injury.

85. The submission by the Crown at trial was wrong insofar as he sought to advance the proposition that Patrick had stopped breathing. This is not a matter of semantics. It was a misrepresentation of the evidence. A finding should be made to this effect.

86. The clinical cause identified in the contemporaneous clinical records and blue book of Patrick[58] is consistent with Hunter Syndrome, or a similar neurotransmitter disorder which can cause ALTE, and respiratory and cardiac problems. The monogenetic cause may have been answered but not a digenetic cause or one involving infection or fever.

87. The statement by Dr Dezordi that back arching is consistent with cerebral irritation is undoubtedly correct. Its temporal connection with other cerebral symptoms, such as epilepsy and obtunded consciousness is indicative that it was caused by cerebral irritation. The possibility that it was caused by reflux should be treated with extreme caution given the negative barium swallow that was administered on request by Dr Springthorpe.

88. The progression of cerebral symptoms confirms the back arching noted in the records is corroborative of the back arching being caused by cerebral irritation. The fact the mother reported back arching prior to this event on the admission on 18 October 1990 is strong evidence of pre-existing cerebral irritation.

89. Evidence presented at the Inquiry established there was a high likelihood of a disease process that was not identified at trial and more importantly, not looked for as an alternative cause to smothering. This evidence included:

(a) The report of Professor Ellaway;

[58] Exh H Exh AL.

(b) The geneticist's results of Prof De Vinuesa;

(c) The evidence of Prof Ryan.

90. Further, if the Crown postulate that the ALTE was caused by smothering, it gave rise to a hypoxic event, whereby the brain was starved of oxygen and brain injury ensued. Evidence given by Prof Ryan (paediatric neurologist) was to the effect the progression of symptoms from the time of admission to the time of death as revealed by the contemporaneous treating records did not follow the pattern that would be expected from hypoxia.[59] Accordingly, the Crown postulate is open to considerable doubt.

91. The death could have been caused by arrhythmia (this was the view of the emergency physician) or an epileptic fit.

Submissions of Counsel Assisting

92. At paragraphs 43 and 44 of Chapter 7 of Counsel Assisting's submission, an attempt is made to summarise the view of the forensic pathologists who gave evidence in the Inquiry.

93. The summary of the evidence found at paragraph 44 of Counsel Assisting's submissions omits or ignores important answers and qualifications given by the forensic pathologists: particularly those of Professors Hilton, Duflou and Cordner who all agreed that Patrick's ALTE could have been caused by an epileptic seizure.[60] No mention of this evidence is made in Counsel Assisting's submissions.

94. Counsel Assisting's submissions at paragraph 49, following the first sentence, represents the evidence of Dr Colley, such as it was, with regards to Patrick's torticollis and back arching prior to his ALTE. Ultimately, Dr Colley's general comments are insightful, in that:[61]

[59] Inquiry T 599.17 - T 600.04.
[60] T 270.17 – T 271.13.
[61] T 591.40-45.

When a catastrophic event happens, obviously mothers, families and doctors try to go back into the history and say is there anything, anything possibly that could give us some clues, and so you often have an overzealous natural wanting to find something beforehand...

95. Such an approach appears to have been adopted at the original trial to explain the death of the four children despite a lack of any evidence to support the smothering hypothesis. The risk of unintentional bias and prejudgement is high. This Inquiry should not adopt such an approach and constrain itself to evidence that demonstrates there are clear alternative causes of death.

96. Whilst Counsel assisting correctly notes Dr Ryan's opinion, at paragraph 53 of Chapter 7, as found her report, that testing for the various conditions that may have been responsible for Patrick's death may be achieved by whole genome sequencing, no regard is had to Prof Ryan's evidence in the Inquiry.[62] The crux of her evidence in this regard was that "*There are other children in whom a similar clinical presentation is seen for which a genetic cause cannot be found*".

97. Prof Fahey agreed with Prof Ryan on this point.[63] The qualifier imposed by Prof Fahey was there was not witnessed seizure.[64]

98. Both Professors Fahey and Ryan agreed that one third of neurological conditions are identified by whole exome or whole genome testing.[65] Prof Fahey hoped that medicine would start to push it over 40 per cent. The effect of this is that genetic testing cannot exclude a neurological cause for this presentation.

99. Further, as set out in the genetics submissions, it cannot be said a genetic cause can be excluded.

100. Counsel Assisting's submission at 70 that "*The medical experts gave broadly consistent evidence at the trial that the ALTE was most likely caused by an asphyxiating event*" is misleading. Neither Prof Ryan nor Prof Fahey proffered

[62] T 604.5-.19.
[63] T 605.6-18.
[64] T 605.21-.23.
[65] T 606.39 – T 607.4.

the opinion that the ALTE was "*most likely caused*" by asphyxiation. Prof Fahey accepted that "*the face value is that Patrick was hypoxic at that time*", being upon his presentation to hospital on 18 October 1990.[66] He went on to opine that Patrick had suffered hypoxia "*at some stage*";[67] and reiterated his acceptance of oxygen saturation levels "*on face value*" whilst:

(a) Acknowledging "the potential for spurious results", and

(b) Conceding Prof Ryan had indeed taken face value presentation of oxygen saturation level of 88 per cent into account.[68]

Summary

101. There is no basis to reject the opinion of Prof Ryan.

102. The ALTE could have been caused by an epileptic seizure or a progressive brain disorder.

103. The death could have been caused by the progressive brain disorder or infection.

104. This evidence is important to raising a reasonable doubt, not only as to the guilt of Ms Folbigg, but also the admissibility of coincidence evidence and the decision to permit joint trials. This affects the reasoning that this Inquiry is entitled to deploy in assessing whether there is a reasonable doubt as to the guilt of Ms Folbigg.

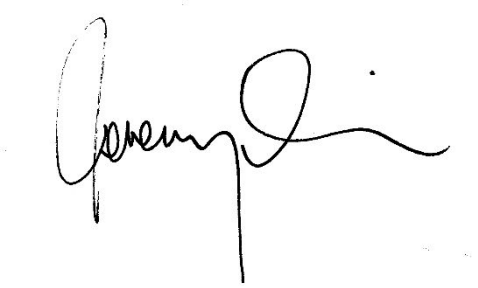
105. There was no evidence of smothering at the time of the ALTE or at Patrick's death. The coincidence notice and evidence has changed significantly since trial and cannot be used to resolve cause of death. In this regard, Ms Folbigg relies on her submissions on coincidence.

[66] T 590.27-.30.
[67] T 590.34.
[68] T 588.47-589.07.

106. There is no evidence that Craig Folbigg had any concern about his wife's emotional state at the time of the ALTE or the death of Patrick.

107. In short, there are reasonably available alternative natural causes of death that cannot be excluded. This evidence is wholly different from the evidence adduced at trial. On this evidence alone, this Inquiry should form a reasonable doubt about Ms Folbigg's guilt as to the murder of Patrick and the assault occasioning grievous bodily harm.

Dated: 7 June 2019



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