SUPREME COURT OF VICTORIA
COURT OF APPEAL

S EAPCR 2020 0184

JESSE VINACCIA Applicant

v

THE QUEEN Respondent

JUDGES:	T Forrest, Emerton and Walker JJA
WHERE HELD:	Melbourne
DATE OF HEARING:	17–19, 22–26 November, 6–7 December 2021
DATE OF JUDGMENT:	7 June 2022
MEDIUM NEUTRAL CITATION:	[2022] VSCA 107
JUDGMENT APPEALED FROM:	[2019] VSC 683 (Croucher J)

CRIMINAL LAW – Appeal – Conviction – Child homicide – Death of infant in applicant's care – Whether jury verdict unsafe and unsatisfactory – Where Crown alleged infant died from abusive head trauma ('AHT') caused by traumatic shaking – Where cause of death not disputed at trial – Whether jury could exclude possibility death caused by pre-existing medical condition – Verdict open to jury – Extension of time to file leave to appeal refused.

CRIMINAL LAW – Appeal – Conviction – Fresh evidence – Application to adduce evidence of expert witnesses casting doubt on validity of AHT diagnosis – Where evidence available at trial – New evidence inconsistent with presentation of applicant's case at trial – Evidence would not have resulted in acquittal if led – No miscarriage of justice – *Criminal Procedure Act 2009* ss 274, 276 – *Bowden v The Queen* (2017) 54 VR 135; *Ratten v The Queen* (1974) 131 CLR 510; *Lawless v The Queen* (1979) 142 CLR 659, considered.

CRIMINAL LAW – Appeal – Conviction – Admissibility of evidence – Where Crown relied upon evidence of 'triad' of symptoms found in victim to support AHT diagnosis – Whether probative value of evidence substantially outweighed prejudicial effect – Probative value of evidence high – Evidence admissible – No miscarriage of justice caused by admission of evidence – *Evidence Act 2008* s 137.

CRIMINAL LAW – Appeal – Conviction – Non-disclosure of evidence – Obligations of expert witness – Where expert witness did not disclose certain PowerPoint slides – Whether slides show witness gave incorrect evidence about existence of scientific controversy as to AHT – Whether disclosure of slides would affect outcome of trial – No miscarriage of justice – Evidence not required to be disclosed.

Counsel

Applicant:	Mr RF Edney with Ms V Drago
Respondent:	Mr CB Boyce QC with Ms A Roodenburg

Solicitors

Applicant:	Doogue & George Pty Ltd
Respondent:	Ms A Hogan, Solicitor for Public Prosecutions

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T FORREST JA
EMERTON JA:

(1) Introduction

1 On 26 June 2019 the applicant, Jesse Vinaccia, was found guilty of one charge of child homicide. He was convicted of causing the death of 16-week-old Kaleb Baylis-Clarke, by handling him in a manner that was unlawful and dangerous or, alternatively, criminally negligent. Kaleb was found to have subdural haemorrhages, retinal haemorrhages and encephalopathy — a constellation of clinical features referred to in this application (and more broadly) as 'the triad', which is said to be suggestive of head trauma. The applicant was charged following a police investigation undertaken in conjunction with the Victorian Forensic Paediatric Medical Service ('VFPMS') following Kaleb's sudden respiratory and cardiac collapse on 23 January 2016 and his death seven days later.

2 An earlier trial (the 'first trial') was aborted in early April 2019 as a result of juror misconduct.

3 The applicant now seeks to appeal his conviction, principally on the basis that the diagnosis of an inflicted head injury based on the presence of three clinical features referred to as 'the triad' was the product of 'junk science'. He proposes the following grounds of appeal:[1]

> Ground Two
>
> A substantial miscarriage of justice occurred because the expert witness, Dr [Joanna] Tully, gave evidence that was incorrect and contrary to her obligations as an expert witness and new evidence should be admitted to demonstrate how the evidence of Dr Tully has caused a substantial miscarriage of justice in the applicant's trial.
>
> PARTICULARS
>
> (i) That there is no scientific controversy, or dispute, in the scientific community as to the diagnostic utility of the 'triad' to confirm that an infant has died as a result of non-accidental physical abuse;
>
> (ii) That there is a 'consensus' in the scientific community that the 'triad' can be used to determine whether the death of an infant is the result of non-accidental physical abuse.
>
> Ground Three
>
> New expert evidence as to the cause of the death of the deceased should be admitted as that evidence demonstrates the applicant's innocence or, at the very least, create[s] a reasonable doubt as to the applicant's guilt because it suggests that the death of the deceased was due to [Benign Enlargement of the Subarachnoid Space] and not … a result of [Shaken Baby Syndrome]/[Abusive

[1] Grounds 2 and 5 were amended, and ground 1 was abandoned, in an Amended Notice of Application for Leave to Appeal, filed after the oral hearing.

Head Trauma].

Ground Four

Evidence as to the 'triad' should not have been adduced in the applicant's trial as the probative value of that evidence was outweighed by its unfair prejudice and the admission of that evidence has occasioned a substantial miscarriage of justice.

Ground Five

The verdict is unsafe and unsatisfactory or cannot be supported having regard to the evidence.

PARTICULARS

(i) The prosecution could not have … excluded the reasonable possibility that the death of the deceased was caused by a pre-existing medical condition independent of any acts of the accused.

(ii) The prosecution could not have excluded the reasonable hypothesis consistent with innocence that the acts or conduct of the accused as described in the record of interview did not amount to unlawful and dangerous act/s or criminal negligence.

4 Proposed grounds 2, 3 and 4 rely on the receipt of evidence not adduced at trial that goes to the cause of Kaleb's death. That evidence, which the applicant now seeks to adduce from three new witnesses, Professors Anders Eriksson, Knut Wester and Ulf Högberg (the 'Scandinavian witnesses'), calls into question the scientific basis for the widely accepted association between three clinical features found in Kaleb — subdural haemorrhages, retinal haemorrhages and encephalopathy — and inflicted head injury,[2] and advances alternative, organic causes for Kaleb's death.

5 For the reasons that follow, we decline to grant an extension of time in which to file the notice of application for leave to appeal.

(a) Extension of time

6 The application for leave to appeal was filed on 4 September 2020, approximately one year after conviction and sentence, and approximately 11 months after the time prescribed by s 279 of the *Criminal Procedure Act 2009* ('*Criminal Procedure Act*'). Accordingly, it is necessary for the applicant to apply for an extension of time within which to file the notice of application for leave to appeal, which he also did on 4 September 2020.

7 The principles that are relevant to such an application can be summarised as follows:

[2] The term 'inflicted head trauma' ('IHT') was used interchangeably with 'abusive head trauma' ('AHT') in evidence, and, depending on context, shall be used interchangeably in these reasons. Shaken Baby Syndrome ('SBS') has also been used in the past to convey a form of IHT/AHT.

- The applicant bears the burden of persuading the Court that an extension of time should be granted.[3]
- Time limits exist for sound reasons. Finality is desirable and in the interests of justice.[4]
- The Court has a wide discretion in determining whether to grant such an application.[5]
- The central consideration is whether it is in the interests of justice for the application for leave to appeal to be heard.[6]
- In determining that question the Court will consider the length of the delay and the reasons for it; the prospects of success of the proposed appeal;[7] and any other relevant circumstance.[8]
- Ordinarily where the delay is considerable the Court will not grant the extension unless it is satisfied that the proposed grounds are sufficiently meritorious to justify the grant of the extension, notwithstanding such delay.[9]

8 The applicant's solicitor, Mr Luke McMahon, filed an affidavit in support of an extension of time on 4 September 2020. In that affidavit, which has not been challenged, he set out various logistical challenges that accompanied the applicant's change of solicitors in late October/early November 2019; the very large volume of material that needed to be examined so as to prepare appropriate appeal grounds; the steps taken to seek opinions from the Scandinavian witnesses; and the impact of the COVID-19 pandemic on the efforts to prepare the written case for the applicant, as well as on the broader preparation for the appeal.

9 It is sufficient to state that we are satisfied that the applicant's current solicitors have acted with efficiency in the preparation of this complex application. Their explanation for the delay is persuasive and we would have granted leave for an extension of time had we have been satisfied that one or more of the proposed grounds of appeal were meritorious.

(b) Procedure in relation to new evidence

10 As we have indicated, this has been a complex application for leave to appeal. In addition to hearing from the three Scandinavian witnesses, the applicant tendered more than 36 documents, many of which were extensive and of a highly technical nature. The respondent was permitted to call evidence in rebuttal from Dr Joanna Tully (a forensic paediatrician from VFPMS who investigated the cause of Kaleb's death), Dr Linda Iles (a forensic pathologist who performed a post-mortem examination of Kaleb) and

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3 *Barber v The Queen* [2018] VSCA 232, [3] (Kyrou and Kaye JJA) ('*Barber*').
4 Ibid.
5 Ibid; *Madafferi v The Queen* [2017] VSCA 302, [11] (Priest, Hansen and Coghlan JJA) ('*Madafferi*').
6 *Kentwell v The Queen* (2014) 252 CLR 601, 613–14 [30]–[32] (French CJ, Hayne, Bell and Keane JJ); [2014] HCA 37.
7 *Madafferi* [2017] VSCA 302, [11]. See also *Woods (a pseudonym) v The Queen* [2021] VSCA 105, [7] (Kaye and Niall JJA).
8 *Barber* [2018] VSCA 232, [3] (Kyrou and Kaye JJA).
9 Ibid.

Professor Michael Ditchfield (a paediatric radiologist who examined MR images of Kaleb's head). The respondent also tendered numerous technical documents and papers.

11 We shall deal with a procedural issue at the outset. On 21 June 2021 at a directions hearing in this matter the respondent objected to the applicant being permitted to advance grounds of appeal based on the new evidence as they were said to be inimical to the manner in which the trial was conducted and it was therefore futile for the Court to receive the new evidence. The respondent submitted that, 'the proposed new evidence could not lead the Court of Appeal to conclude that the conviction should be set aside'.[10] On 17 August 2021, the Court determined that it was 'neither necessary nor appropriate … to rule finally on the objection at this stage. That will be a matter for the bench which hears the substantive application for leave to appeal … Specifically, that will be a context where the evidence will have been heard and considered.'[11] The Court went on to rule that the respondent's objection should not 'stand in the way, at this point, of the applicant preparing and presenting the new evidence ground'.[12]

12 This ruling was based on the need for the Court to hear the new evidence in order to determine whether to uphold the respondent's objection to the applicant advancing the new evidence grounds. It was necessary for the Court to hear the new evidence in order to decide whether it could lead the Court to conclude that the applicant's conviction should be set aside.

13 Theoretically, this meant that in this, the substantive application for leave to appeal, we would hear the new evidence and determine whether to admit it in the substantive application (stage 1). If we determined to admit it, we would then consider its impact on the proposed grounds of appeal that rely on that evidence (stage 2). However, the two stages cannot be sensibly separated. The evidence now sought to be adduced by the applicant which was not adduced at trial should only be received if the Court is persuaded that it must lead to the setting aside of the applicant's conviction. That requires consideration of the proposed grounds. If stage 2 is determined in the applicant's favour, stage 1 will fall away.

14 We therefore decided to hear the evidence and consider its impact on the grounds that the applicant seeks to raise in reliance on that evidence.

(c) The offence of child homicide

15 Section 5A of the *Crimes Act 1958* creates the specific offence of child homicide. That offence is committed when a person by his or her conduct kills a child under the age of six years in circumstances that, but for s 5A, would constitute manslaughter. In this case, at trial the prosecutor alleged child homicide either by unlawful and dangerous act or alternatively by gross or criminal negligence.

16 Thus at trial the prosecution assumed the burden of proving, in the case of unlawful and dangerous act:

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10 *Vinaccia v The Queen (Ruling)* (Victorian Court of Appeal, Maxwell P, Emerton and Walker JJA, 17 August 2021) [5].

11 Ibid [6].

12 Ibid [7].

(a) that the applicant committed the act that caused Kaleb's death;

(b) that the act was committed consciously, voluntarily and deliberately;[13]

(c) that the act involved a breach of the criminal law;[14] and

(d) that a reasonable person in the position of the applicant, performing that act, would have realised that he was exposing Kaleb to an appreciable risk of serious injury.[15]

17 Or, in the case of criminal negligence, the prosecution needed to prove:

(a) that the applicant owed Kaleb a duty of care;

(b) that he breached that duty by criminal negligence — that is, that his conduct involved a great falling short of the standard of care which a reasonable person would have exercised in all of the circumstances, and that there was a high risk that death or serious injury would result from that conduct;[16] and

(c) that the breach of that duty of care caused Kaleb's death.

(d) Factual overview

18 The discussion within our analysis of the grounds of appeal will be more readily understood against a broad factual background of relevant and undisputed facts. We will examine the evidence, where necessary, in greater detail when considering the individual grounds.

19 Kaleb Baylis-Clarke was born to Erin Baylis-Clarke on 4 October 2015 at Casey Hospital. Kaleb's birth was by emergency caesarean section after plans for a natural birth were abandoned. Kaleb was delivered after a healthy pregnancy, although he was undersized for a normal term baby. The circumference of his head was in the third percentile for newly born infants.[17]

20 Partway through her pregnancy Ms Baylis-Clarke had separated from Kaleb's father, Shannon Spackman. Ms Baylis-Clarke stayed with her mother for the balance of her pregnancy, then moved in with her sister, Sarah Baylis-Clarke, and her sister's partner, Sean Bertram, at an address in Cranbourne West.

21 The applicant had previously lived with Natalie Van Bree. They shared a child, Wyatt, born in December 2014, although the relationship did not survive beyond 2015. The

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13 See *Ryan v The Queen* (1967) 121 CLR 205; [1967] HCA 2; *R v Haywood* [1971] VR 755; *R v Winter* [2006] VSCA 144.

14 Cf manslaughter by criminal negligence: *Wilson v The Queen* (1992) 174 CLR 313, 333 (Mason CJ, Toohey, Gaudron and McHugh JJ); [1992] HCA 31 ('*Wilson*'). See also *Pemble v The Queen* (1971) 124 CLR 107; [1971] HCA 20.

15 *Wilson* (1992) 174 CLR 313, 333; [1992] HCA 31; *R v Holzer* [1968] VR 481, 482 (Smith J). See also *R v Klamo* (2008) 18 VR 644; [2008] VSCA 75.

16 *Nydam v The Queen* [1977] VR 430, 444 (Young CJ, McInerney and Crockett JJ); *Bouch v The Queen* (2017) 270 A Crim R 478, 505 [107] (Priest JA); [2017] VSCA 86. See also *Aston v The Queen* [2019] VSCA 225, [59], [63] (Priest, Beach and Kaye JJA).

17 Meaning 97 per cent of newly born infants have a greater head circumference than Kaleb did.

applicant knew Ms Baylis-Clarke from his school days and in late 2015 they reconnected through social media. A relationship developed and they were effectively partners from towards the end of 2015. The relationship evolved quickly and by about Christmas 2015 the applicant had moved into the Cranbourne West house. The applicant was then 22, and Ms Baylis-Clarke 21. He was an intermittently employed bricklayer, and she resumed work around November 2015 as a waitress.

22 Kaleb was brought home on about 7 October 2015 to an immaculately kept house. Ms Baylis-Clarke was a caring and proud mother. The applicant was actively involved in Kaleb's care and in housekeeping more generally. On occasions the applicant would bring Wyatt, then about one year old, to the Cranbourne West house.

23 There was a living room area in that house where a play mat and play equipment had been set up. A change table was nearby, as were a bouncer, a rocking chair and a couch for a small child. Kaleb's cot was in the children's bedroom and was a few metres from his play mat and the change table.

24 Over the course of October, November and December 2015 Kaleb was regularly assessed by maternal and child health nurses. No particular concerns were raised by the nurses about his health, although Ms Baylis-Clarke became concerned about the apparently disproportionately increasing size of Kaleb's head.

25 Kaleb was seen by his mother's general practitioner Dr Belinda Zhou at the Camms Road Medical Centre in Cranbourne on three occasions:

- On 17 November 2015 for vaccinations.
- On 4 January 2016 for vomiting that was thought to be connected to reflux.
- On 11 January 2016 for the purpose of having his head circumference measured and examined. At that stage the circumference of Kaleb's head was at the 85th percentile. While Ms Baylis-Clarke later raised concerns about bruising to Kaleb's ear and showed a photograph of this to the emergency department doctor at Monash Medical Centre ('MMC'), Dr Zhou gave evidence that she was not shown any photograph of the bruise to Kaleb's ear during this appointment and did not recall any concerns being raised about bruising at any stage.

26 On 14 January Ms Baylis-Clarke and the applicant took Kaleb to the Casey Hospital. Ms Baylis-Clarke had noticed an egg-shaped protrusion in the top centre of Kaleb's head and called the applicant at work to ask him to come home, which he did. A raised fontanelle was diagnosed. At about this time Kaleb's vomiting increased. After waiting for some hours Kaleb was transferred to the emergency area, and an ultrasound examination of his head was performed. Kaleb was transferred to MMC later that day.

27 We will examine the investigations undertaken and the resulting findings in more detail later in these reasons. For present purposes it is sufficient to state that Kaleb's head was observed to be abnormally large, and that it had grown at a concerning rate. An MRI was conducted on 15 January and mild ventricular dilation was observed together with small bilateral frontal subdural hygromas. No intra-axial haemorrhage was observed and this fact was noted.

28 The neurosurgical team reviewed Kaleb and considered performing a diagnostic tap of his fontanelle, but Kaleb's improving condition led the team to decide against such a course. He was discharged on 17 January for outpatient follow-up.

29 Kaleb returned home apparently more settled, happy and active, although he seemed to his mother to 'sleep a lot'. His mother thought he was 'basically back to normal'. Ms Baylis-Clarke was concerned enough, however, to cancel her waitressing commitments for the next week — from 17 to 23 January. She said that she 'just wanted to be [at home] to make sure he kept improving. They said, if he did go downhill again, to bring him straight back, so I didn't want to risk not being able to do that.' On 20 and 21 January Ms Baylis-Clarke texted the applicant on a number of occasions expressing concern that Kaleb was still unwell and vomiting regularly.

30 We should emphasise that it was no part of the prosecution case that Kaleb's enlarged head or apparent bruise to the ear area or any other pre-23 January condition or injury were caused by any wrongful conduct by the applicant or anyone else.

31 On 23 January Kaleb woke early. Ms Baylis-Clarke played with Kaleb for a while until he went back down for a sleep. The applicant assisted with his care. Kaleb appeared to visitors to be well and happy during that part of the morning, although when he awoke he appeared to his mother to be grizzly and overtired. In the afternoon the applicant and Ms Baylis-Clarke took Kaleb to Fountain Gate shopping centre. They returned home at about 3:45 pm. The applicant gave Kaleb a bottle. Kaleb was on his mat playing, happy and alert, when his mother left for work at about 4:30 pm. Kaleb seemed tired but well when Sean Bertram and Kaleb's aunt Sarah left to go out for dinner at 6:00 pm.

32 The applicant was then alone with Kaleb in his care. During this time he engaged with Shannon Spackman on Facebook. Mr Spackman had shared an apparently provocative post implying that he was being denied appropriate access to Kaleb. The applicant responded to the post, saying that he thought it was inappropriate. Messages were exchanged between them during which Spackman stated that he was Kaleb's father, not the applicant, and that he would 'appreciate it if [the applicant] wouldn't post photos with [his] son every day.'

33 In a record of interview that police conducted on 26 January 2016 the applicant admitted, in substance, that:

- He put Kaleb down 'a bit hard' as he was angry about a Facebook post made by Shannon Spackman.
- He picked up Kaleb from the play mat 'a bit hard' and placed him in his bed 'like, pretty rough'.
- He was 'just feeling … angry'.
- He placed him down 'probably a bit hard'.
- He had his hand under Kaleb's head (which he demonstrated) and placed him down on the bed 'so probably hit his head a bit hard on the bed'. He then wrapped Kaleb up in a blanket.
- It was 'a bit of a … swing' (which he again demonstrated) as he placed Kaleb in bed. This was not a backward-and-forward motion, but 'one motion'.

- When he carried Kaleb to his cot it could have been 'a bit bouncy and stuff'.
- He went back into the bedroom half an hour later to check Kaleb for wind and to check on him because of the way he had placed him down.
- He thought at this point, 'maybe I did hurt him'.
- Kaleb cried for between five and 10 minutes when put down.
- He believed his actions 'possibly' caused Kaleb's injuries.

34 Questions 434–7 of the record of interview consist of the following sequence of questions and answers:

> Q [I'll put it to you that when you've picked Kaleb up on] Saturday afternoon that you've shaken him with — with quite significant force. What do you say to that?
>
> A Possibly when I lifted him up, yeah.
>
> Q Is it you don't remember or - - -
>
> A No, I was just frustrated and stuff. Like, I just picked him up ---
>
> Q Yeah.
>
> A - - - And then I went in there and then it frustrated me, yeah.
>
> Q So just - - -
>
> A And put him in bed, yeah.

35 We are of the view that this sequence of questions and answers did not constitute admissions to any more than was previously admitted by the applicant, and is not capable of constituting an admission to applying violent shaking with accelerative and decelerative force. Senior counsel for the respondent to this application, having considered this sequence of questions and answers, accepts that it did not constitute an admission to shaking Kaleb repeatedly with rapid accelerative and decelerative force.

36 At 6:45 pm Ms Baylis-Clarke received a message from the applicant to the effect that Kaleb was not breathing and was 'acting funny'. At 6:46 pm the applicant called '000' for an ambulance. The operator advised the applicant to perform CPR until the arrival of paramedics, which he did. Paramedics arrived at 6:54 pm and found Kaleb to be in cardiac and respiratory arrest. They were able to restore cardiac output and Kaleb was transported to MMC in a critical condition.

37 In the immediate period following Kaleb's collapse, the applicant gave the following accounts:

(a) While paramedics were attempting to revive Kaleb, he told a CFA officer that he last saw Kaleb when he put him to bed half an hour before returning to check him. When he checked on Kaleb he was unable to wake him. Kaleb was gasping heavily every five to 10 seconds. The applicant said he tried to wake the baby

and picked him up. He then realised the baby was unresponsive and floppy and that his breathing had stopped. He called Ms Baylis-Clarke and then '000';

(b) He told other paramedics that he put Kaleb down at 6:00 pm, and went to check on him and change his nappy at 6:30 pm, when he found him unresponsive, not breathing and blue;

(c) Karen McBride, the aunt of Mr Spackman, arrived at the house with Mr Spackman. The applicant also told her that he had fed Kaleb, put him to bed, and when he had checked on him half an hour later, he was not breathing;

(d) In the car ride to the hospital, the applicant told Mr Spackman, Ms McBride and Lisa Glendenning (Mr Spackman's mother) that he had fed Kaleb, put him to bed, checked him half an hour later, lifted him out of the cot to change his nappy and then realised Kaleb was limp and unresponsive. He stated that he would never forgive himself if anything happened to Kaleb.

(e) At the hospital, the applicant told Mr Bertram, the partner of Sarah Baylis-Clarke, that Kaleb was limp when he picked him up and that he had stopped breathing. While at the hospital the applicant was obviously distressed.

(f) Kaleb's maternal great-grandmother, Joyce Clarke, noting the applicant's visible distress, asked that he be taken to a private room so that a nurse could examine him. Ms Clarke remained in that private room with him. She said that at some point Mr Vinaccia sat up on the bed, put his head in his hands and said, 'It's all my fault.' Ms Clarke asked him what had happened, and what was 'all his fault'. The applicant told her he had fed Kaleb and then put him on the play mat, where he fell asleep. He had then picked Kaleb up, put him in his bed and returned to the lounge room. Half an hour later he had thought he should check Kaleb. He had found Kaleb lying with his eyes wide open and thought he might need his nappy changed. He had picked Kaleb up and he was 'dead in his arms'.

(g) On 25 January 2016, the applicant told forensic paediatrician Dr Joanna Tully that he had fed Kaleb and put him to bed at 5:00 pm.[18] He had checked on him half an hour later, and found that Kaleb had soiled his nappy. He had picked him up. Kaleb's arms were floppy, his eyes were open, his arms were stiff and he was not breathing. The applicant had changed Kaleb's nappy before contacting Ms Baylis-Clarke. He had then dialled '000' and was given CPR instructions. Kaleb was vomiting and blue-purple in colour.

38 Kaleb was taken to MMC. He did not present with any bruising, skin discolouration or redness on any part of him. Tests taken on 23 January revealed that the extra-axial space[19] was greater than might have been expected, there was evidence of recent bleeding in the subdural space and there were no observable skull fractures.

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18 This is probably an error either on the part of the applicant or of Dr Tully (in documenting the conversation). All other evidence suggests Kaleb was put down at 6:00 pm. The Crown did not make anything of this either in this Court or at trial.

19 The space within the skull but outside the functional tissue of the brain, encompassing the subdural and subarachnoid spaces.

39 An ophthalmology consultant conducted retinal examinations on Kaleb on 25 and 26 January. Multiple retinal haemorrhages were observed bilaterally, including at the posterior pole[20] and retinal periphery.[21]

40 On 27 January Kaleb was found to have no brain activity. An MRI conducted on that day demonstrated that Kaleb had suffered a brain injury.

41 On 30 January, with the acquiescence of his parents, Kaleb's life support was withdrawn. He died at 10:02 am.

42 On 26 January, at the behest of police, Ms Baylis-Clarke participated in a covertly recorded conversation with the applicant. She asked him whether he had done anything on the night of 23 January that might have contributed to Kaleb's medical condition. The applicant denied being rough with Kaleb or shaking him. Shortly after that conversation, a police officer asked the applicant, 'Is there anything you want to tell me?' The applicant then stated, 'The only thing I can think of is that I put Kaleb down in his cot a bit hard.'

43 An 'informal' record of interview, and later a 'formal' record of interview, were conducted between the applicant and police on 26 January. The substance of the applicant's answers are set out at [34] of these reasons. The applicant was initially charged with recklessly causing serious injury. Subsequently the offence of child homicide replaced this charge.

44 No objection was taken at trial to the admissibility of the pretext conversation, or of the subsequent police interviews, and indeed the applicant relied on their content as part of his defence.

45 Dr Tully provided the opinion that Kaleb had died as a result of a traumatic head injury, most likely caused by acceleration and deceleration and rotational forces. A clinical exome trio analysis performed on Kaleb by Professor Martin Delatycki, geneticist, revealed no evidence of any underlying bleeding disorder or connective tissue disorder.

46 A post-mortem examination was conducted on 1 February 2016 by Dr Linda Iles, a forensic pathologist employed by the Victorian Institute of Forensic Medicine ('VIFM'). She concluded that Kaleb had suffered a severe brain injury with extensive bilateral retinal haemorrhages. There was no evidence of bruising to the under-surface of the scalp such as to indicate impact. All evident bruising was attributable to medical intervention. Widespread subdural haemorrhages were evident, as were deficiencies in the bridging veins.[22] Patchy subarachnoid haemorrhages were also seen, along with severe hypoxic ischaemic encephalopathy. It was this encephalopathy that led to necrosis of parts of the brain as a consequence of lack of oxygen. Ischaemic myelopathy to the spinal cord was also observed.

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20 The rear portion of the retina, including the macula.

21 The area of the retina outside the macula.

22 The blood vessels extending from the surface of the brain to the under surface of the skull.

47 Thus the post-mortem examination demonstrated severe brain injury, and that a head injury caused Kaleb's death. The likely mechanism of that head injury was shaking and/or impact trauma.

48 As indicated, we shall return to the evidence of Drs Tully and Iles at trial when considering the individual grounds of appeal.

49 Other evidence was adduced at trial. This included:

- Accounts of Kaleb's appearance and affect in the week between his 14–17 January hospital admission and his ultimate collapse on 23 January. Sarah Baylis-Clarke said that Kaleb was 'a bit sooky' and 'just wanted to be held all the time', that '[t]hroughout the week he began to vomit a little bit after feeding' and that this was out of the ordinary. Joyce Clarke saw Kaleb at a baby shower to which medical staff had allowed his mother to take him during his first hospital admission. She observed that 'he didn't look very well', seeming 'a bit lethargic', and she was surprised that medical staff had allowed him to leave the hospital. After Kaleb's ultimate discharge from hospital, Ms Clarke observed that Kaleb still 'didn't look quite right' and looked sleepy.
- Accounts of the swelling on Kaleb's head and his mother's concerns about it. Neville Holden, Ms Baylis-Clarke's stepfather, visited the Cranbourne West address on the morning of 23 January. Ms Baylis-Clarke told him that Kaleb had been sick a lot so she had taken him to hospital and fluid had been found on his brain. Mr Holden observed that Kaleb still had some swelling on his forehead, which he pointed out to Ms Baylis-Clarke. She told him that it was 'nothing compared to what it was like'. Ms Clarke recalled that around mid-January, immediately prior to Kaleb's first admission to hospital, Ms Baylis-Clarke had expressed concerns to her about 'a bit of swelling' on Kaleb's head and stated she planned to take him to the doctor.
- Maryanne Florisson, the mother of the applicant's former partner and grandmother of his child, Wyatt, gave evidence that when Wyatt was three or four weeks old, she witnessed the applicant yelling at the crying baby, saying, 'Shut up, shut the fuck up', holding him under the armpits and shaking him. This evidence was admitted as tendency evidence, said to support a tendency of the applicant to 'act out violently towards young children when in a state of anger or frustration'.[23]

50 Against this background it is convenient to begin by considering ground 5.

GROUND 5

51 Ground 5 does not rely on the evidence of the Scandinavian witnesses (collectively, the 'Scandinavian evidence'), and is formulated as an orthodox challenge to the jury's verdict on the ground that it is unsafe and unsatisfactory. Pursuant to s 276(1) of the *Criminal Procedure Act*, a court must allow an appeal against conviction if the appellant satisfies the court that the verdict of the jury is unreasonable or cannot be supported having regard to the evidence.

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23 As expressed in the Amended Tendency Notice filed prior to the commencement of the first trial.

52 This section, and others like it, have received considerable curial scrutiny since its enactment in 2009. The following general propositions can be extracted from the authorities:

- To succeed an appellant must demonstrate that it was not open to the jury to be satisfied beyond reasonable doubt of his or her guilt of the offence charged.[24]
- In determining this question the court must make its own independent assessment of the evidence, giving full weight to the jury's advantage in seeing and hearing the witnesses.
- The jury is the 'constitutional tribunal for deciding issues of fact'.[25] To set aside a jury verdict on the grounds that it is unreasonable is a 'serious step' and not to be taken without particular regard to the jury's advantage in seeing and hearing the evidence.[26]
- In most cases a doubt experienced by an appellate court will be a doubt that the jury ought to have experienced. It is only where a jury's advantage in seeing and hearing the evidence is capable of resolving a doubt experienced by a court of criminal appeal that the court may conclude that no miscarriage of justice occurred.[27]
- In a circumstantial case, a jury may only be satisfied beyond reasonable doubt of the accused's guilt if the circumstances are capable of excluding any reasonable hypothesis consistent with innocence.[28]

53 A large body of evidence was called on the appeal that concerned grounds 2, 3 and 4. None of that evidence concerns this ground, which is focused entirely on the question of whether the verdict at trial was unreasonable or unsupported having regard to evidence at trial. It is axiomatic that the jury verdict, under this ground, can only be considered on the basis of the evidence that was before the jury.

(1) The relevant evidence at trial

54 The evidence at trial relevant to this ground of appeal came largely from Drs Iles and Tully. We shall summarise their evidence.

(a) Dr Tully

55 As we have noted, Dr Tully is a forensic paediatrician employed by VFPMS. She described a forensic paediatrician as a paediatrician 'who provide[s] a medical service

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24 *M v The Queen* (1994) 181 CLR 487, 492–3 (Mason CJ, Deane, Dawson and Toohey JJ); [1994] HCA 63 ('*M*'); *SKA v The Queen* (2011) 243 CLR 400, 405 [11] (French CJ, Gummow and Kiefel JJ); [2011] HCA 13; *Pell v The Queen* (2020) 268 CLR 123, 145 [39] (Kiefel CJ, Bell, Gageler, Keane, Nettle, Gordon and Edelman JJ); [2020] HCA 12.

25 *Hocking v Bell* (1945) 71 CLR 430, 440 (Latham CJ); [1945] HCA 16, quoted in *R v Baden-Clay* (2016) 258 CLR 308, 329 [65] (French CJ, Bell, Keane and Gordon JJ); [2016] HCA 35 ('*Baden-Clay*').

26 *Baden-Clay* (2016) 258 CLR 308, 329 [65]; [2016] HCA 35.

27 *M* (1994) 181 CLR 487, 494–5 (Mason CJ, Deane, Dawson and Toohey JJ); [1994] HCA 63.

28 *Barca v The Queen* (1975) 133 CLR 82, 104 (Gibbs, Stephen and Mason JJ); [1975] HCA 42, quoted in *Baden-Clay* (2016) 258 CLR 308, 324–5 [46] (French CJ, Kiefel, Bell, Keane and Gordon JJ); [2016] HCA 35.

to children who are deemed to be at risk or in whom [there are] concerns about physical injury, sexual assault, emotional maltreatment or neglect'.

56 Dr Tully examined Kaleb on 25 January at MMC, having been told that he had been admitted to the Intensive Care Unit ('ICU') following a cardiorespiratory arrest at home. She based her report on information gathered from Erin Baylis-Clarke, the applicant, the informant Detective Sergeant Rachel Kennedy, consultant radiologist Dr Padma Rao, consultant paediatrician Dr Sarah Jame, under whose care Kaleb had come at MMC on 23 January, and medical records from MMC.

(i) Information reported by others

57 Dr Tully spoke with Ms Baylis-Clarke on 23 January and was given a brief history of the events of the previous day as well as Kaleb's medical history. Ms Baylis-Clarke told her that Kaleb had been 'his normal self' during that day, until Ms Baylis-Clarke left for work at around 4:30 pm. Kaleb had not been 'quite right' for about a month leading up to his hospital admission on 14 January: his head circumference had been increasing and he had been generally sleepier and not feeding as well as he had previously.

58 Ms Baylis-Clarke told Dr Tully that the reason she had taken Kaleb to the hospital on 14 January was because when she woke him that morning she noticed that his fontanelle was 'sort of bulging upwards' and he was vomiting more and sleepier than he normally was. An ultrasound performed at Casey Emergency Department revealed some 'abnormalities that included the possibility that he had some fluid in the subdural space'. A subsequent MRI performed at MMC showed that Kaleb had some fluid between his brain and skull and the medical team could not tell whether this fluid contained blood, as this would require drainage by way of a 'tap'. As we have mentioned, a tap of the fluid was planned but never carried out. This was because Kaleb's condition improved, and he was sent home on 17 January. Ms Baylis-Clarke told Dr Tully that, although Kaleb had stopped vomiting, she still felt that his fontanelle was bulging a little and he was still not back to 'the normal self that he had been prior to the last month'.

59 On 25 January Dr Tully spoke with Dr Sarah Jame. Dr Jame reported the same symptoms of drowsiness, vomiting, a full fontanelle and increasing head circumference leading to his 14–17 January hospital admission that Ms Baylis-Clarke had described. Dr Jame said the MRI performed during this first admission to MMC had showed some mild enlargement of the extra-axial spaces, but no sign of acute (meaning recent) bleeding. Dr Jame said the neurosurgical team had reviewed Kaleb and decided he did not require a tap of the fluid; Kaleb was discharged with a plan to review him two weeks later as an outpatient.

60 Dr Jame told Dr Tully that on his 23 January admission to MMC, Kaleb was critically unwell with subdural haemorrhages on both sides of his brain. CPR had been performed for a period of approximately 20 minutes before he was transported to hospital. On arrival at MMC emergency department, Kaleb had a Glasgow Coma score of 3, meaning he was unconscious and unresponsive. Both pupils were fixed and dilated, meaning they did not respond normally to light. His heart rate was 130, he had been intubated and a needle had been inserted into his shin bone in order to administer fluid and drugs. A ventricular tap was performed: about 60ml of bloodstained fluid was withdrawn from around Kaleb's brain. A CT scan showed acute subdural haemorrhages on both sides of

the brain, causing him to be transferred to the paediatric ICU where a second tap of intracranial fluid was performed. Dr Jame told Dr Tully that on 24 January Kaleb had an MRI scan, which showed extensive hypoxic damage to his brain, meaning damage resulting from lack of oxygen. Dr Tully said that this overall presentation — an infant who was critically unwell following a sudden collapse which resulted in his heart and breathing stopping, with subsequent CT and MRI scans giving findings that 'raised concerns about how that had been caused' — had prompted Dr Jame to refer Kaleb to VFPMS.

61 On 25 January Dr Tully spoke with the applicant about the events leading to Kaleb's admission on 23 January. He told her that he had been caring for Kaleb while Ms Baylis-Clarke was at work. He had fed Kaleb his bottle between 4:30 and 5:00 pm, after which Kaleb fell asleep on his play mat. Kaleb did not take the whole bottle, though this was not unusual, and he did not vomit. The applicant told Dr Tully that he had put Kaleb in his cot at around 5:00 pm[29] and had then gone to check on him after about 30 minutes. The applicant said that it was apparent that Kaleb had soiled his nappy so he went to lift him out of the cot to change it. Kaleb was not breathing and he was floppy when picked up, though his heart was beating. His eyes were open and his arms were stiff. The applicant told Dr Tully that he had changed the baby's nappy and then messaged Ms Baylis-Clarke to tell her that something was wrong with Kaleb. He had then listened to Kaleb's chest and found that his heart had stopped beating. At this point, the applicant told Dr Tully, he called '000' and was instructed on how to perform CPR on an infant by the call-taker. The applicant said that vomit kept coming out of Kaleb's nose and he kept wiping it away. Kaleb was blue-purple in colour.

62 In order to inform her ultimate assessment, Dr Tully also took a history of her pregnancy from Ms Baylis-Clarke. She was told that the pregnancy was unplanned but that she was happy about it. Ms Baylis-Clarke had a viral infection during her pregnancy and was induced at 37 weeks (almost full-term) because Kaleb was not growing well and his movements had decreased. As we have mentioned, Kaleb was born by caesarean section, weighing 2.52 kilograms, which Dr Tully said was 'a bit small'. He was well following delivery, no problems were identified and he was discharged on his fourth day of life. Ms Baylis-Clarke said he was generally well and had been an easy baby to care for. He was not on any medications and had his first round of immunisations.

(ii) 14–17 January hospital admission

63 Dr Tully gave more detailed evidence about Kaleb's 14 January admission to MMC, based on what she was told by Dr Jame. On that day he was taken to the emergency department at Casey Hospital, with vomiting (about five times a day) and an increasing head circumference. As previously outlined, his mother was also concerned about his raised fontanelle, which she described as being 'like an egg on his head', his intermittent crying and his reduced appetite, only taking about half the volume of milk he normally would. Ms Baylis-Clarke reported that he was otherwise developing well, was able to hold his head up and was smiling. His maternal and child health nurse had referred him to the paediatric outpatients clinic as she was concerned about his abnormally increasing head circumference. Dr Jame said that Ms Baylis-Clarke had reported previous

[29] See [18] above.

unexplained bruising to Kaleb's ear during her presentation at Casey emergency department, and to her GP, Dr Zhou. No bruising was visible on examination at Kaleb's admission on 14 January, however.

64 The ultrasound referred to above was performed by Dr Monica Pahuja. That ultrasound found that two of Kaleb's ventricles[30] were 'a little bit bigger than they normally would be' and that his extra-axial spaces appeared to be larger than they should have been. Dr Pahuja also noted probable fluid in the subdural space. This ultrasound revealed no evidence of bleeding either within the brain tissue or around the surface of the brain, however she recommended an MRI scan to be sure. In her evidence Dr Tully said that any observed bleeding would have been a significant finding.

65 The MRI scan was performed on 15 January at MMC, and reported by radiologist Dr Poonam Thakur. This confirmed the ultrasound finding that the ventricles were 'a little larger than they normally are' and that there were small bilateral subdural hygromas[31] in the frontal area of Kaleb's head. Again, there was no evidence of haemorrhage either within the brain tissue or around the brain. As such, the MRI findings, though providing more detail, were not essentially any different from those of the ultrasound; they confirmed that the fluid detected by the ultrasound was subdural fluid (ie hygromas).

66 Kaleb was reviewed by the neurosurgical team on 17 January. They asked whether there was any history of a fall and noted that Kaleb's feeding had improved since his admission. They noted that his fontanelle was full, or 'a bit bulgy', and also noted his head circumference. Having regard to the MRI findings, the team considered whether to extract fluid from the area (ie perform a tap) in order to exclude the possibility that the fluid contained blood. As mentioned, this course was ultimately abandoned.

67 In cross-examination, Dr Tully conceded that a tap of the fontanelle serves a therapeutic as well as a diagnostic purpose, reducing pressure on the brain, and that she 'would have liked for [the tap] to have been done' on Kaleb, as well as an examination of his eyes to check for retinal haemorrhages — which was also not performed — during this first hospital admission.

68 Dr Tully conceded that, due to the failure to perform a tap of Kaleb's fontanelle between 14 and 17 January, there remained a reasonable possibility that raised intracranial pressure may have persisted beyond that time, and, due to the failure to conduct an eye examination, 'we don't know whether he had retinal haemorrhages at that stage or not'. Dr Tully accepted that the gold standard for observation of retinal haemorrhages was to examine the eyes within 24–48 hours of a patient's presentation. However, while it could not be known whether Kaleb had retinal haemorrhages during his first admission, even if he did, they would not have been the same retinal haemorrhages seen on 25 and 26 January, as retinal haemorrhages almost always resolve within a week. The appearance, pattern and distribution of the retinal haemorrhages seen in Kaleb during his second admission indicated that they were recent.

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30 Spaces in the centre of the brain filled with craniospinal fluid.

31 Collections of fluid in the subdural space. The fluid has the same or similar signal intensity as craniospinal fluid.

(iii) 23 January hospital admission and Dr Tully's examination of Kaleb

69 Dr Tully examined Kaleb herself on 25 January, following his second admission to MMC on 23 January. At this point, Kaleb was 'critically unwell' and in the ICU. Her examination was 'very limited' and largely confined to a visual inspection of the front of Kaleb's body due to his fragile condition and the risk that moving him would result in clinical deterioration. Kaleb was connected to a life support machine. Dr Tully observed a small amount of dried blood around his nostrils but no visible skin injury. His pupils were small and not reacting to light. His tummy felt soft and there was no indication that that physical examination was causing him any pain.

70 Dr Tully found no 'grasping injuries' to Kaleb's trunk, and no skeletal injuries. She could not say whether there was any trauma to the third or fifth cervical spine resulting from hypertension or hyperflexion to the neck as no MRI scan was performed on Kaleb's spine. She conceded that this is a procedure now performed on every child 'in these circumstances', but was not routine at the time of Kaleb's death.

71 At this point Kaleb's head circumference was around 44 or 45 centimetres, which placed him in the 95th percentile for his age and gender.

72 A number of examinations were undertaken in order to determine the causes of Kaleb's clinical presentation. Blood tests excluded any blood-clotting disorder, any problems with his kidney function, liver function, the chemistry of his bones, enzymes produced by his heart and pancreas, his blood sugar level, and any evidence of infection. Kaleb's craniospinal fluid was checked for any infection, and studies were performed to detect any viruses. His urine was checked for infection and for any drugs of abuse. A metabolic screen was performed, to check for any underlying metabolic disorder. None of these investigations revealed anything of significance to Kaleb's presenting condition.

73 A CT scan of Kaleb's head conducted on 23 January showed enlarged extra-axial spaces and evidence of recent bleeding in the subdural space which extended into the parafalcine region.[32] No skull fracture was shown.

(iv) Working diagnosis of inflicted head trauma

74 At the conclusion of the various tests and investigations carried out on Kaleb, the working diagnosis was formed that he had a head injury caused by IHT. This diagnosis, Dr Tully said, is one that must be made with care and with rigorous attention to detail. It arises, she said, as a result of a 'constellation' of findings, including subdural haemorrhage of a particular pattern, distribution and location within the subdural space;[33] evidence of damage to the brain itself; retinal haemorrhages of a particular

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32 The large groove extending down the centre of the brain.

33 The medical experts in this case appeared to use the terms 'subdural haemorrhage' and 'subdural haematoma' interchangeably. For ease of reference, we shall refer to these phenomena as 'subdural haemorrhages'.

pattern and distribution (collectively, the 'triad injuries'[34]); and exclusion of all alternative causes.

(v) Retinal examination

75 Dr Tully gave evidence that a retinal examination of Kaleb was performed by ophthalmology consultant Dr Sophia Leikin on 25 and 26 January, examining one eye on each day. Dr Leikin detected in both Kaleb's eyes multiple retinal haemorrhages including the posterior poles and retinal peripheries. This meant there were 'lots and lots' of areas of bleeding, extending from the centre to the very edges of the retina.

76 In Dr Tully's opinion, the retinal haemorrhages observed on the 25 and 26 January would have existed on 23 January.

(vi) Dr Tully's findings and diagnosis

77 Based on her own examinations, consultations with specialists and records, Dr Tully found that Kaleb had the following conditions:

- Recent subdural and subarachnoid haemorrhages on both sides of the brain on a background of subdural hygromas.
- Mild dilatation of the spaces inside his brain, 'which can be called hydrocephalus sometimes'.
- Big extra-axial spaces; in total, the subdural and subarachnoid spaces were bigger than they should have been.
- An increasing head circumference.
- Chronic symptoms of vomiting and drowsiness.
- A past history of unexplained bruising to the ear.
- Widespread, multi-layered retinal haemorrhages in both eyes.
- A severe hypoxic ischaemic brain injury, resulting from a lack of oxygen and blood supply.

78 Dr Tully's diagnosis based on these findings was, in the absence of an explanation involving significant trauma (meaning accidental trauma), IHT. She stated, 'I think the combination of these findings, when an infant has been fully investigated for any other medical reason and there's no history of significant trauma, then we don't have another diagnosis other than inflicted head trauma.' She further stated that 'the current understanding is that … that combination of findings is most likely to be caused by forceful shaking with or without associated impact against a firm surface'.

79 Under cross-examination, Dr Tully agreed that in order to form her expert opinion, she relies in part upon the interpretations and reports of other medical specialists

[34] We use the term 'triad injuries' to describe the clinical features of subdural haemorrhage, retinal haemorrhage and encephalopathy. In using this term we are not assuming that the injuries were caused by the application of external force.

(radiologists, ophthalmologists and, to some extent, pathologists, interpreting blood tests).[35]

80 Dr Tully also accepted that Kaleb had pre-existing conditions prior to 23 January 2016, including enlarged extra-axial spaces, and that this condition may have predisposed him to subdural haemorrhage, which can occur in those circumstances either spontaneously or with 'trivial' forces.

81 She did not accept as a reasonable possibility that Kaleb's enlarged extra-axial spaces and subdural fluid may have rendered him more vulnerable to the injuries with which he presented on 23 January. While she agreed that this 'pre-existing abnormality inside [Kaleb's] head' may have slightly increased his risk of subdural bleeding (although the converse could also apply, given that the fluid cushions the brain against impact against the skull), that vulnerability did not extend to the retinal haemorrhaging in the pattern and severity that was observed.

(vii) Causal connection between shaking and triad injuries[36]

82 Dr Tully explained the connection between forceful shaking and the triad injuries in infants as follows:

> So, what happens when you shake a baby: a baby has a relatively big head compared to its body which is relatively heavy. And as you probably know, babies also have relatively weak necks, and babies, it takes a while for them to be able to lift their head, they need to strengthen their neck. What happens when you shake a baby is that generally the baby is grasped around the chest and forcefully shaken backwards and forwards. What that does is, it means that the baby's head goes back and forwards and round and round, poorly supported by a relatively weak neck and shoulder musculature; they can't splint their head, so their head is moving in multiple directions back and forward, and we call that acceleration–deceleration and rotational movement which causes/applies forces to the baby's head. That means that the brain, which is very, very soft in an infant, much softer than it is in an older child or adult, what happens to the brain is, it moves back and forwards within the skull which is fixed. What that causes is for the brain itself to sustain some damage, by banging effectively back and forward and side to side against the skull, and that those bridging veins that we talked about earlier that go from the surface of the brain up to the inner table of the skull are stretched and sometimes torn; that results in subdural bleeding. In addition, a similar process happens within the eyes. So those layers of the retina actually sheer against one another to cause retinal bleeding within the layers, and the jelly-like substance inside your eyeball moves back and forward against the retina itself, causing [bleeding] in other parts of the retina, so you get this pattern of multilayered retinal haemorrhages. The third part of this is that there is damage, we think, to the upper part of the spinal cord as it comes up through that hole and to the brainstem that sits down there at the base of the brain, so there is disruption to some of the … nerve centres … in there, that results in the infant stopping breathing, problems to their heart rate, et cetera. That therefore results in a reduction or a loss of oxygen supply to the brain. That,

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35 Though she would only agree to this proposition 'partly' in cross-examination, she stated exactly this as a limitation to her expert opinion in her first written report.

36 Dr Tully clarified during her evidence that 'the triad' is a term applied more often in legal settings than in medical settings, though it is 'rightly associated with the three features: subdural haemorrhage, retinal haemorrhage and damage or disruption to the brain, encephalopathy'.

> in combination with probably some direct damage to the tissue of the brain from the movement, means that you get injury — hypoxic ischaemic injury to the brain. It starts to swell. Like anything does when you injure it, you get inflammation and swelling. That process, and it's very complex and we don't fully understand it, that process of swelling probably further reduces the blood supply to the brain, because it's basically squashing the blood vessels, so you get what is called a secondary cascade of events within the brain that makes the actual damage to the brain sometimes unsurvivable [sic].

83 Dr Tully clarified that it is probably the combination of direct damage to the brain caused by shaking and damage to the spinal cord and brainstem that results in the cessation of breathing and cardiac arrest, rather than the subdural haemorrhages themselves.

84 She stated that the action or event that would cause cardiorespiratory arrest as a result of brain injury of this nature would almost certainly have occurred very closely in time to the infant's collapse. In particular, retinal haemorrhaging of the pattern and severity seen in Kaleb was 'indicative of significant, high-level forces being applied to Kaleb just prior to his collapse'.

(viii) Significance of other indications: absence of external injuries; torn bridging veins; increased head circumference; raised intracranial pressure; history of ill health

85 Kaleb's lack of any external injuries, such as bruising or fractures, did not alter Dr Tully's opinion that his injuries were caused by inflicted head trauma. She stated that 'probably about half, maybe slightly less than half' of babies with inflicted head trauma also present with bruising, and that some literature holds that a quarter, or up to two thirds of such babies present with fractures, 'but certainly not all of them'.

86 Dr Tully agreed that rib fractures are thought to arise from compressive forces such as squeezing on the chest. She agreed that fractures at the end of long bones (eg those in the limbs) are, when seen, important 'in terms of the mechanism' but are not always seen in cases of AHT.

87 Dr Tully stated that torn bridging veins (the condition of which can only be determined at autopsy) indicate that trauma has occurred.

88 As to Kaleb's increasing head circumference, Dr Tully stated that weighing the significance of this was difficult. While she could not determine the rate at which it was growing based on the number and intervals of measurements available to her, she confirmed that Kaleb's head circumference had increased 'more than it should have done' and the rate of the increase was 'concerning'. She stated that the likely cause of the increase was the 'enlargement of the space between his brain and the skull and fluid in the subdural space that shouldn't be there, as well as those … fluid-filled spaces in the centre of his brain being a little bigger', as shown on the ultrasound and MRI scans conducted during Kaleb's 14–17 January admission. Her opinion, however, was that the increasing rate of growth and abnormal size of Kaleb's head was not the cause of his death. She further stated that the increased head circumference was not caused by the internal trauma to Kaleb's head seen at the time of his 23 January admission; it was likely attributable to a previous event, related to the fluid identified during his first

hospital admission. Kaleb's collapse on 23 January was, in Dr Tully's opinion, caused by a supervening event: 'Something has happened in the interval [between Kaleb's two hospital admissions], almost certainly just before he collapsed, to cause that.'

89 As stated above, Dr Tully conceded the reasonable possibility that Kaleb continued to experience raised intracranial pressure beyond the time of his 17 January discharge from MMC. She accepted that the discharge summary of Casey Hospital had indicated Kaleb had 'sunsetting eyes', which is a clinical indication for 'quite significant' raised intracranial pressure of some duration. She also conceded that she had not known that sunsetting eyes had been observed in Kaleb when she had reached her conclusions as to the cause of Kaleb's injuries. Dr Tully did not, however, view the clinically observed sunsetting eyes as relevant to her diagnosis because they were observed only by one middle grade doctor, and were not documented by the paediatric consultant or neurological consultant. She stated that 'the relevance of [Kaleb's] raised intracranial pressure to his final presentation is … a complex issue'.

90 Dr Tully stated she was aware that Kaleb had 'not been his normal self' for about a month prior to 23 January, that he had been vomiting (including in the two immediately preceding days) and drowsy, and that he had had raised intracranial pressure and subdural fluid. This did not, however, affect her assessment. She did not resile from her statement during the first trial, that, '[i]n an infant that … was previously well who collapses at home with severe hypoxic brain injury, that particular pattern and distribution of subdural haemorrhage and the particular pattern and distribution of retinal haemorrhages, there's very few other causes'. She stated that, in the context of a sudden collapse, it is the relative stability of the child's condition in the preceding period that is relevant:

> [W]e're not talking about a baby who for a few hours had been becoming increasingly, increasingly, increasingly unwell. We have a state that he's more or less stable for a month. Absolutely, there is something going on and we know that, because this child has got fluid inside his subdural space. He has some raised intracranial pressure as a result of that and therefore is not, as Erin identified, hasn't been his normal self. However, when he suddenly collapsed it is that rapid and sudden change that's important.

(ix) Level of force required to cause triad injuries

91 Dr Tully stated that it is not possible to measure the magnitude of force required to cause the triad injuries, as to do so would require shaking babies with a particular measured force and noting the results. She explained that, in substitute, there have been attempts to produce biofidelic models (models that accurately represent human infants), but that results of studies using different biofidelic models are contradictory, leading to the conclusion that it is not possible to create a model exactly representing a human infant. This meant that the current understanding of the force threshold required to produce the triad injuries was based on, among other things, 'confessional data' — data collected from individuals who have confessed to shaking a baby resulting in such injuries. One study based on confessional data found that in every case the forces applied would be described as 'violent'. Dr Tully stated:

> It's certainly fair to say that these are not forces generated during normal

> handling, obviously, otherwise we would see this very, very frequently. It's highly unlikely that they are forces generated during rough handling. So while I can't … give a figure, I can't quantify the force, I think … the best we can say, is that these are forces that are well beyond or significantly beyond the normal handling of an infant.

92 Dr Tully said that retinal haemorrhages in the pattern and distribution observed in Kaleb are 'really only seen in a few other circumstances … and they are: motor vehicle accidents, high velocity, crush injuries to the head and falls from a height'. She accepted that it would be unusual for a child who had sustained injuries in such a way to present with no external injuries.

93 Dr Tully conceded that it is not known what effect Kaleb's pre-existing pathologies, in particular his enlarged extra-axial spaces, would have had on the degree of force required to produce his subdural haemorrhages. She said that this uncertainty did not extend to his retinal haemorrhages, however: '[E]nlarged extra-axial spaces in your head don't affect your eyes, and we know that to cause widespread multi-layered retinal haemorrhages requires significant forces.'

94 Asked to give some content to the term 'significant force', Dr Tully clarified that this meant force 'well beyond' ordinary handling. She explained that, as the specific pattern of retinal haemorrhaging seen in Kaleb is only seen in association with trauma in situations of 'significant force' such as high-velocity motor vehicle accidents, crush injuries to the head or falls from a height, 'we can make some assumptions, while we can't measure it, about [the] level of force required to cause it'.

95 Dr Tully's opinion was that the actions demonstrated by the applicant in his record of interview could not account for Kaleb's injuries as they would not be capable of generating the requisite force. She stated that 'significant force' of the kind described above had been applied to Kaleb, based on, at least, two of the triad injuries observed in him: the retinal haemorrhages and severe damage to the brain.

(x) Controversy surrounding connection of triad injuries and level of forces necessary for their production

96 Under cross-examination, Dr Tully agreed that the finding of the triad injuries (subdural haemorrhage, retinal haemorrhage and encephalopathy) do not 'immediately and conclusively' indicate non-accidental injury; they merely raise concerns. She accepted that there is a controversy as to the level of force required to produce these injuries, due to the aforementioned inability to conduct controlled trials of the effects of baby-shaking ethically, or to produce an accurate biofidelic model of a human infant.

97 She conceded that the available confessional data, on which she based her opinion that 'violent' force was required to produce the triad injuries, 'probably' did not include reference to a child with the same age, symptoms and history as Kaleb.

98 Dr Tully did not, however, agree that there was a medical controversy as to the ability to diagnose inflicted head trauma when the triad injuries are present with 'very specific features' and a 'rigorous and accurate medical diagnostic pathway is followed'.

99 She specified that the term 'triad' had a narrow meaning, and the diagnosis of inflicted head trauma could only be made based on the so-called triad elements when each of those elements had very specific features. Retinal haemorrhages were crucial to the ability to make that diagnosis because of their high association with inflicted head trauma.

(b) Dr Iles

100 As stated above, Dr Linda Iles is a pathologist employed by VIFM. She performed an autopsy on Kaleb on 1 February 2016, pursuant to which she made the following observations:

- Kaleb was well-nourished, and of normal weight and length.
- He had no external signs of injury. Minor bruises were attributable to medical intervention.
- His head circumference was 45 centimetres, being in the 97th percentile.
- There was no evidence of any bruising (not attributable to medical intervention) of the under surface of the skull which would indicate impact.
- There was no evidence of skull fracture.

101 Primary or acute findings (relating directly to Kaleb's collapse) were:

- Extensive subdural haemorrhages on the top and under the surface of the brain and on the membrane between the hemispheres.
- Injury to the bridging veins, which was the cause of the subdural haemorrhages. There were only a very small number of intact bridging veins.
- 'Patchy' subarachnoid haemorrhages.
- Severe hypoxic ischaemic encephalopathy with extensive cortical, subcortical, cerebella and brainstem necrosis (ie all parts of the brain were severely affected by the hypoxic ischaemic injury), due to inadequate blood and oxygen supply.
- Ischaemic myelopathy, meaning there were ischaemic changes to the spinal cord, also due to inadequate blood supply.

102 Remote findings (not relating directly to Kaleb's collapse but to earlier injury) included:

- A chronic subdural membrane, which was evidence of a previous blood collection beneath the subdural space which had since healed.
- A small area of organised left parietal extradural haemorrhages, which was evidence of previous bleeding on top of the dura. The evidence of healing indicated it had occurred prior to Kaleb's collapse, though the precise time could not be determined.

(i) Cause of death

103 Severe brain injury due to lack of blood and oxygen was, in Dr Iles' opinion, the ultimate cause of Kaleb's death.

104 No potential cause of the subdural haemorrhages other than mechanical head trauma was identified, and it was Dr Iles' opinion that that mechanism was shaking and/or impact trauma of the brain, ie a shaking injury. She could not exclude the possibility of impact despite the lack of bruising to the scalp or fracture of the skull, as an impact to a broad, soft surface would be capable of causing Kaleb's internal injuries without causing those external injuries.

105 The subdural and subarachnoid haemorrhages were not the cause of Kaleb's death nor injuries in themselves, but rather the presence of blood in the subdural and subarachnoid spaces was indicative of the mechanism by which the underlying brain was injured. The mechanism indicated was an acceleration–deceleration action, causing the brain to go backwards and forwards inside the scalp. The brain injury was caused by the effect of the acceleration–deceleration force on the brainstem, which controls respiration and heart rate. The consequent dysfunction of the brainstem and resulting lack of oxygen and blood supply was the ultimate cause of catastrophic brain injury.

106 Kaleb's injured bridging veins indicated that this acceleration–deceleration force had been applied. The resulting movement of the brain inside the cranial cavity puts tension on these thin and delicate veins, which consequently tear and produce haemorrhaging.

(ii) Eye examination

107 Dr Iles referred the examination of Kaleb's eyes externally. They were macroscopically examined and sectioned at St Vincent's Hospital in Sydney and the sections were examined by a neuropathologist, Dr Michael Rodriguez. The findings from that examination were bilateral optic nerve sheath haemorrhages and extensive bilateral retinal haemorrhages extending from the optic nerve to the ora serrata and involving all layers of the retina.

108 Dr Iles stated that the more extensive retinal haemorrhages are, the more specific they are as an indication of trauma. Kaleb's retinal haemorrhages were 'very extensive'. The presence of optic nerve sheath haemorrhages are also used as an indicator of a traumatic cause of retinal haemorrhages.

109 Dr Iles said that retinal haemorrhages are a common feature of shaking injuries, though they may be caused by a number of things. Other types of trauma associated with retinal haemorrhages include crushing injuries such as those seen in motor vehicle accidents, occipital impacts or where a heavy object has fallen on a child's head.

110 Dr Iles said that her evidence as to retinal haemorrhages was qualified by ascertainment bias — in her function as a forensic pathologist, she only examined the eyes of infants when inflicted injury is suspected. She did not have much experience of the appearance of the eyes of infants affected by accidental trauma. She stated that therefore the opinion of clinicians on the subject of retinal haemorrhages was to be preferred to her own.

(iii) Level of force required to cause Kaleb's injuries

111 Dr Iles described the same difficulties that Dr Tully had pointed out in determining with any certainty the level of force required to produce the triad injuries — the lack of accurate biofidelic models for human infants and impossibility of experimenting on real

children. The best available indicator of the requisite force was, Dr Iles said, clinical experience, and the frequency with which the triad injuries were presented: '[W]hat we know is that we do not see children with this spectrum of injuries often, particularly not this type of catastrophic head injury, so the implication for that is that the force must be significantly in excess of the normal force applied when handling a child.'

112 Dr Iles made several concessions as to the current understanding of the requisite force. These concessions can be summarised shortly here:

- There is 'consternation and disagreement' in the literature about the particular levels of force needed to produce the triad injuries in a normal case.
- It cannot be known with certainty what kind of force beyond normal handling is needed to produce the triad injuries in the normal case.
- The presence of pre-existing conditions such as Kaleb's makes the uncertainty as to the requisite level of force even greater.
- The pre-existing conditions identified in Kaleb — chronic subdural membrane (evidence of a previous subdural collection) and an expanded subdural space containing blood and/or fluid — created a potential vulnerability the significance of which Dr Iles was unable to determine. There was a reasonable possibility that his pre-existing conditions made Kaleb more susceptible to the injuries he suffered.
- She could not say whether Kaleb's pre-existing pathology had the capacity to significantly affect the amount of force that might be required to produce the triad injuries, because that data did not exist.
- She could not exclude the possibility of some extant, birth-related issues.

(iv) Whether triad injuries could be caused by the applicant's admitted actions

113 Dr Iles reported that whether the triad injuries can be produced by shaking alone, absent some additional impact, has been the subject of significant scientific controversy. She added in her oral evidence that the addition of an impact to a shaking incident would make the forces applied to the brain significantly higher.

114 Dr Iles agreed that she had not detected any injuries in Kaleb outside of the central nervous system and the eyes. There were no external marks to the trunk, fractures to the ribs or paravertebral region, no skeletal injuries from chips in the bones or from flailing limbs, nor injury to the neck (which might indicate shaking).

115 Dr Iles conceded that she could not say one way or the other whether the actions the applicant described undertaking in his record of interview, in combination with Kaleb's pre-existing conditions, caused his injuries. She accepted that what he had said was 'relatively non-specific and open to a wide range of interpretation[s]'. Given the uncertainties she had conceded as to both the requisite force for the triad injuries generally and the significance of Kaleb's pre-existing conditions to the requisite force in his case, as well as the vagueness of the applicant's description of what he did, she did not have an adequate evidential basis to determine whether his description of those events could account for Kaleb's injuries.

(v) Temporal relationship of traumatic event and collapse

116 Based on the literature she had read, and predominantly on confessional data, Dr Iles stated that there is typically a very short period between the traumatic event and the child collapsing.

(2) Consideration

(a) Ground 5(i)

117 Ground 5 makes two contentions. The first (ground 5(i)) is that the evidence at trial did not allow the prosecution to exclude as a reasonable possibility that Kaleb's death was caused by a pre-existing medical condition independent of any acts of the applicant.

118 This contention can be dismissed relatively shortly. The MRI investigation carried out at MMC on 15 January revealed existing small bilateral hygromas and mild ventricular dilation. Clinically at that time he presented with an abnormally enlarged head, and a raised fontanelle indicative of elevated intracranial pressure. A history of drowsiness and vomiting was provided. No intra-axial haemorrhage was observed and this fact was specifically noted. By his release on 17 January, his fontanelle had mostly retreated and was more settled. Upon admission on 23 January 2016 he was unconscious, his extra-axial space was greater than might have been expected and there was evidence of subdural bleeding. Retinal haemorrhages bilaterally were observed on 25 and 26 January and the autopsy conducted on 1 February 2016 revealed severe hypoxic ischaemic brain injury with extensive subdural haemorrhages and patchy subarachnoid haemorrhages. The bilateral retinal haemorrhages were also noted.

119 Dr Tully's opinion was that, notwithstanding the other pathologies listed above, the findings of severe hypoxic ischaemic brain injury, widespread multi-layered retinal haemorrhages and, to a lesser extent but not insignificantly, bilateral subdural haemorrhages, in the absence of some known significant (accidental) trauma, were diagnostic of inflicted head trauma. In the absence of any medical reason for these three findings, and in the absence of any apparent direct (accidental) trauma, there was no other diagnosis than inflicted head trauma. Further, she opined that 'current understanding is that … that combination of findings is most likely to be caused by forceful shaking with or without associated impact against a firm surface'. In cross-examination defence counsel extracted from Dr Tully the opinion she expressed in the conclusion of her report:

> 'Kaleb Baylis-Clarke died as a result of traumatic head injury. The pattern of injuries observed is indicative of inflicted head trauma with acceleration–deceleration and rotational forces being the likely mechanism. Due to the severity of his clinical presentation it is likely that the episode of trauma leading to his presentation on 23 January 2016 occurred in close proximity to his collapse.' That was, I'm reading from the words of your report?---Yes.
>
> And those are your conclusions?---Yes.
>
> You stand by them, of course?---Yes.

120 Dr Iles confirmed that, in her opinion, Kaleb's death was caused by a head injury, likely the result of shaking and/or impact. She could not exclude the possibility of impact, notwithstanding that there was no bruising or skull fracture on examination as, if an impact took place over a broad, soft surface, there may not be any visible evidence of it. In cross-examination she was not challenged on the cause of death; the focus of the cross-examination was on the force required to produce the injuries observed. We shall return to this evidence when considering the second contention under this ground.

121 No evidence was led by the defence on the cause of death.

122 It was comfortably open to the jury to conclude that the cause of death was some action undertaken by the applicant shortly before Kaleb's decline. It follows that it was open to the jury to exclude, as a reasonable possibility, that Kaleb's death was caused by a pre-existing medical condition, independent of any act of the applicant. The applicant has failed to establish ground 5(i).

(b) Ground 5(ii)

123 Ground 5(ii) is more complex and requires longer examination.

124 Under this limb of ground 5 the applicant contends that it was not open to the jury to conclude that the applicant minimised or understated his actions when describing them to police in the formal record of interview. Assuming this to be the case, so the argument proceeded, it was not open to the jury to be satisfied beyond reasonable doubt that he was guilty of the offence charged, whether it be by unlawful and dangerous act or by criminal negligence.

125 The starting point for this consideration is the formal police record of interview. We set out its written substance at [33]–[34] of these reasons. We observe that the transcript of this interview does not provide a complete record of the applicant's account. Many of his answers were accompanied by visual demonstrations. We have watched the audio-visual recording of the interview and take into account these demonstrations when stating our conclusions as to the nature and extent of the admissions made by the applicant.

126 In our view, it was open to the jury to conclude, and we conclude, that the formal record of interview discloses that the applicant became frustrated while Kaleb was lying on his play mat. The source of the applicant's frustration was Kaleb's father, who had shared, in the applicant's view, an inappropriate Facebook post and then engaged in a heated messaging exchange with the applicant. The applicant picked Kaleb up from his mat sharply and walked him brusquely to his cot some metres away. He supported Kaleb's head while doing this.[37] He put Kaleb down 'pretty roughly' in his cot, 'probably a bit hard'. To do this he supported Kaleb with one hand under his neck and the other under his trunk.[38] Kaleb probably hit his head 'a bit hard on the bed'. He put Kaleb down with one swinging motion. It was not a backward-and-forward motion. Half an hour later he went back to check on Kaleb for wind, and to check on his welfare because of the way he had placed him down.

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37 Apparent from the applicant's demonstration.

38 Apparent partially from the applicant's demonstration and partially from his verbal account.

127 The applicant appeared to us to be anxious and concerned about Kaleb's welfare throughout the interview. He appeared to be eager to provide information that may have explained Kaleb's then critically ill condition. Although the activities admitted in the record of interview contained greater detail than his initial accounts, and he disclosed his frustration for the first time during that interview, his overall account remained relatively consistent.

128 As we have mentioned at [35], we do not consider that the applicant's answers to Questions 434–7 provide any more than a further particular to what was previously admitted by the applicant. In those answers the applicant left open the possibility that when he picked up Kaleb, walked him to the bedroom and put him in the cot the baby was 'shaken' and 'with quite significant force'.[39] In the context of the entire record of interview the applicant was accepting that his already admitted actions may have 'shaken' the baby with this degree of force. As we have said, it was an admission that he may possibly have subjected the baby to significant forces when he picked him up, transported him to the bedroom and placed him in the cot.

129 The question then arises, how does this account fit with the other evidence in the case? Witnesses described the applicant as a gentle, concerned carer to Kaleb.[40]

130 Against this is the tendency evidence of Ms Florisson, who claimed to have seen the applicant yelling at the three-to-four-week-old Wyatt, holding him under the armpits and shaking him backwards and forwards. This one episode was said to be probative of the applicant's tendency to 'act out violently towards young children when in a state of anger or frustration'. We doubt this evidence advanced the prosecution case to any degree. This was a single episode, said to have happened two years before Kaleb died, and which resulted, apparently, in no injuries to Wyatt at all. Like the evidence of the applicant's good qualities, this disputed evidence of a poor quality is not direct evidence of what occurred on 23 January, or how it occurred. Further, evidence of both tendencies (that is towards gentleness and towards violence) is, in our view, of little assistance in answering the question as to the mechanism of Kaleb's death.

131 Leaving to one side the applicant's accounts, the evidence about this is almost exclusively confined to Drs Tully and Iles. This evidence is indirect in the sense that it provides post-fact evidence of injuries and then offers opinions as to how those injuries were sustained. In most respects their evidence is consistent.

132 We shall endeavour to avoid repetition of evidence that we have already set out in some detail, however, to properly analyse this ground of appeal, some repetition is inevitable.

133 Drs Tully and Iles, as we have observed, agreed as to the ultimate cause of death. They also agreed as to the mechanism that brought about death. Dr Tully stated that there must have been forceful shaking with or without an associated impact on a firm surface; she described an acceleration–deceleration and rotational movement which applied forces to the baby's head. Dr Iles broadly agreed with this and could not rule out impact on a softer broad surface notwithstanding the lack of external injury. Both doctors opined that there was a very short period between the traumatic event and Kaleb's

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39 See [34] above.

40 Evidence of Erin Baylis-Clarke and Joyce Clarke.

collapse. Dr Tully stated that the pattern and severity of retinal haemorrhages indicated forces being applied 'just prior to his collapse'. Dr Iles relied on the literature — as a pathologist, she did not have clinical experience of this.

134 Critically to the first question raised under ground 5(ii), both doctors agreed that it was impossible to measure with precision the degree of force required to produce the triad injuries. As we have said, Dr Tully stated that the 'best we can say' is that the requisite force is 'well beyond, or significantly beyond' normal handling and that mere 'rough handling' would be highly unlikely to be sufficient. Dr Iles agreed that the 'threshold of forces required to produce this constellation of findings is not known', however, 'it should be considered to be considerably beyond that associated with normal handling of an infant'.

135 As to the significance of Kaleb's enlarged extra-axial spaces, as we have observed, both doctors agreed that it may have predisposed Kaleb to subdural haemorrhages — either spontaneously or with trivial force (Dr Tully), or that it may have 'significantly affect[ed] the amount of force that might be required to produce the triad' (Dr Iles).

136 Dr Iles considered that the expanded 'subdural space'[41] made injury to the bridging veins more likely, as the veins (which span this space) would be stretched, and thus more vulnerable to tearing. Dr Iles also thought it possible that the underlying brain parenchyma[42] had increased vulnerability to injury through acceleration–deceleration forces. Injury to the parenchyma would result in encephalopathy. Dr Tully on the other hand considered that the severe encephalopathy observed in Kaleb could not be explained by his expanded subdural space.

137 Central to both doctors' opinions on the mechanism of death and the degree of force that this must necessarily have involved was the presence, extent and pattern of retinal haemorrhaging observed by an expert ophthalmologist. It will be recalled that Dr Tully sought the expert opinion of Dr Leikin, who reported that there were multiple bilateral retinal haemorrhages including at the posterior poles and the retinal peripheries. Dr Tully, who professed clinical expertise in the diagnosis of AHT,[43] opined that the enlarged extra-axial spaces in themselves could not have caused the widespread multi-layered retinal haemorrhages observed in Kaleb, which required 'significant forces' and a supervening event for their production.

138 Dr Tully's opinion was that the pattern and distribution of retinal haemorrhages seen in Kaleb's eyes is only seen in a few circumstances other than abusive head trauma; essentially these are high-impact injuries such as significant falls and high-speed motor vehicle accidents. Dr Tully considered the retinal haemorrhages to be crucial to the triad-based diagnostic process and that the pattern and distribution of those symptoms as seen in this case were only seen in association with 'trauma in situations of significant force'. Dr Iles, who relied on the expert opinion of the Sydney ophthalmologist based on slides of Kaleb's retinas, deferred to clinicians concerning the significance of retinal haemorrhages due to a self-perceived risk of 'ascertainment bias'. However, she opined

41 The subdural space is part of the extra-axial space, which also includes the extradural and subarachnoid spaces.

42 The functional tissue of an organ. Cf connective and supportive tissue.

43 See [55] above.

that retinal haemorrhages were a common feature of 'shaking injuries' and that the more extensive they are, the more specific they are as indicators of trauma.

139 The absence of external injuries did not affect Dr Tully's conclusion that Kaleb's injuries were caused by an inflicted head injury; while Dr Iles was asked to confirm the absence of external injuries evident on autopsy, she was not asked to comment specifically on the significance of this in relation to the diagnosis of inflicted head injury.

140 Dr Iles could not say one way or the other whether the applicant's admitted actions in the police interview were sufficient to cause Kaleb's injuries and subsequent death. This was because (a) the relevant force threshold is not known, and (b) the admissions themselves lacked specificity. By contrast, Dr Tully was firm that the mechanism described by the applicant in his interview would not have generated the significant force required to cause those injuries.

141 We consider that it was open to the jury to conclude that the forces applied to Kaleb were well beyond those to which the applicant admitted in his police interview. The (essentially uncontradicted) medical evidence leads inexorably to that conclusion. Whilst the subdural haemorrhages, or some proportion of them, may possibly have been caused by trivial movement, and whilst the encephalopathy observed upon autopsy, according to Dr Iles, might conceivably have been caused or contributed to by Kaleb's underlying medical condition, both the subdural haemorrhages and encephalopathy are also consistent with inflicted head trauma. The retinal haemorrhages are, however, in the circumstances of this case, *only* consistent with inflicted head trauma. On this basis, it was entirely open to the jury to accept (as we do) the evidence that the mechanism that caused death was a forceful shaking action involving acceleration–deceleration and rotational forces. This conclusion is fortified to some extent by the applicant's admissions that he was frustrated at the time, and that as a consequence of that handling he saw it fit to check on Kaleb's welfare. It was also open to the jury to consider Ms Florisson's tendency evidence, although for the reasons we have advanced we consider it to be of relatively little probative weight and we have not accorded it any weight in our independent evaluation of the evidence.

142 Assuming this conclusion was open to the jury, it was also open to the jury to convict the applicant of the offence charged.

143 Specifically, having concluded that the applicant shook Kaleb with force that went significantly beyond the normal handling of a three-month-old infant, in our view it was open to the jury to conclude beyond reasonable doubt:

- that the applicant caused Kaleb's death;
- that his actions in shaking Kaleb with the above-mentioned degree of force were carried out consciously, voluntarily and deliberately;
- that the act involved a breach of the criminal law, namely assault; and
- that a reasonable person in the position of the applicant (including being fixed with the applicant's knowledge of Kaleb's pre-existing ill health) would have realised that he or she was exposing Kaleb to an appreciable risk of serious injury.

144 Had the jury approached their reasoning in this way it was open to them to convict the applicant of the offence on an 'unlawful and dangerous act' basis.

145 Alternatively, we consider that it was open to the jury to convict the applicant of the offence on a 'criminal negligence' basis. It was open to the jury to conclude beyond reasonable doubt:

- that the applicant owed Kaleb a duty of care;
- that he breached that duty with conduct that involved a great falling short of the standard of care that a reasonable person would have exercised in all the circumstances, and that there was a high risk that death or serious injury would result from that conduct; and
- that the breach of the duty that the applicant owed to Kaleb caused Kaleb's death.

146 We observe that if we are incorrect in our conclusion that it was open to the jury to accept that the applicant's conduct extended well beyond that to which he admitted to police, on the basis of those admissions and the other evidence in the case it would still have been open to the jury to convict the applicant on a criminal negligence basis. As at 23 January 2016 the applicant knew Kaleb was only three months old; he knew that the infant had experienced chronic ill health thus far in his short life and that his head had recently swollen massively; he knew that Kaleb, only a few days earlier, had a raised fontanelle and had spent several days as an inpatient at MMC; he knew that Kaleb remained tired, irritable, vomiting and grizzly. In these circumstances, it was open to the jury to conclude that, in picking up Kaleb from his mat 'a bit hard', carrying him to his cot 'a bit bouncy and stuff', placing Kaleb in his cot with 'a bit of a swing' and so that he 'probably hit his head a bit hard on the bed', the applicant's conduct fell greatly short of the standard of care incumbent upon a reasonable person knowing all the factors outlined above, and carried with it at least a high risk of serious injury. It would also have been open to the jury to conclude that this admitted conduct caused Kaleb's death (which was not seriously contested at trial) and that the applicant owed Kaleb a duty of care (which was not contested at trial at all).

147 For the above reasons it was open to the jury to find that all elements of child homicide, whether by unlawful and dangerous act or by criminal negligence, were satisfied. Put another way, the jury was not bound to acquit.

148 Leave for an extension of time in which to file notice of application for leave to appeal will not be granted under this ground, as we do not consider it to be meritorious.

GROUND 3

(1) Introduction

149 Ground 3 is that new expert evidence as to the cause of Kaleb's death should be admitted, as that evidence demonstrates the applicant's innocence or, at the very least, creates a reasonable doubt as to the applicant's guilt because it suggests that Kaleb's death was due to a condition known as Benign Enlargement of the Subarachnoid Space ('BESS') and was not the result of inflicted head trauma.

150 The applicant contends that there has been a substantial miscarriage of justice in that the jury did not have the benefit of the evidence upon which he now seeks to rely. The applicant brought his application for leave to appeal on the basis that this was 'new' evidence, which, while available at the time of his trial, ought now to be received on appeal due to the fact that its overwhelming strength requires that he be acquitted. In submissions filed after the hearing of the appeal, he submitted that the evidence was 'fresh' evidence because, although much of it was technically available at the time of his trial, he did not have the means to access it.

151 Fresh/new evidence – Legal principlesAs the applicant recognised, the authorities distinguish between 'fresh' and 'new' evidence by reference to its availability at the time of trial. In short, evidence is 'fresh' if it was not available at the trial, or could not have been obtained by the accused with reasonable diligence.[44] Conversely, evidence is 'new' if it was either available at trial, or discoverable with reasonable diligence.[45] Both 'fresh' and 'new' evidence can be adduced to correct a miscarriage of justice, but the threshold for proving a miscarriage of justice has been held to differ for each. Broadly stated, 'fresh' evidence can be adduced to overturn a conviction if there is a 'significant possibility (or perhaps, a likelihood) that the evidence, if believed, would have led the jury, acting reasonably, to acquit the applicant if the evidence had been before it at the trial'.[46] In contrast, 'new' evidence must either show the convicted person to be innocent, or raise such a doubt about his or her guilt in the mind of the court that the verdict should not be allowed to stand.[47]

152 However, it has also been said that there can be 'no absolute or hard and fast rules'[48] and there will be cases in which the practical guidelines referred to above are inapplicable. As Winneke P explained in *R v AHK*:

> [A]t the end of the day, it should not be forgotten that the expressions of judicial opinion to which I have referred are practical guidelines which do not detract from the force of the fundamental principle that an appellate court must allow an appeal if a miscarriage of justice is shown to have occurred. An appellate court will always receive 'fresh evidence' if it can be clearly shown that the failure to receive it might have the result that an unjust conviction is permitted

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44 See, eg, *Rodi v Western Australia* (2018) 265 CLR 254, 263 [28] (Kiefel CJ, Bell, Keane, Nettle and Gordon JJ); [2018] HCA 44 (*'Rodi*').

45 *Bowden (a pseudonym) v The Queen* (2017) 54 VR 135, 143 [34] (Priest JA, Maxwell P and Kidd AJA agreeing at 1); [2017] VSCA 46 (*'Bowden*'), quoting *Mallard v The Queen* (2003) 28 WAR 1, 6 [11] (Parker, Wheeler and Roberts-Smith JJ); [2003] WASCA 296 (*'Mallard*'). See also *R v Kucma* (2005) 11 VR 472; [2005] VSCA 58; *Lawless v The Queen* (1979) 142 CLR 659; [1979] HCA 49 (*'Lawless*').

46 *Bowden* (2017) 54 VR 135, 144 [36] (Priest JA, Maxwell P and Kid AJA agreeing at 1); [2017] VSCA 46, citing *Gallagher v The Queen* (1986) 160 CLR 392, 395 (Gibbs CJ), 402 (Mason and Deane JJ), 410 (Brennan J) (*'Gallagher*') and *Mickelberg v The Queen* (1989) 167 CLR 259, 301 (Toohey and Gaudron JJ); [1989] HCA 35.

47 *Bowden* (2017) 54 VR 135, 144 [36] (Priest JA, Maxwell P and Kid AJA agreeing at 1); [2017] VSCA 46, quoting *Ratten v The Queen* (1974) 131 CLR 510, 517–8 (Barwick CJ); [1974] HCA 35 (*'Ratten*').

48 *Gallagher* (1986) 160 CLR 392, 395 (Gibbs CJ), quoting *Green v The King* (1939) 61 CLR 167, 175 (Latham CJ).

to stand.[49]

153 Moreover, as Walker JA explains at [646] of her reasons, while the tests associated with the distinction between 'new' and 'fresh' evidence are of assistance in determining whether there has, in a particular case, been a substantial miscarriage of justice, they cannot replace the statutory text of s 276 of the *CPA*. Whether there has been a substantial miscarriage of justice remains the ultimate question that an appellate court must assess when determining whether to allow an appeal based on evidence that was not adduced at trial.[50]

154 In substance, our task in this case is to determine whether the applicant has established that he has suffered a substantial miscarriage of justice as a result of his trial being conducted without the Scandinavian evidence.

(2) The new or 'fresh' evidence

155 As discussed above, following the ruling made on 17 August 2021, we determined to hear the Scandinavian evidence and evidence in rebuttal in the application for leave to appeal. Accordingly, we heard evidence from three medical experts called by the applicant: Professors Anders Eriksson and Ulf Högberg are medical specialists based in Sweden; Professor Knut Wester is a medical specialist based in Norway. The Scandinavian witnesses are known to each other, have worked together over the past few years and identify as members of a group seeking to effect a paradigm shift in the acceptance of AHT. They challenge what they say is the presumption that the presence in an infant of 'the triad' (subdural haemorrhage, retinal haemorrhage and encephalopathy — which we refer to as the triad injuries) indicates that the infant has been subjected to traumatic shaking. In fact, they seek to sever any connection between the elements of the triad and AHT, asserting that there is no scientific basis for the proposition that any one of these elements, or all three in combination, is the product of AHT.

156 At its base, the Scandinavian evidence challenges the proposition that the presence of 'the triad' of clinical features found in Kaleb upon his admission to MMC on 23 January and/or on autopsy — subdural haemorrhages, retinal haemorrhages and encephalopathy — can be used as evidence that he suffered AHT. According to the Scandinavian witnesses, there is no scientific foundation for any such association. Professors Wester and Högberg advanced alternative hypotheses for the cause of Kaleb's death, both of which were disease processes unrelated to head trauma: intracranial pressure caused by BESS and intracranial pressure caused by the rebleeding of existing subdural hygromas. This intracranial pressure was said to be sufficient to cause the extensive cerebral and retinal haemorrhages found in Kaleb, and ultimately his death.

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49 [2001] VSCA 220, [8] (emphasis added) ('*AHK*'), citing *R v McIntee* (1985) 38 SASR 432, 435 (King CJ). See also *Greensill v The Queen* (2012) 37 VR 257, 274–5 [71]–[72] (Redlich, Osborn and Priest JJA); [2012] VSCA 306; *Weng v The Queen* (2013) 279 FLR 119, 126–8 [29]–[32] (Osborn JA); [2013] VSCA 221.

50 Ibid. See *Bowden* (2017) 54 VR 135, 142–3 [33] (Priest JA) [2017] VSCA 46; *Ramlagun v The Queen* [2015] VSCA 337, [50] (Whelan JA, Santamaria JA agreeing at [70], Kaye JA agreeing at [71]).

157 The position of the Scandinavian witnesses on the triad injuries' lack of diagnostic utility is based on a 2016 study by the Swedish Agency for Health Technology Assessment and Assessment of Social Services (the 'SBU Report') that called into question the epistemological basis for the connection between the triad and SBS.[51]

158 It was not submitted by the applicant that the evidence challenging the epistemological[52] basis for the triad was not available or capable of being adduced at the time of his trial. The SBU Report, in particular, comfortably pre-dated the applicant's trial. However, the applicant submits that he was not in a position, even with reasonable diligence, to put that evidence before the jury. He submits, in any event, that the Scandinavian expert reports are of such quality and the expertise of the authors so impressive, that the new evidence strongly supports the contention that an innocent person has been convicted, or, at least, that there must exist a reasonable doubt about the applicant's guilt. Put another way, the applicant contends that whatever label is placed upon the Scandinavian evidence,[53] the evidence is sufficiently cogent to compel at least a reasonable doubt about his guilt.

159 Professor Eriksson gave evidence about the genesis and methodology of the SBU Report and its conclusion that there is no scientific support for the proposition that subdural haemorrhages, retinal haemorrhages and encephalopathy are evidence of traumatic shaking; Professors Wester and Högberg, subscribing to the methodology and conclusions of the SBU Report, advanced alternative causes for Kaleb's death based on organic factors related to his low birth weight and size, his sex and the fact of his rapidly increasing head circumference.

160 Professor Eriksson was an expert member of the project group that prepared the SBU Report. He is the Professor of Forensic Medicine at Umeå University in Sweden and senior consultant in forensic medicine on the Swedish National Board of Forensic Medicine. He is also an expert advisor in specified projects at the SBU, the National Board of Health and Welfare, the Swedish Civil Contingencies Agency and the Swedish Prosecution Authority. He was licenced to practice medicine in Sweden in 1977, obtained a PhD from Umeå University in 1979 and has been a professor in forensic medicine at Umeå University since 1992. In addition, he is currently the Chair of the Swedish Society for Forensic Medicine having previously occupied that position in the 1980s and then again between 2002 and 2009.

161 Professor Eriksson prepared a report dated 14 August 2020 and gave evidence in the appeal. In his evidence he outlined the reasons for and the findings of the SBU Report and its conclusion that there is insufficient scientific evidence on which to assess the diagnostic accuracy of the triad injuries in identifying traumatic shaking and limited scientific evidence that the triad injuries, and therefore its components, can be associated with traumatic shaking.

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51 Swedish Agency for Health Technology Assessment and Assessment of Social Services, *Traumatic Shaking: The Role of the Triad in Medical Investigations of Suspected Traumatic Shaking* — A Systematic Review' (Report No 255E, 2016).

52 Epistemology is the theory of knowledge especially with regard to its methods, validity and scope, and the distinction between justified belief and opinion.

53 That is, whether it is 'fresh' or 'new'.

162 Professor Wester is a retired neurosurgeon who practiced principally in Norway. He was a consultant in the Department of Neurosurgery, Haukeland University Hospital, Bergen between 1986 and 1990 and again between 2001 and 2010. He was the clinical department head at the same institution between 1990 and 2001. He was a professor of neurosurgery at the University of Bergen between 1989 and 2010 and has been a Professor Emeritus since then. He has also been a visiting professor of neurosurgery in Addis Ababa, Ethiopia. He was a founding member and subsequently President of the Scandinavian Neurotrauma Committee.

163 Professor Wester gave evidence that he has had extensive experience with general paediatric neurosurgery both in Norway and Ethiopia, and with hydrocephalus in children, both clinically and scientifically. He has contributed to national and international discussions on SBS and AHT. In 2019, he published an article raising the possibility that BESS could be misdiagnosed as AHT.[54]

164 Professor Wester prepared a report dated 13 August 2020 and gave evidence in the appeal over two days. Professor Wester's evidence was that Kaleb most likely died from complications from BESS.

165 Professor Högberg is a professor of obstetrics and gynaecology at Uppsala University and Umeå University in Sweden. He has had about 200 original scientific papers published in scientific journals in obstetrics, gynaecology, perinatal medicine, global health, public health and medical history. He is the principal investigator in a research project entitled 'Differential diagnostic study of skeletal injuries/findings and/or intercranial haematomas in infants: Abuse or complications from the foetal period, birth, or neonatal period?'.

166 Professor Högberg prepared a report dated 17 July 2020 and an undated addendum report prepared shortly before the hearing of the appeal, and gave evidence in the appeal. He posited that Kaleb died of organic causes related to intracranial haemorrhages, both subdural and subarachnoid, causing increased intracranial pressure and global cerebral ischemia.

167 Dr Tully, Dr Iles and Professor Ditchfield gave evidence in response to the evidence of Professors Eriksson, Wester and Högberg and prepared written reports in rebuttal that were tendered in the application for leave to appeal: Professor Ditchfield's rebuttal report is dated 17 September 2021; Dr Tully's rebuttal report is dated 28 September 2021; and Dr Iles' rebuttal report is dated 13 October 2021.

168 This rebuttal evidence, along with the evidence of the Scandinavian witnesses, is discussed in detail below.

(3) Background to the new evidence

169 By way of background, it is necessary to understand that BESS was raised and investigated by both the prosecution and the applicant as a potential factor in Kaleb's collapse and death prior to trial.

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54 Knut Wester, 'Two Infant Boys Misdiagnosed as "Shaken Baby" and Their Twin Sisters: A Cautionary Tale' (2019) 97 *Perspectives in Paediatric Neurology* 3.

170 Dr Tully entertained the possibility that Kaleb had BESS in her initial medical report of 8 February 2016, when considering findings from the MRI scan performed during Kaleb's first hospital admission on 15 January. The 15 January MRI scan revealed that he had prominent extra-axial spaces and mild ventricular dilation. Dr Tully concluded that while Kaleb might have had BESS, it did not explain his symptoms prior to admission or his clinical presentation on 23 January.

171 In August 2018, Professor Ditchfield was asked to provide an opinion in relation to the 15 January MRI and respond to the following questions: first, whether there was any radiological evidence of BESS; and, secondly, whether the subdural fluid identified in the MRI contained blood. Professor Ditchfield concluded that Kaleb's subarachnoid space was within the normal range for his age, albeit at 'the upper range of normal', and that the 15 January MRI did not demonstrate BESS. As to the second question, Professor Ditchfield described the subdural fluid as 'complex' and as most likely caused by haemorrhage (ie blood).

172 In her addendum report dated 29 August 2018, Dr Tully re-considered BESS in light of Professor Ditchfield's opinion, confirming that there was no radiological evidence of BESS in the 15 January MRI scan and that the subdural space identified on that scan was likely to be the result of prior subdural haemorrhages.

173 Prior to trial, the applicant obtained his own expert report on the cause of Kaleb's death from consulting forensic pathologist, Professor Johan Duflou.[55] Professor Duflou was asked to examine aspects of Kaleb's pre-existing medical condition and its possible impact on the cause of his death. He reviewed Kaleb's medical records, as well as the medical and autopsy reports prepared by Dr Tully and Dr Iles respectively. Among other matters, Professor Duflou recorded Dr Tully's opinion that:

> An alternative to subdural hygromas in this case is [BESS]. However, there is usually no vomiting, drowsiness or poor feeding, and the typical MRI features of BESS were not present in this case. Even if Kaleb had BESS, they would not explain his sudden collapse and widespread retinal haemorrhages, and there is no increased risk of subdural haemorrhage in BESS.

174 In his own discussion of the cause of Kaleb's death, Professor Duflou opined:

> CAUSE OF DEATH: I agree with Drs Tully and Iles that death is due to a head injury. There is in my opinion no plausible alternative cause of death in this case. In common with Dr Iles, I am of the view that pre-existing conditions present in this case may have resulted in Kaleb being more susceptible to sustaining the head injury than would be the case in a normal infant. I also agree with Dr Iles that it is very likely that the head injury seen in this case is in excess of what would be expected if there had been only 'normal handling' of this infant.

175 Professor Duflou considered the possibility that birth-related subdural haemorrhages were present and played a role in Kaleb's death and he considered the possibility of a rebleeding of subdural haemorrhages. In his discussion of the diagnosis of subdural

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55 Professor Duflou prepared reports dated 23 February 2017 and 15 August 2018 that were not ultimately relied upon by the applicant at trial.

hygromas made at the time of the 15 January MRI, Professor Duflou stated that the prominent extra-axial spaces and mild ventricular dilation were features consistent with typical observations in BESS, namely enlargement of the subarachnoid spaces (most evident in the frontal regions), prominence of the basilar cisterns and the anterior interhemispheric fissure, and mild ventriculomegaly. Professor Duflou continued:

> In my opinion, the MRI appearances of these lesions on 15 January 2015 [sic] are entirely consistent with a diagnosis of BESS. Further, the growth charts of Kaleb suggest progressive enlargement of the head over a prolonged period of time, with deviation from his original 15th percentile first identified on 22 October 2015, less than 3 weeks after birth, indicating that whatever the actual pathological process was which caused enlargement of the head had been present shortly after birth, and not a condition which only first developed in the weeks prior to death.

176 Professor Duflou therefore considered the possibility of BESS and/or the rebleeding of a subdural haemorrhage. He did so in the context of considering whether those conditions made Kaleb more susceptible to injury from 'rough handling'.

177 The applicant did not seek to call evidence from Professor Duflou at trial.

178 Dr Byron Collins, forensic pathologist, also produced a report for the applicant on Kaleb's death in March 2019. Dr Collins agreed in general terms with the cause of death as stated in Dr Iles' autopsy report and with Dr Iles' statement that whilst the force required to produce a subdural haemorrhage is, as yet, unknown it 'should be considered to be considerably beyond that associated with normal handling of an infant'. Dr Collins also observed as follows:

> Role of the chronic subdural haemorrhages is pivotal to the mechanism of causation of the acute subdural haemorrhages, in relation to the degree of force required.
>
> Acute/chronic subdural haemorrhages widespread and not space occupying.
>
> No overt evidence of pre-existing pathological conditions (apart from chronic subdural haemorrhages), which could have caused or contributed to the demise.
>
> The diagnosis of BESS is definitely within the bailiwick of the Radiologist and not the Pathologist, although I accept the comments contained in the report prepared by Dr Ditchfield.

179 Again, the applicant did not seek to call evidence from Dr Collins at trial.

180 Although neither Professor Duflou nor Dr Collins gave evidence at trial their reports were tendered in evidence (by the respondent) in this application.

(4) Scientific basis for 'the triad' as a diagnostic tool

(a) The SBU Report

181 The premise of the SBU Report is that in cases of suspected traumatic shaking, the diagnosis of traumatic shaking has conventionally been based on three findings, referred

to collectively as 'the triad': subdural haematoma (bleeding between the dura mater and the brain), retinal haemorrhages and various forms of brain symptoms (encephalopathy). In this context, its evaluation aimed to:

> [D]etermine how reliably the triad or its components can be explained by traumatic shaking of children up to one year of age.[56]

182 More specifically, the SBU Report articulated the question to be addressed as follows:

> With what certainty can it be claimed that the triad, subdural haematoma, retinal haemorrhages and encephalopathy, is attributable to isolated traumatic shaking (ie when no external signs of trauma are present)?[57]

183 The SBU report was authored by a project team comprised of seven Swedish paediatric experts, including Professor Eriksson.

184 While the SBU project team recognised that the term 'abusive head trauma' had been introduced, it decided to apply the term 'traumatic shaking'[58] to the trauma mechanism under investigation and the term 'triad' to the signs and symptoms, recognising that in the scientific literature, the collective noun 'triad' had been adopted for the most frequently occurring injuries (subdural haematoma, retinal haemorrhage and encephalopathy).[59]

185 The SBU project team did not carry out any empirical research. Instead, it conducted a systematic review of the scientific literature about the diagnosis of traumatic shaking in children under the age of 12 months. In other words, the project team evaluated the reliability of existing empirical studies that had been used to establish an association between traumatic shaking and the clinical features that make up the triad.

186 The SBU project team's search of the relevant scientific literature yielded 3,773 abstracts, of which 1,065 were retrieved in full text. Of these, 1,035 were excluded because they did not meet the inclusion criteria. Of the 30 remaining studies, two were assessed to be of moderate quality and none to be of high quality.[60]

187 This paring back of the available scientific literature to almost nothing flowed in large measure from the fact that the SBU project team decided to include in its review only studies of cases of traumatic shaking that were witnessed or in which the perpetrator had confessed to shaking the child. The project team excluded studies which included cases where there was external injury to the head and/or fractures or other injuries. Hence, studies that included cases in which other physical signs of abuse were reported in association with traumatic shaking, including bruising to the chest and fractures to

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56 SBU Report, 7.

57 Ibid 17.

58 'Traumatic shaking' is defined as occurring when a child is shaken in such a way that its head is flung backwards and forwards. Injuries are believed to occur because the head is subjected to acceleration–deceleration and rotational forces: ibid 9.

59 Ibid 10.

60 Ibid 21.

the ribs and shin bone(s), were not considered. The project team also excluded studies of fewer than 10 cases.[61]

188 Based on this analysis, the SBU review of the scientific literature produced the following graded results:

- There is insufficient scientific evidence on which to assess the diagnostic accuracy of the triad in identifying traumatic shaking (very low-quality evidence).
- There is limited scientific evidence that the triad and therefore, its components can be associated with traumatic shaking (low-quality evidence).[62]

189 The only two studies that the SBU project group accepted as being of moderate (acceptable) quality were based on cases in which the perpetrator confessed to subjecting the child to traumatic shaking. The first, by Vinchon et al,[63] was based on a register of traumatic head injury in children under two years of age who were admitted to hospital over a nine-year period in a relatively large catchment area. The Vinchon study looked at 412 cases, of which 124 were classified as inflicted head injury and 288 as accidental trauma. In the group with inflicted injury, there were 45 confessed cases, 30 involving traumatic shaking and 15 in which the perpetrator admitted to other external trauma. The group of 30 children subjected to traumatic shaking (inflicted trauma) was compared with 39 cases in which accidental trauma was witnessed in a public place.

190 In the group with inflicted trauma, 82 per cent had a subdural haematoma compared to 44 per cent in the accidental trauma group; 84 per cent had retinal haemorrhages compared to 17 per cent in the accidental trauma group; and 27 per cent had cerebral ischemia compared to 3 per cent in the group that suffered accidental head trauma.

191 The second study, by Adamsbaum et al,[64] compared 29 confessed cases of traumatic shaking to a group of 83 unconfessed cases. The criteria for inclusion in the study were the presence of subdural haematoma and confession by the suspected perpetrator. As subdural haematoma was one of the criteria for inclusion in the traumatic shaking group, only the results for retinal haemorrhages could be used by the SBU project team.[65] In the cases in which there was a confession of traumatic shaking, 83 per cent had retinal haemorrhages. However, the other group included children who had been shaken in an attempt at revival or who had suffered accidental injury, and some children for whom no explanation for the condition was given. The SBU project team considered that this group was not an acceptable reference group.[66]

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61 Ibid 18

62 Ibid 22.

63 Matthieu Vinchon et al, 'Confessed Abuse versus Witnessed Accidents in Infants: Comparison of Clinical, Radiological, and Ophthalmological Data in Corroborated Cases' (2010) 26(5) *Child's Nervous System* 637.

64 Catherine Adamsbaum et al, 'Abusive Head Trauma: Judicial Admissions Highlight Violent and Repetitive Shaking' (2010) 126(3) *Pediatrics* 546.

65 SBU Report 23.

66 Ibid.

192 Nonetheless, the SBU Report accepted that the Vinchon and Adamsbaum studies both demonstrated that traumatic shaking could cause subdural haematoma and retinal haemorrhages. According to the SBU Report, although both studies had methodological limitations and were only of moderate quality, they supported the hypothesis that isolated traumatic shaking could give rise to the triad injuries.[67]

193 The significance of the SBU Report lies in its epistemological analysis. According to the SBU project group, its review of the scientific evidence disclosed a number of methodological issues in the published studies. The critical methodological shortcoming was described as 'circular reasoning', which was said to arise, in particular, from the role the child protection team plays in the investigation of cases of suspected traumatic shaking. Over the years, these teams have developed criteria based on certain symptoms and signs, some of which are associated with the carer's credibility. This clinical rather than scientific approach means that the criteria used are not tested in systematic studies of the association between the triad and traumatic shaking. The untested criteria applied by the child protection team infect the scientific investigation and hypothesis testing, which, in turn, reinforces rather than tests the conventional approach to diagnosing SBS/AHT.[68]

194 Put more simply, the impugned 'circular reasoning' process has been described by reference to a series of steps as follows:

(a) The determinations of SBS/AHT by clinical teams are based on the assumption that if there is no acceptable explanation for the triad injuries, the infant must have been shaken violently;

(b) The determinations of the clinical teams are subsequently used by researchers when classifying SBS/AHT cases in scientific studies;

(c) The outcome of the scientific studies becomes statistically very strong and supports the assumptions of the clinical teams when determining SBS/AHT cases.[69]

(d) Thus the assumption that a baby has been violently shaken is imported into the conclusion that is to the same effect.

195 This circular reasoning, said to be endemic to the field of study, is given as the reason why the SBU project group accepted as study cases only those where shaking was witnessed or confessed to.

196 The SBU Report is radical in its approach and conclusions. It seeks to set aside decades of study on the consequences of AHT and the wide-spread acceptance that AHT may

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67 Ibid 27.

68 According to the SBU Report, if it has already been assumed that the question to be addressed by the study has been answered, ie the association between the symptoms/signs of the triad and traumatic shaking has already been described, then circular reasoning occurs. This results in a high risk of bias, which means the researcher does not know what is being compared. In order to avoid circular reasoning, study cases and control cases must be identified incontrovertibly: ibid 30.

69 Niels Lynøe et al, 'Easier to See the Speck in Your Peers' Eyes than the Log in Your Own? Response to Debelle et al' (2018) 103(7) *Archives of Disease in Childhood* 714.

cause the constellation of clinical features known as 'the triad'. It does so by excluding nearly all of the available learning. In lieu, it signposts as the task for future research the identification of organic causes for the thousands of cases of infant death and disability hitherto attributed to AHT and for future incidents that would otherwise be 'misdiagnosed' as involving head trauma. Thus, under the heading 'Issues for future research', the SBU Report proposed the investigation of alternative causes for presence of the triad injuries in infants along the following lines:

> There is a lack of detailed knowledge about the pathophysiology of the development of subdural and retinal haemorrhages associated with vaginal delivery. Although most bleedings related to delivery are symptomless and disappear (are resorbed) within a few months, occasionally a haemorrhage can degenerate into a hygroma. This circumscribed collection of fluid is contained by a membrane in which small vessels form and it is considered that this in turn can lead to renewed bleeding (rebleeding) and a chronic subdural pool of fluid. The possibility cannot be discounted that in certain cases, rebleeding can cause symptoms. This could be one reason why a child suddenly exhibits signs of encephalopathy (lethargy, apnoea and/or seizures), causing a carer to seek medical attention. Hypothetically such rebleeding could occur spontaneously or in response to minor trauma. There is therefore an urgent need for research into the pathophysiology and the natural course of subdural and retinal haemorrhages.[70]

197 As is discussed in detail below, Professors Wester and Högberg have pursued this task and have re-interpreted the evidence supporting a small number of diagnoses of AHT in Sweden and Norway as evidence of a disease process. This is, in substance, what they have sought to do in their evidence about the cause of Kaleb's death.

(i) Criticisms of the SBU Report

198 The SBU Report has been the subject of quite damning criticism in the 'mainstream' medical community. The respondent referred the Court to a critique published in 2018 on behalf of the Royal College of Paediatrics and Child Health in the United Kingdom (the 'Royal College paper').[71] The Court was also referred for a more general expression of support for the triad as a diagnostic tool to the 'Consensus Statement on Abusive Head Trauma in Infants and Young Children' (the 'Consensus Statement').[72]

199 The Royal College paper directly challenges the methodology and conclusions of the SBU Report. It describes the SBU Report as misleading and dangerous, and calls for its withdrawal.[73] In summary, it criticises the SBU Report for posing a 'clinically irrelevant' question, conducting an inadequate literature search, using a poorly designed research format, applying no standards for confirmation of the key clinical features and using a risk of bias assessment that relies solely on case ascertainment and confirmation

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70 SBU Report, 34 (citations omitted).

71 Geoffrey Debelle et al, 'Abusive Head Trauma and the Triad: A Critique on Behalf of RCPCH of "Traumatic Shaking: The Role of the Triad in Medical Investigations of Suspected Traumatic Shaking"' (2018) 103(6) *Archives of Diseases in Childhood* 606.

72 Arabinda Choudhary et al, 'Consensus Statement on Abusive Head Trauma in Infants and Young Children' (2018) 48(8) *Pediatric Radiology* 1048.

73 Royal College paper, 609.

of shaking. It also identifies methodological limitations relating to the ophthalmological component of the review.[74]

200 The Royal College paper describes the fact that the SBU Report excluded case studies where there was external injury to the head and/or fractures and other injuries as 'an extraordinary omission' that demonstrates an ignorance of the mechanics of 'isolated traumatic shaking'.[75] According to the Royal College paper, a comprehensive review aiming to identify all relevant high-quality studies would not have excluded studies where other injuries were present, as it is well established that shaking has the potential for metaphyseal fractures of the long bones, and forceful gripping of the infant chest may result in bruising and/or rib fractures. Furthermore, this exclusion criterion was not applied with rigour as both studies that were included (Vinchon and Adamsbaum) clearly describe cases with associated injuries.[76]

201 More fundamentally, the Royal College paper questions the premise of the SBU Report that there exists a 'healthcare principle' that the triad injuries are attributable exclusively to traumatic shaking. According to the Royal College paper, and consistently with what the Court was told by Drs Tully and Iles, there is no such 'healthcare principle': the diagnosis of AHT is never made exclusively on the basis of a triad of clinical features.[77] In the same vein, the paper criticises the SBU Report for investigating 'isolated traumatic shaking' (as opposed to AHT), as the term is not used in published studies and it is neither identified nor diagnosed clinically. Likewise, while 'the triad' is the index test in the SBU Report's review of the scientific literature, it is not used as a diagnostic test in clinical practice and it was therefore unlikely that the SBU review would identify a study that set out to evaluate 'the triad' as a diagnostic test.[78]

202 The Royal College paper criticises the SBU Report for failing to describe how the presence or absence of the triad was identified in any retrieved paper. Moreover, it says, there was no interrogation of individual aspects of the triad and, in particular, no clear definition of encephalopathy, which is a clinical condition that presents with a broad range of symptoms. In the absence of any search terms for the clinical signs of encephalopathy or a categorical definition of the condition as applied within the triad, it is impossible to know, for example, whether, if the infant's neurological status is simply described as 'drowsy' in the presence of retinal and subdural haemorrhages, the study would be included in the SBU review.[79]

203 As for the SBU Report's reference test of 'admitted or witnessed traumatic shaking or other trauma', the Royal College paper argues that while there is no 'gold standard'

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74 Ibid.

75 Ibid 608.

76 Ibid.

77 Ibid 607. The Royal College paper says that in clinical practice, the decision regarding the likelihood of abusive head trauma is made after 'a rigorous assessment of the history, examination, assessment of comprehensive clinical investigation findings, in the context of a forensic assessment of the proposed mechanism of injury and family risk factors'. Moreover, during clinical investigations, the clinician considers a differential diagnoses of all potential causes for the presenting symptoms and signs. The purpose is ultimately to determine whether an infant with no independent mobility, who is entirely dependent on their carer and unable to offer a history, needs protection from future harm: ibid.

78 Ibid.

79 Ibid.

diagnostic test for AHT, setting the threshold for inclusion at the level of admitted or witnessed shaking is unrealistic, as this level of certainty is infrequently recorded in the real world setting. It says that the SBU Report introduced this new standard without justifying or reporting it, and failed to describe what would constitute a valid admission or video evidence. Furthermore, the reference standard does not define when the condition is absent, yet implies that in the absence of 'admitted or witnessed traumatic shaking or video documentation', shaking has not occurred.[80]

204 The Royal College paper concludes:

> Crucial errors were made in the setting of the question, search strategy, lack of standardised definitions for terms used, inadequate inclusion/exclusion criteria (including incorrect study design choices), critical appraisal tools and synthesis of included studies … Contrary to the proposal that the scientific community involved in the care of infants with intracranial injury, some of whom have been abused, are not open to challenging established ideas, any search of the scientific literature will identify an increasing body of high-quality scientific studies that sets out to explore new ways to delineate characteristics that improve the identification and understanding of AHT.[81]

205 The Consensus Statement is a policy document, not a medical or scientific study, although it makes reference to a number of such studies.[82] It is expressed to build on consensus statements from 15 national and international professional medical societies and organisations confirming the validity of the AHT diagnosis[83] and purports to address 'significant misconceptions and misrepresentations about the diagnosis of [AHT] in infants and young children', its stated aim being to expose 'the fallacy of simplifying the diagnostic process to a "triad of findings" [which is] a legal argument and not a medically valid term'.[84]

206 The Consensus Statement describes AHT as 'a scientifically non-controversial medical diagnosis broadly recognised and managed throughout the world. When diagnosed, it signifies that accidental and disease processes cannot plausibly explain the aetiology of the infant-child's injuries'.[85]

207 According to the Consensus Statement, no single injury is diagnostic of AHT. Rather, a multiplicity of findings provide clues to diagnosis, including evidence of intracranial and spinal involvement, complex retinal haemorrhages, rib and other fractures inconsistent with the provided mechanism of trauma, as well as the severity and age of

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80 Ibid.

81 Ibid 609 (citations omitted).

82 It records that professional medical societies use consensus statements to communicate general physician acceptance on a particular topic. Consensus statements are vetted by the membership and designed to help physicians, news media and the public distinguish accurate medical information from non-evidence-based or 'court room only' causation theories: Consensus Statement, 3.

83 The Consensus Statement is supported by the Society for Pediatric Radiology, the European Society of Paediatric Radiology, the American Society of Pediatric Neuroradiology, the American Academy of Pediatrics, the European Society of Neuroradiology, the American Professional Society on the Abuse of Children, the Swedish Paediatric Society, the Norwegian Pediatric Association and the Japanese Pediatric Society.

84 Consensus Statement, 2.

85 Ibid.

the findings. Subdural haematoma is the most frequently identified intracranial lesion, but brain parenchymal injury is the most significant cause of morbidity and mortality. While there is a high incidence of ligamentous cervical spine injury among victims of inflicted injury, the absence of such injury does not exclude AHT. In suspected cases of AHT, alternative diagnoses must be considered and, when appropriate, explored. The question to be answered is, 'Is there a medical cause to explain all the findings or did this child suffer from inflicted injury?'[86]

208 As to the status of any controversy about the use of the triad injuries as a diagnostic tool, the Consensus Statement reports that 'denialism of child abuse has become a significant medical, legal and public health problem'. It refers specifically to 'courtrooms in the United States', where it says 'defence attorneys and the medical witnesses who testify for them have been disseminating inaccurate and dangerous messages that are often repeated by the news media'.[87] According to the Consensus Statement, efforts to create doubt about AHT include the deliberate mischaracterisation and replacement of the complex and multifaceted diagnostic process by a near mechanical determination based on 'the triad'. This (bogus) critique has been sensationalised in the mass media in an attempt to create the appearance of a 'medical controversy' where there is none. The Consensus Statement describes the triad argument as a 'straw man' that ignores the fact that AHT diagnosis typically is made only after careful consideration by a multidisciplinary team of all historical, clinical and laboratory findings, as well as radiologic investigations.[88]

209 The Consensus Statement does not address the methodology and conclusions of the SBU Report.

210 The publication of each of the SBU Report, the Royal College paper and the Consensus Statement pre-dated the applicant's trial and all were readily available at that time.

(ii) Professor Eriksson's evidence

211 Professor Eriksson was asked to address four questions centred on the conclusions of the SBU Report, commencing with a request that he cite any documents or studies in which he was personally involved that examined the scientific and medical basis for SBS or AHT and whether, in his opinion, a medical controversy exists in the diagnosis of these matters.

212 In answer to these questions, Professor Eriksson outlined and defended the methodology and conclusions in the SBU Report. He was also asked his opinion about the Consensus Statement (which was incorrectly put forward by the applicant as being responsive to the SBU Report). Finally, he was asked to comment on the nature of the evidence given by Dr Tully and Dr Iles at trial by reference to the SBU Report.

213 Professor Eriksson described the central thesis of the SBU Report to be that, as a consequence of there being insufficient scientific evidence on which to assess the diagnostic accuracy of the triad injuries in identifying traumatic shaking and the limited

86 Ibid.
87 Ibid 4.
88 Ibid.

or low scientific evidence that the triad (or its components) could be associated with traumatic shaking, it is not possible to estimate the incidence or prevalence of SBS/AHT. He explained that the classification of SBS/AHT cases is based on a value-impregnated choice of theory that the baby was shaken, and these values-based decisions, made by child protection teams, are obscured because they are presented as being based on purely scientific reasoning. However, they are supported by the increased societal sensitivity to child abuse and, in this manner, the 'SBS scientific enterprise' has become ideological and has facilitated the continued use of circular reasoning, resulting in the over-estimation of the prevalence of SBS/AHT cases. Professor Eriksson continued:

> The strongly biased research methodology resulting in the concept that the triad means that the child must have been shaken violently (after other known causes are excluded) has been self-reinforced and repeated in a plethora of publications, in clinical guidelines.

214 Professor Eriksson was also critical of the reliability of confession evidence. He identified a number of circumstances in which confession evidence might be unreliable, concluding that this meant that the confession-based studies were quite low-quality evidence overall. However, these were the only studies not classified as having a 'high risk of bias'.

215 In the context of discussing whether there is a medical controversy surrounding the diagnosis of SBS/AHT, Professor Eriksson dismissed the argument that 'the whole picture' is considered in the diagnosis of SBS/AHT, not just the triad findings. According to Professor Eriksson, in case after case it is clear that as soon as the triad injuries are detected, the clinical doctors readily exhibit a strong suspicion that the infant must have been violently shaken. Proponents of SBS/AHT point to the use of differential diagnoses, yet the assumption is that shaking is the default mechanism, and that shaking must have occurred if no other explanation is found.

216 Professor Eriksson accepted that the proponents of the SBU Report are in the minority, but suggested that the collective of concerned paediatricians and other doctors who have endorsed SBS/AHT as a diagnosis and rejected the SBU Report can be explained either by 'group-think,' the 'philosophy of scientific paradigms', or by individual characteristics and traits (by which he meant conformity and a lack of moral courage and/or career investment). He suggested that the criticisms of the SBU Report were more emotional than scientific and motivated by a concern that to admit there is controversy would cause the courts to conclude that there is reasonable doubt in criminal cases, and they could no longer continue with the diagnostic procedures they have applied for 50 years.

217 Unsurprisingly, Professor Eriksson was unimpressed by the Consensus Statement, describing it as the product of a 'limited and narrow field of people known to comply with the traditional SBS dogma [who] were invited to participate in its publication' and as 'merely a policy statement of people who are already in agreement'. According to Professor Eriksson, the methods used in the Consensus Statement to reach the conclusions are 'flawed and substandard', as there is no information about whether the literature search is systematic or non-systematic, about inclusion and exclusion criteria, or about how results have been synthesised. Moreover, there is no assessment of

scientific value by type of study, there is no unified structure and no summarised assessment of the strength of the scientific evidence is presented, and literature which does not support the conclusions of the authors is dismissed in broad brush and condemnatory terms. The Consensus Statement is not, according to Professor Eriksson, a credible systematic literature review or an 'authentic' consensus report.

218 218 Insofar as it might be suggested that the Consensus Statement purports to be a systematic literature review (credible or otherwise), plainly it does not. It is not a scientific study, but a document directed to educating the public about AHT diagnosis more generally. However, it does show wide-spread acceptance by the medical community of the use of the triad injuries in the diagnosis of AHT.

(iii) Evidence of Professor Högberg and Professor Wester on 'the triad'

219 Professor Högberg strongly endorsed the SBU Report's conclusions. He agreed that the SBU Report had been criticised, and that the Royal College and other experts had called for its retraction. However, he disagreed that the retraction was sought because serious flaws had been identified in the SBU Report.

220 According to Professor Högberg:

> The evidence-based knowledge indicates that there is weak scientific support for [subdural haematoma] resulting from traumatic shaking alone, and that there is insufficient scientific support for assessing the diagnostic accuracy for identifying shaking based on [subdural haematoma], retinal haemorrhage, and encephalopathy.
>
> Based on current knowledge, it cannot be claimed that Kaleb's symptomatic subdural hygromas with increased intracranial pressure, as seen on 14.01.16, and his respiratory arrest on 23.01.16, due to subdural and subarachnoid haemorrhages with increased intracranial pressure, encephalopathy, and retinal haemorrhages, were caused by SBS/AHT.

221 Professor Högberg gave as an example of the 'constitutional crisis' in the SBS/AHT paradigm the difference in the proportion of abuse diagnoses involving subdural haemorrhage between different countries:

> Sweden has a lower incidence of [subdural haematoma] among infants than Great Britain and Ireland, 16.5 cases per 100,000 and 24.5 cases per 100,000, respectively. The proportions of [subdural haematomas] with an abuse diagnosis were 2.3 and 14.3, respectively, corresponding to a six-fold difference. Every seventh Swedish infant with [a subdural haematoma] was diagnosed as having suffered abuse, while one in two infants with [a subdural haematoma] in Great Britain and Ireland was assessed as having a non-accidental head injury. One explanation for this large discrepancy is likely connected to the differing support for various theories on the origins of [subdural haematomas] among clinically active physicians in different countries.[89]

[89] Citations omitted.

222 For his part, Professor Wester explained that one of the underlying premises of his evidence was that in spite of more than 1,000 articles having been published on SBS/AHT, there is no solid scientific evidence that allows use of the triad or its components as 'solid proof' of violent shaking. This also applies to other medical findings that have been viewed as typical of SBS/AHT such as rib fractures.

223 Professor Wester is the author of an article published in 2019 entitled, 'Two Infant Boys Misdiagnosed as "Shaken Baby" and Their Twin Sisters: A Cautionary Tale'[90] (the 'twins paper'), which describes evidence that he gave in two appeals against convictions for baby-shaking, resulting in the acquittal of the accused in each case. As he did in Kaleb's case, Professor Wester diagnosed a pre-existing organic cause for the collapse of the babies, being a form of external hydrocephalus. In this context, he said:

> My concern in this report is that many infants appear to be diagnosed as shaken babies without any clear signs of an inflicted trauma (impact). The scientifically weak documentation of a causal relationship between the triad and the criminal act of violent shaking without impact cannot be used as judicial evidence for child abuse, *beyond reasonable doubt*.
>
> When suspecting the shaken baby syndrome or abusive head trauma, external hydrocephalus should always be ruled out as a possible alternative diagnosis, as this condition may have medical findings compatible with those claimed to prove violent shaking. Unfortunately, many physicians who deal with child abuse seem unfamiliar with the manifestations of benign external hydrocephalus in children.[91]

224 More specifically, Professor Wester noted that subdural haematomas occur in newborns, especially in pre-term deliveries and twins, and that these subdural blood collections may gradually develop into larger haematomas over time which may bleed easily. He also noted a male preponderance in cases of AHT, external hydrocephalus, and subdural haematoma. As external hydrocephalus predisposes for subdural haematoma and as there are so many striking epidemiologic similarities, Professor Wester questioned whether some infants who have been characterised as victims of shaking simply suffered from BESS.[92] As for retinal haemorrhages, Professor Wester states that 'they may not be pathognomonic for abusive head traumas; they can also be seen in infants not related to abuse, eg, in a large number of healthy newborns, in infants with "macrocephaly", after "high-risk" deliveries, following acute life-threatening events, and after cardiopulmonary resuscitation.'[93] As for the other elements of the triad, 'there is no high-quality evidence that proves a causal relationship between retinal bleedings and violent shaking. The problem lies again in the lack of objective proofs of shaking.'[94]

225 As will be seen, much of the analysis and discussion in the twins paper is replicated in Professor Wester's expert report on Kaleb.

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90 Knut Wester, 'Two Infant Boys Misdiagnosed as "Shaken Baby" and Their Twin Sisters: A Cautionary Tale' (2019) 97 97 *Perspectives in Paediatric Neurology* 3.

91 Ibid 10 (emphasis in original).

92 Ibid 9.

93 Ibid 9–10 (citations omitted).

94 Ibid.

(b) Evidence of Dr Tully and Dr Iles on 'the triad'

226 Dr Tully gave evidence that the terms 'the triad' and 'Shaken Baby Syndrome' are outdated and not in widespread use in clinical practice.[95] The term 'the triad' should not be used to infer causation as a result of AHT. Rather, it is merely a descriptive term for a constellation of findings, each element of which can occur in differing patterns and have a number of possible causes. This constellation of findings is the position from which the evaluation of an infant with these symptoms begins rather than, as Professor Eriksson suggests, from the assumption that the presence of the triad injuries indicates forceful shaking as their cause. According to Dr Tully, this is the false premise upon which the Scandinavian witnesses all appear to have based their criticisms of the diagnostic process.

227 Dr Tully agreed with the Scandinavian witnesses that the presence of the triad injuries alone should not be used to conclude that an infant has been subject to forceful shaking. She stated that the medical assessment of an infant in respect of whom AHT is suspected requires a thorough and comprehensive evaluation that includes information gained from protective services and social work evaluation. The diagnosis is made on the basis of clinical, pathological and radiological findings, in the context of the infant's medical history, the history provided by the parents or carers and information provided by other professionals' assessments. Thus, a medical diagnosis following forensic assessment of an infant is based on the following: first, a detailed history (including past medical and birth history, family history, and developmental history); secondly, a thorough examination that looks for signs of rare medical diseases and additional signs of trauma; and, thirdly, a comprehensive panel of pathology and radiology tests. Dr Tully stressed that rigorous consideration of alternative causes must be undertaken and any realistic possibility of an alternative cause must be acknowledged. In short:

> A complex inferential and deductive reasoning process is undertaken that allows a diagnosis to be made that best explains the entire medical picture.

228 Dr Tully confirmed that the SBU Report has been widely criticised internationally since its publication. Nonetheless, she pointed out that its results clearly demonstrate that shaking can be a cause of the triad injuries. In brief, Dr Tully outlined and adopted the criticisms in the Royal College paper and concluded:

> The SBU Report does not contribute anything further to what is already known within clinical practice. It is my firm opinion that the conclusions of the SBU Report should not be used to cast doubt on the medical diagnosis of AHT. This is particularly so in a comprehensively investigated infant such as Kaleb who presented with severe brain swelling as well as subdural and retinal haemorrhages of a nature, extent and specific characteristic pattern well recognised in a large body of peer reviewed and published evidence to be

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95 Dr Tully reports that the term 'Shaken Baby Syndrome' is not used in Australia and that 'abusive head trauma' is currently considered the most appropriate and inclusive diagnostic term to use. AHT as a medical diagnostic term is an umbrella term that reflects the literature and clinical experience when the constellation of findings is thought to be caused by trauma as a result of a variety of possible mechanisms, including blunt impact and mechanisms that generate acceleration-deceleration and rotational forces such as shaking alone or shaking with impact. Medical professionals do not diagnose shaking, they diagnose AHT.

associated with AHT.

229 Dr Tully disagreed that the criticisms of the SBU Report had arisen largely out of the inability of the 'child abuse community' to accept a study with conclusions that are at odds with current consensus. She stated that the widespread criticisms of the SBU Report are based on its flawed methodology rather than a dislike of its conclusions.

230 Having said that, Dr Tully agreed with the Scandinavian witnesses that circular reasoning, which arises when features used to assess outcomes are also used to categorise subjects, needs to be considered when evaluating the evidence base surrounding AHT. According to Dr Tully, there is a body of literature that seeks specifically to address the problem of circular reasoning by ensuring that AHT diagnostic criteria exclude the features of interest such as retinal haemorrhages.[96] Additionally, there is a body of evidence, from a variety of disciplines, that examines subdural haemorrhages, retinal haemorrhages and encephalopathy in groups of children in whom abuse is not suspected. This body of evidence is not subject to circular reasoning and provides important information, for example, about patterns of retinal haemorrhages in children with conditions such as raised intracranial pressure or hypoxia or following accidental trauma. Given the inability to perform prospective randomised clinical trials regarding AHT, as discussed above, clinicians must rely on the critical evaluation of a large body of well-performed retrospective epidemiological studies as well as advances in clinical, pathophysiological and biomechanical knowledge and understanding informed by animal studies, computational modelling, improved biomechanical modelling and advances in radiological techniques.

231 As to the relevance of confessional evidence, Dr Tully opined that the information obtained from studies that directly analyse confessions from individuals who have made statements about harming their infants and children is important and it clearly indicates that shaking can cause subdural haemorrhages, retinal haemorrhages and encephalopathy. She referred to a systematic review of the role that confession evidence plays in the diagnosis of AHT published in June 2020 which involved 434 confessions across four continents.[97] The results of this review, according to Dr Tully, provide compelling support for the utility of confession evidence in understanding the mechanisms of AHT and strongly refute the argument that there are insufficient data within the published literature to support shaking as a mechanism for AHT.

232 As to whether, as Professor Eriksson proposed, there is an 'ongoing multinational controversy' surrounding the diagnosis of AHT, Dr Tully acknowledged the existence of a 'perceived controversy', propagated in the media, in the legal forum and by a small sector of the scientific community, as to whether 'the triad' indicates, and 'isolated shaking' is a mechanism for, traumatic head injury in infants and young children.

233 Dr Tully told the Court that she 'fully' acknowledges that there is 'unsettled science' in relation to thresholds and the magnitude of force required to cause the triad injuries. However she agrees with the conclusions in the Consensus Statement and does not

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96 See generally Sandeep Narang, 'A *Daubert* Analysis of Abusive Head Trauma/Shaken Baby Syndrome' (2011) 11(3) *Houston Journal of Health Law and Policy* 505

97 George A Edwards et al, 'What Do Confessions Reveal about Abusive Head Trauma? A Systematic Review' (2020) 29(3) *Child Abuse Review* 253.

accept there is a valid controversy about the medical diagnosis of AHT when the proper diagnostic process, as outlined above, is followed.

234 For her part, Dr Iles acknowledged the limitations arising from the unavailability of randomised controlled trials analysing the effects of shaking in infants and the absence of accurate biofidelic systems modelling all aspects of the human infant brain. As a result, the evidence-base for pure shaking events causing subdural haemorrhages, retinal haemorrhages and encephalopathy is necessarily indirect and weaker than the evidence of other types of inflicted injury. It relies on exclusion of other causes for the elements of the triad injuries, clinical observations in infants that have unequivocal evidence of traumatic injuries beyond the triad, and perpetrator confessions. She stated, however:

> What is certain is that [subdural haemorrhages], [retinal haemorrhages] and encephalopathy/hypoxic ischemic encephalopathy *are* manifestations of traumatic head injury in infants. There is abundant evidence for this in case series literature, and in criminal law in infants who have this 'triad' of findings in the setting of unequivocal injury outside of the central nervous system.[98]

235 Dr Iles agreed that there are causes of all or some of the triad injuries other than traumatic head injury (accidental or inflicted). The conclusion that an infant has sustained a traumatic head injury is not made on the basis of the presence of 'the triad' and there is no 'basic health care principle that the triad is attributable exclusively to traumatic shaking'.[99] Like Dr Tully, she told the Court that a diagnosis of AHT is only made following an extensive medical multidisciplinary diagnostic process that includes history, examination findings, and radiological and laboratory investigations, with consideration given to excluding conditions known to produce one or more of the triad injuries. Excluding alternative courses that may be realistically applicable in a particular infant is a critical component of the diagnostic process.

236 In Dr Iles' view, framing the analysis of inflicted head injury in infants as a binary, non-qualitative process is misleading and incorrect.[100] When an infant presents with severe encephalopathy, subdural haemorrhages and retinal haemorrhages (with or without other features of head injury), accidental trauma, inflicted trauma and non-traumatic medical/congenital causes must be considered. According to Dr Iles, attributing the diagnosis of inflicted trauma simply to the identification of 'the triad' misrepresents the complex and multifaceted diagnostic process undertaken, which encompasses historical, radiological and laboratory studies assessing intracranial, ocular and extracranial pathology to determine the presence or absence of natural disease processes that might also account for the medical findings, or might be considered a vulnerability in the setting of possible inflicted trauma.

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98 Emphasis in original.

99 Quoting SBU Report, 9.

100 According to Dr Iles, the assessment of head injury in infants may be complex in order to exclude presentations that may be due to natural disease processes. The differential diagnosis of subdural haemorrhages in infants and children includes birth and accidental trauma, metabolic and genetic diseases, haematological and clotting disorders, oncological and autoimmune diseases, vascular anomalies, congenital malformations, and others.

237 Dr Iles acknowledged that the SBU Report highlights important issues in the medico–legal literature, the most important being an awareness of circularity when both conducting and interpreting studies, and an awareness of the limitations of confession studies. However, Dr Iles criticised the SBU Report for failing to assess the nature/extent of characteristics of the elements of the triad. In particular, retinal haemorrhages are not a binary diagnostic feature and the unqualified use of the generic term 'retinal haemorrhages' is misleading and inappropriate.

238 Dr Iles also opined that, given the potential for confession evidence to be unreliable, the SBU Report's deliberate exclusion of cases that include evidence of other injuries is illogical, and it was unfortunate that the SBU project group rejected papers describing children with injuries at multiple sites because they did not conform to the study design.

239 Dr Iles also pointed out that while the SBU Report discounted studies of confessions of AHT that contained fewer than 10 cases, it included single case reports in its list of alternative causes of the triad injuries. In other words, it did not apply the same scrutiny to the list of alternative causes for the triad injuries.

240 Finally, Dr Iles emphasised the fact that the SBU Report *did* find evidence that the triad elements are associated with traumatic shaking. This finding, taken together with a number of the remaining 28 studies deemed to have a 'high risk of bias' which showed an association between the triad injuries and traumatic shaking, supports the conclusion that there is sufficient evidence that the triad injuries are associated with traumatic shaking.

(c) Studies supporting/applying the SBU paradigm

241 Following the publication of the SBU Report, Professors Wester and Högberg, persuaded that there was little or no scientific support for the association between the triad injuries and SBS/AHT, embarked on the suggested project of investigating organic causes for the clinical features of the triad. Together, and with others, they have carried out a series of retrospective studies of infants investigated for child abuse in Sweden and Norway and re-analysed the medical information available for these infants to identify disease processes. The starting point for these studies, in accordance with the conclusions in the SBU Report, is that the presence of subdural haemorrhages, retinal haemorrhages and/or encephalopathy in the infants in question did not indicate AHT. AHT is effectively eliminated from the (narrow) range of possible causes for the clinical features recorded.

242 The principal investigations in question, which post-date the applicant's trial, resulted in the following publications which we have read in considering the possible impact of the new evidence on the applicant's trial:

(a) Ingemar Thiblin, Knut Wester, Ulf Högberg et al, 'Medical Findings and Symptoms in Infants Exposed to Witnessed or Admitted Abusive Shaking: A Nationwide Registry Study' ('Thiblin 2020');[101]

[101] (2020) *PLoSOne* e0240182:1–14.

(b) Ingemar Thiblin, Knut Wester, Ulf Högberg et al, 'Retinal Haemorrhage in Infants Investigated for Suspected Maltreatment Is Strongly Correlated with Intracranial Pathology' ('Thiblin 2021');[102] and

(c) Knut Wester et al, 'Re-Evaluation of Medical Findings in Alleged Shaken Baby Syndrome and Abusive Head Trauma in Norwegian Courts Fails to Support Abuse Diagnoses' ('Wester 2021').[103]

(i) Thiblin 2020

243 Both Thiblin papers (2020 and 2021) reviewed cases of children identified in the Swedish National Patient Registry who, between 1997 and 2014, were investigated for suspected maltreatment. Thiblin 2020 identified 337 cases for possible review, but only a small number met the study's inclusion criteria which, like the SBU Report, required witnessed or admitted abuse by shaking.

244 Of the 337 cases, 36 cases were identified that satisfied the inclusion criterion of witnessed or admitted physical abuse by shaking. The study examined the presence of subdural haemorrhage, retinal haemorrhage, rib fractures and classical metaphyseal lesions in the 36 infants that the authors accepted were shaken. The results were as follows:

> For 30 infants, no findings or symptoms were reported. The shaking was described as forceful/hysterical in 13 of these cases (12 examined with neuroimaging and fundoscopy); one was filmed, and three were observed by nonrelated witnesses. None of the 27 infants who underwent a full-body x-ray had rib fractures or [classical metaphyseal lesions]. Thus, no infant subjected to shaking with or without blunt force trauma and without any possibly predisposing factors had any of the findings regarded as highly specific for AHT.[104]

245 These findings were discussed as follows:

> In contrast to earlier studies in which shaking or combined shaking/blunt trauma was wholly or partly inferred from [subdual haemorrhages], [retinal haemorrhages], seizures, apnoea, and long bone fractures, we found no strong association between such findings and shaking with or without blunt force impact. None of the infants with reported isolated shaking had any of these findings.[105]

246 The authors explained:

> Many studies have yielded extremely high specificity and positive predictive values of [subdural haemorrhages] and [retinal haemorrhages] for AHT. However, these values were based on circular reasoning and other methodological flaws [referring to the SBU Report]. *The study design of the current study does not allow for conclusions regarding the specificity of medical*

102 (2022) 111(4) *Acta Paediatrica* 800.
103 (2022) 111(4) *Acta Paediatrica* 779.
104 Thiblin 2020, 8–9.
105 Ibid 8–9.

findings for AHT.[106]

247 And:

> To our knowledge, this is the first study assessing the association of [subdural haemorrhages] and [retinal haemorrhages] from prior witnessed or admitted physical abuse by shaking. … The results of the present study do not support the notion that certain medical findings are highly suggestive of shaking to the exclusion of other possible causes. The results are in agreement with those obtained by the SBU report concluding 'There is limited scientific evidence that the triad and therefore its components can be associated with traumatic shaking (low quality evidence).'[107]

248 In respect of the 36 'shaking' cases identified, Thiblin 2020 concluded:

> No infant subjected to shaking with or without blunt force trauma and without any possibly predisposing factors had any of the findings regarded as highly specific for AHT. Our findings imply that [subdural haemorrhages] or [retinal haemorrhages] have low sensitivity for AHT, entailing a risk for false negatives if these features are believed to have a high negative predictive value. The results also indicate that isolated shaking may cause intracranial haemorrhage with or without [retinal haemorrhages] in vulnerable infants.[108]

249 The conclusion that the results of the present study 'do not support the notion that certain medical findings are highly suggestive of shaking to the exclusion of other possible causes' assumes that AHT was or would be diagnosed simply by reference to the presence of the triad injuries and would not involve the exclusion of other possible causes. The statement that '[t]here is limited scientific evidence that the triad and therefore its components can be associated with traumatic shaking (low quality evidence)' flows from the uncritical acceptance of the central tenet of the SBU Report.

250 In her rebuttal report, Dr Tully commented on Thiblin 2020 in the following terms:

- The researchers employed the same narrow inclusion and broad exclusion criteria as the SBU Report with the result that the total number of cases identified over the 17-year period was only 36. By way of contrast VFPMS sees an equivalent number of children and infants with head injuries for assessment over a period of one to two years.
- In 30 of the 36 cases no evidence of injury was found, although five infants had no cranial imaging, four did not have eye examinations and nine were not x-rayed to look for bony injury. Failure to search for and failure to detect injury (ie an absence of evidence) is not evidence of absence. The findings overall are in line with clinical experience. VFPMS sees many cases where caregivers admit to shaking the infant and/or someone states that they have witnessed a shaking event and in many of those cases investigations do not reveal evidence of injury.

251 Dr Tully continued:

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106 Ibid 9 (emphasis added, citations omitted).
107 Ibid 11 (citations omitted).
108 Ibid.

> While small, [the] study supports the assertion by many health professionals from multiple disciplines across the globe that in a percentage of infants who are witnessed to have been shaken or shaken with impact (or confessions were obtained of the same), findings such as [subdural haemorrhages], [retinal haemorrhages], bruises and fractures can be seen, individually and in combination. This finding is entirely compatible with our clinical experience and the findings reported in the literature.
>
> The idea that shaking (with or without impact) will always cause this constellation of [injuries] is without merit and is analogous to the hypothesis that falling from a swing will always result in a head injury.
>
> In 6 cases, evidence of physical abuse was found. In these cases, Professor Högberg and his team attribute the injuries to spurious 'vulnerabilities' of the infants. *Their conclusions demonstrate active rejection of the most logical conclusion in favour of hypothetical speculative theories about causation. This mirrors Professors Högberg and Wester's approach to causation in Kaleb's case.*[109]

(ii) Wester 2021

252 Wester 2021 is a retrospective review that seeks to cast doubt on abuse diagnoses by medical experts in the Norwegian courts by scrutinising the underlying medical documentation. It does so with a view to identifying organic causes for the death or impairment of the infants in question, as it is predicated on there being no connection between the presence of triad injuries and AHT.

253 Wester 2021 identified 17 children, each of whom was diagnosed as suffering AHT by court appointed experts, in the data registry for Norwegian courts for the period 2004–2015 and obtained medical documentation about those cases from relevant health institutions. Fifteen of the 17 cases were infants (with a mean age 2.6 months) and 11 of them were boys. A high proportion (41.2 per cent) were born to immigrant parents and 31.3 per cent were born prematurely.[110]

254 According to Wester 2021, the medical findings can be explained by alternative diagnoses in 16 of the 17 children: eight boys had clinical and radiological characteristics compatible with external hydrocephalus (ie BESS) complicated by chronic subdural haematoma. Six children had findings compatible with hypoxic ischaemic insults.[111]

255 According to Wester 2021, the similarity in gender and age distribution between the BESS-like group and the gender and age distribution in a nationwide study of BESS infants added further doubt about the correctness of the abuse diagnosis in these boys.[112]

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109 Italicised emphasis added.

110 Wester 2021, 1.

111 Ibid.

112 Ibid 8–9, noting that in the study group there was found to be (citations omitted):

> [A] striking male preponderance, almost two-thirds were boys, and most of these boys had clinical and radiological characteristics compatible with external hydrocephalus – BEH/BESS

256 Wester 2021 therefore concluded that in the majority of the 17 cases studied, there were non-traumatic causes of the identified injuries:

> Our results indicate that the head injuries in the majority of the investigated children possibly, or even probably, had a non-traumatic cause, and consequently, that these children not necessarily had been subjected to an inflicted head injury caused by shaking or direct impact. We emphasise that we cannot, based on the present study, rule out that these children had been shaken, in the same way as it is impossible to prove that any of them had been shaken, based only on clinical and radiological findings made after the alleged violence.
>
> There appears to be a need for a strict investigation protocol for cases of alleged SBS/AHT.[113]

257 Wester 2021 generated an immediate critical response from a group of Norwegian medical specialists, who published a paper entitled 'Re-evaluation of abusive head trauma in Norway appears flawed' ('Stray-Pedersen').[114] The authors of Stray-Pedersen had first-hand knowledge of three of the case studies in Wester 2021. They found that Wester 2021 omitted important facts from the description of those case studies.

258 For example, according to Stray-Pedersen, Wester's description of one case omitted the clinical presentation and several pertinent injuries. Wester 2021 describes an intracerebral haematoma. However, the baby presented with an acute subdural haematoma that required surgical evacuation. In addition, CT and MRI scans revealed multiple contusion injuries in the brain parenchyma. Multiple bruises were present, as well as a posterior rib fracture. Metaphyseal and corner fractures were identified in both femurs and both tibiae during the radiological evaluation.[115]

259 In respect of another case, Wester 2021 states that the injuries were due to a hypoxic-ischaemic insult and that the infant had old rib fractures and a clavicle fracture. However, according to Stray-Pedersen:

> [A]gain, critical details are missing. This infant presented with new, not old, fractures to the clavicle, scapula and two ribs. Multiple bruises were documented. CT and MRI scans showed bilateral acute subdural haematoma, as well as a rapidly evolving brain oedema with hypoxic-ischaemic changes, which resulted in end-stage cystic global encephalopathy.
>
> [Wester 2021] does not report that, critically, the defendant confessed to having

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> — complicated by a chronic [subdural haematoma] or hygroma. Such haematomas or hygromas are common complications to BEH. Numerous studies have shown a similar gender distribution in BEH populations as in the present material. In the only population-based epidemiological study on BEH, the Norwegian male preponderance was even higher — 86.4%. Adamsbaum et al and Vinchon et al reported large numbers of allegedly abused children, with a pooled population of 157 infants. This population had a similar marked male preponderance (73%) as in populations of infants diagnosed with BEH. This similarity indicates that BEH may have been misdiagnosed as AHT. Such cases have indeed been reported, and several authors have warned that BEH may represent a pitfall in the diagnosis of inflicted head injury. Also, Vinchon et al have demonstrated this predisposition for [subdural haematoma] in BEH: Wester 2021.

113 Ibid 12.

114 Arne Stray-Pederson et al, 'Re-Evaluation of Abusive Head Trauma in Norway Appears Flawed' (2022) 111(4) *Acta Peadiatrica* 793.

115 Ibid 796.

> repeatedly shaken the child back and forth. He demonstrated this act on camera during the criminal investigation and his early confession was substantiated during the court hearings.[116]

260 Stray-Pedersen, generously describing these omissions as only puzzling, raised the following concern:

> We do not have access to all cases in [Wester 2021], but this preliminary review raises concerns about why significant facts are missing. While we welcome challenges to the diagnosis and legal proceedings surrounding these cases, we believe that this should involve high-quality research that is fully transparent. This should include all of the clinical information and be presented in a manner that is objective and avoids circular reasoning. We are also concerned about the impact that the incomplete representation of the three cases … we specifically comment on may have on clinical judgement and medical expert testimony in the future. This can make it even more difficult to protect those infants who are most at risk and ensure justice for them and their parents.[117]

(iii) Thiblin 2021

261 The third paper generated by the SBU Report, Thiblin 2021, used the same Swedish National Patient Register data as Thiblin 2020 to identify infants investigated for suspected physical abuse. It compared medical findings and reported types of trauma with the presence or non-presence of retinal haemorrhages. The aim was to test the two prevailing hypotheses regarding the aetiology of infant retinal haemorrhages: (a) traction forces exerted by the lens and/or corpus vitreum on the retina during infant shaking; and (b) retinal vessel leakage secondary to intracranial pathology and raised intracranial pressure. In other words, Thiblin 2021 investigated intracranial pressure as the cause of retinal haemorrhages in infants suspected of having been abused.

262 Thiblin 2021 identified the same 337 infants with a maltreatment diagnosis in the Swedish database, and retrieved medical records for 257 (76 per cent) of them. The inclusion criterion was that the infant had been examined with both fundoscopy and neuroimaging by CT and/or MRI, which resulted in the inclusion of 148 infants. However, the examining ophthalmologists' original reports were not always available and, when present, were not systematically detailed. For this reason, retinal haemorrhages were not graded beyond unilateral or bilateral.[118]

263 The 148 infants who satisfied the inclusion criteria were first separated into two groups based on the presence or non-presence of retinal haemorrhages, and the 29 retinal haemorrhage cases identified were then divided into subgroups of unilateral retinal haemorrhages and bilateral retinal haemorrhages for further comparisons of 'the main outcome'.

264 The main outcome was defined as 'any kind of intracranial pathology', including intracerebral haemorrhage, acute subdural haemorrhage, chronic subdural haemorrhage, hygroma, acute subarachnoid haemorrhage, brain oedema, cortical vein

116 Ibid.
117 Ibid 797.
118 Thiblin 2021, 801.

thrombosis, sinus vein thrombosis, and contusion.[119] '[A]ny kind of intracranial pathology' was recorded in 15 (13 per cent) of the 119 non-retinal haemorrhage cases and in 27 (97 per cent) of the 29 retinal haemorrhage cases. All 19 infants with bilateral retinal haemorrhages were found to have some kind of intracranial pathology. Small and isolated retinal haemorrhages were also found in two infants without intracranial pathology.

265 Witnessed or admitted physical abuse of any kind was reported in 35 (29 per cent) of the 119 non-retinal haemorrhage cases and in 2 (7 per cent) of the 29 retinal haemorrhage cases.[120] Of 27 infants subjected to witnessed or admitted shaking, only one had bilateral retinal haemorrhages, and this infant was pre-term with both chronic and acute intracranial conditions.

266 As a result, Thiblin 2021 found that retinal haemorrhage as a consequence of vitreoretinal traction was not supported. Instead, it concluded:

> [T]here was a strong association between retinal haemorrhage and intracranial pathological conditions in infants with suspected shaken baby syndrome/abusive head trauma. Almost all (97%) infants with retinal haemorrhage also had intracranial pathology, whereas only a small proportion (13%) without retinal haemorrhage did so. Conversely, a large proportion (62%) of infants with intracranial pathology also had retinal haemorrhage. Retinal haemorrhage without intracranial pathology was found in only two cases.[121]

267 Thiblin 2021 posited that non-birth-related retinal haemorrhages in infants are secondary to traumatic or non-traumatic intracranial pathology associated with raised intracranial pressure and that isolated shaking is unlikely to cause retinal haemorrhages independently of intracranial pathology. It therefore concluded that the presence of retinal haemorrhages provides no reliable information about the aetiology of the intracranial pathology, and abusive head trauma cannot be inferred solely based on the combination of the two.[122]

268 Dr Iles was unimpressed by Thiblin 2021:

> There is a major flaw in this paper and they acknowledge that flaw because they just talk about retinal haemorrhages [being] present or absent and in terms of paediatric ophthalmological practice, that is really insufficient in terms of

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119 Findings consistent with raised intracranial pressure were defined as any statement of papillary oedema, rapidly increasing head circumference, sunset gaze, increased suture diastasis, compressive effect of extra-cerebral effusion such as midline shift or compressed ventricles, alternatively an extra-cerebral effusion (subarachnoid or subdural) in combination with enlarged ventricles as seen in external hydrocephalus or objectively measured or observed raised intracranial pressure (such as 'fluid emptying under high pressure') during neurosurgical interventions. Symptoms consistent with raised intracranial pressure were defined as vomiting, seizures, and lowered level of consciousness: ibid.

120 Witnessed or admitted shaking *without* blunt force impact was reported in one (3%) of the 29 retinal haemorrhage cases and in 22 (18%) of the 119 non-retinal haemorrhage cases. Witnessed or admitted shaking *with* blunt force was reported in one (3%) of the 29 retinal haemorrhage cases and in three (2.5%) of the 119 non-retinal haemorrhage cases. Witnessed or admitted/reported blunt force was documented in nine (31%) of the 29 retinal haemorrhage cases and in 38 (32%) of the 119 non-retinal haemorrhage cases: ibid 804.

121 Ibid 805.

122 Ibid 806.

> looking at a sophisticated variable and saying, where there is a spectrum of findings, whether it's present or absent is really insufficient data to draw any meaningful conclusion, in my view.

269 In other words, the inability to have regard to the extent and nature of the retinal haemorrhages in the relevant case studies rendered Thiblin 2021 of very limited scientific value in assessing the diagnostic value of the triad injuries for AHT.

270 However, in a brief review of Thiblin 2021 published in the same 2022 issue of *Acta Paediatrica*, Dr Waney Squier, a controversial neuropathologist from the United Kingdom and a longstanding sceptic of SBS/AHT, described the results of Thiblin 2021 as 'striking':

> [A]ll babies with bilateral [retinal haemorrhages] had intracranial pathology, and just two babies with unilateral [retinal haemorrhages] did not. On the contrary, there was no association between [retinal haemorrhages] and shaking; of 27 babies who suffered confessed or witnessed shaking without impact, only two had [retinal haemorrhages] and one of these had intracranial pathology with suspected cortical vein thrombosis. Just 7% of babies with [retinal haemorrhages] were reported to have suffered physical abuse of any kind compared with 29% of babies without [retinal haemorrhages]. Infants without [retinal haemorrhages] were more likely to have objective evidence of extracranial trauma, such as fractures or bruises or to have been referred because a sibling was thought to have been abused.[123]

271 Dr Squier continued:

> These findings come soon after Binenbaum, an ophthalmologist and protagonist of the shaken baby hypothesis, also effectively refuted previous claims for the specificity of [retinal haemorrhages] for shaking. He described a significant change in the way paediatricians should think of [retinal haemorrhages]. Patterns that might distinguish traumatic from medical causes of [retinal haemorrhages] are not perfectly correlated with abuse. 'There is generally more overlap than appreciated between abusive and accidental retinal hemorrhages'.[124]

272 Dr Squier noted, however, that recorded data were insufficiently detailed to further grade the retinal haemorrhages beyond unilateral or bilateral, and only one baby had a follow-up eye examination.[125]

273 We shall return to Dr Binenbaum's views in more detail below. Suffice at this point to say that we do not accept Dr Squier's statement that Dr Binenbaum has 'refuted' the specificity (ie particular distributions and patterns) of retinal haemorrhages for AHT or 'shaking'.

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123 Waney Squier, 'Infant Retinal Haemorrhages Correlate with Chronic Subdural Haemorrhage, Not Shaking' (2021) *Acta Paediatrica* 714, 714 ('Squier').

124 Ibid 2 (emphasis altered).

125 Ibid.

(iv) Andersson 2021

274 Dr Squier referred to a further paper produced in the line of studies following the SBU Report, to which we were also referred by its co-authors, Professors Wester and Högberg: Jacob Andersson et al, 'External Hydrocephalus as a Cause of Infant Subdural Haematoma; Epidemiological and Radiological Investigations of Infants Suspected of Being Abused' ('Andersson 2021').[126]

275 The aim of Andersson 2021 was to investigate the extent to which external hydrocephalus (or BESS) was present in infants with acute and chronic subdural haematomas who were undergoing evaluation for abuse. It examined 85 infants who met the criteria, distinguishing between those with 'chronic' subdural haematomas and those with 'acute' subdural haematomas to conclude that a 'substantial proportion of infants with [chronic subdural haematomas] had findings suggesting external hydrocephalus'.[127]

276 As summarised by Dr Squier:

> [The authors] identified infants with unexplained [subdural haematomas] from the register of the Swedish National Board of Forensic Medicine. They assessed the radiological age of [subdural haematomas] and recorded head circumference and measurements of intracranial fluid compartments, looking for evidence of long-standing extracerebral fluid collections. They found a similar preponderance of chronic [subdural haematomas] and a significant correlation with increased head circumference. A substantial proportion of infants with chronic [subdural haematomas] also had findings suggesting external hydrocephalus, a natural condition with increased fluid in the subarachnoid space, which itself may predispose to spontaneous subdural bleeding. This underlines the importance of head circumference charts and of scrutinising brain images for evidence of pre-existing fluid collections, in order not to confuse these babies with those with traumatic [subdural haematomas].[128]

277 Once again, this represented an attempt to re-classify as signs of natural disease what would otherwise have been investigated as signs of inflicted or accidental head injury.

278 In his oral evidence, Professor Ditchfield was scathing about Andersson 2021. He said that there was a 'major problem' with the definitions used for 'acute' and 'chronic' collections (which was a significant distinction underlying the methodology and the findings of Andersson 2021) as it was not possible to determine the type of fluid in the collection without a tap. He described the project of taking patients who had benign or 'chronic' subdural haematomas and trying to ascertain how many of them had BESS as deploying 'circular reasoning' because, by including the subdural collection in the measurement, nearly every patient was likely to meet the threshold for BESS. Furthermore, while the study purported to use as the threshold the 95th percentile in the normative data, it in fact used the 50th percentile. Importantly, patients with acute trauma were excluded, which meant that the factors that might suggest actual abuse were excluded, making it, he said, 'a very convenient way to study it'.

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126 (2021) 126 (January) *Paediatric Neurology* 26.
127 Ibid 33.
128 Squier, 714.

(d) Retinal haemorrhages as a sign of AHT

279 It is useful in this context to consider some of the medical evidence placed before the Court concerning the association between retinal haemorrhages and AHT. The proponents of that connection stress the importance of the extent and patterns of retinal haemorrhages in the diagnosis of traumatic head injury. As discussed, Thiblin 2021 associated retinal haemorrhages in infants with intracranial pressure rather than with head trauma. However, it did not — and could not due to the limitations of the available information — consider the extent or distribution of the retinal haemorrhages identified.

280 The Court was referred by the respondent to a 2017 paper by Dr Alex Levin that considered retinal haemorrhage as a signifier of AHT in the context of the SBU Report.[129] According to Dr Levin, there is a 'plethora of scientific evidence' that retinal haemorrhages are a cardinal feature of AHT characterised by repeated acceleration–deceleration forces with or without blunt impact trauma.[130] They have high diagnostic sensitivity and specificity, in particular when they are multi-layered, too numerous to count, extend to the retinal edge and occur with macular retinoschisis[131] with or without surrounding retinal folds. Dr Levin pointed out that nowhere in the SBU Report is anything other than generic 'retinal haemorrhage' considered, a concept which, according to Dr Levin, runs counter to the very fabric of ophthalmology and its tools, which strive to distinguish and characterise specific types and patterns of haemorrhagic retinopathy.[132]

281 According to Dr Levin:

> Hundreds of papers support our knowledge about retinal haemorrhage in abusive head trauma including clinical studies of child victims, postmortem studies, studies of accidental trauma and other nonabuse clinical scenarios, animal studies, finite element analysis and dummy models. There is also the wealth of clinical experience shared by hundreds of paediatricians and other specialists who have attended the bedsides of child victims and their families. The SBU panel did not include a single child abuse paediatrician — individuals uniquely trained in this specialty area — or paediatric ophthalmologist.[133]

282 However, due to impossibly strict criteria, the SBU Report included only a small amount of the information available and considered 'retinal haemorrhage' generically throughout. Dr Levin was also critical of its use of a paper by Firsching et al,[134] which showed that increased intracranial pressure (referred to as 'iICP' in Levin) correlates with venous outflow pressure in adults, to state that 'retinal haemorrhage' may occur due to increased intracranial pressure. However, none of Firsching's patients had retinal haemorrhages.

283 Dr Levin continued:

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129 Alex V Levin, 'The SBU Report: A Different View' (2017) 106(7) *Acta Paediatrica* 1037 ('Levin').

130 Ibid 1037.

131 Retinoschisis refers to the separation of the layers of the retina.

132 Ibid.

133 Ibid.

134 Referring to Raimund Firsching et al, 'Noninvasive assessment of intracranial pressure with venous opthalmodynamometry' (2011) 115(2) *Journal of Neurosurgery* 371.

> They [the SBU project team] also cite Lashutka, a very weak study on adults that says nothing about retinal haemorrhage: it is actually a study about increased intraocular pressure in patients with iICP. Although peripapillary preretinal and intraretinal haemorrhage (usually superficial nerve fibre layer haemorrhage) and prepapillary haemorrhage can occur from iICP, there is much evidence that more widespread retinal haemorrhage does not occur in children except in the setting of hyper-acute spikes in iICP as seen in fatal head crush injury, fatal motor vehicle accidents or ruptured aneurysms and other vascular malformations, entities which are readily distinguished from child abuse at the bedside.
>
> …
>
> Considering 'retinal haemorrhages', generically, fails us both clinically and scientifically. Ignoring the full breadth of scientific evidence about a clinical scenario misrepresents the knowledge base. The authors state 'If and when a case comes to court, it is important for the court to have access to scientific expertise to express an opinion in accordance with the professional ethical principles and applicable legal rules concerning certificates and opinions.' It seems that they have failed to heed the principles of their own advice.[135]

284 Dr Levin had previously studied the relationship between retinal haemorrhages and intracranial pressure in 2012 in a paper co-authored with Tiffany Shiau, entitled 'Retinal Hemorrhages in Children: The Role of Intracranial Pressure'.[136] This was a review of published clinical, post-mortem and experimental research findings worldwide, the result of which was expressed as follows:

> In general, elevated [intracranial pressure] does not cause extensive haemorrhagic retinopathy. Papilledema [the swelling of the optic nerve as it enters the back of the eye due to raised intracranial pressure] may be associated with a small number of haemorrhages on or around the optic disc. There are isolated case reports that severe hyperacute [intracranial pressure] elevation, unlike the subacute pressure increase in abusive head injury, in children may rarely result in extensive retinal haemorrhage. These diagnoses are readily distinguished from child abuse.[137]

285 Shiau and Levin concluded that increased intracranial pressure may result in papilledema[138] with small numbers of intraretinal and preretinal haemorrhages in or around the optic nerve. There was some evidence based on isolated case reports in unusual circumstances that severe hyperacute intracranial pressure elevation in children may result in extensive retinal haemorrhaging. However,

> [t]he concept that extensive [retinal haemorrhage] in very young children, in the absence of the few readily recognizable rare circumstances mentioned herein, is due to increased [intracranial pressure] is not supported by peer-reviewed published evidence or empirical experience. … These diagnoses are readily distinguished from abuse. In most cases of AHT, elevated [intracranial pressure] is seen but even in this scenario, [intracranial pressure] appears to play only a

135 Levin, 1038 (emphasis altered) (citations omitted).
136 (2012) 166(7) *Archives of Pediatrics & Adolescent Medicine* 623.
137 Ibid 623.
138 The swelling of the optic nerve as it enters the back of the eye due to raised intracranial pressure.

> small role, perhaps modulating the appearance of haemorrhages in a particular child along with other factors.[139]

286 The other paper on this topic frequently referred to in evidence before us was the 2013 study by Gil Binenbaum et al entitled, 'Patterns of Retinal Haemorrhage Associated with Increased Intracranial Pressure in Children' (Binenbaum 2013).[140]

287 Binenbaum 2013 examined the incidence and patterns of retinal haemorrhages associated with increased intracranial pressure, measured by lumbar puncture, in children without trauma. It described a study of 100 children undergoing lumbar puncture as part of their routine clinical care that looked for retinal haemorrhages where elevated intracranial pressure was present. Inclusion criteria were absence of trauma, lumbar puncture opening pressure greater than or equal to 20 cm of water, and a dilated fundus examination by an ophthalmologist or neuro-ophthalmologist.

288 The result of Binenbaum 2013 was as follows:

> Only a small proportion of children with nontraumatic elevated [intracranial pressure] have [retinal haemorrhages]. When present, [retinal haemorrhages] are associated with markedly elevated [lumbar puncture opening pressure], intraretinal, and invariably located adjacent to a swollen optic disc. This peripapillary pattern is distinct from the multilayered, widespread pattern of [retinal haemorrhage] in abusive head trauma. When [retinal haemorrhages] are numerous, multilayered, or not near a swollen optic disc (eg, elsewhere in the posterior pole or in the retinal periphery), increased [intracranial pressure] alone is unlikely to be the cause.[141]

289 Binenbaum 2013 found that a small proportion of children with elevated opening pressure on lumbar puncture had retinal haemorrhages, but stated:

> The [retinal haemorrhages] observed in association with raised [intracranial pressure] were not consistent with the severe haemorrhagic retinopathy seen in many victims of AHT ... Even when children had severe papilledema and markedly elevated [lumbar opening pressure], the [retinal haemorrhages] were neither widespread nor numerous, were not present in the peripheral retina, and did not even extend throughout the posterior pole of the fundus. Instead, the [retinal haemorrhages] were limited to the peripapillary (peri-optic nerve head) region. The types of haemorrhage were limited to optic disc and intraretinal haemorrhage, in contrast to the multilayered [retinal haemorrhages] often seen in children with AHT. Finally, splitting of the retinal layers (retinoschisis) and perimacular retinal folds were not observed in the study, even in a case of papilledema causing vision loss severe enough to warrant optic nerve sheath fenestration.[142]

290 It continued:

> There are also differences between some of the milder [retinal haemorrhage] patterns that can be seen with accidental or AHT and the [retinal haemorrhages]

139 Ibid 627.
140 (2013) 132(2) *Pediatrics* e430.
141 Ibid e434.
142 Ibid e432.

> observed in this study. The intraretinal haemorrhages in children with raised [intracranial pressure] were superficial, nerve fibre layer haemorrhages, whereas children with head trauma commonly also have deeper, dot-and-blot intraretinal haemorrhages … In addition, all of the study children with [retinal haemorrhages] had severe papilledema, but disc swelling is an uncommon finding in children with AHT, present in [less than] 9% of cases.[143]

291 Relevantly, Binenbaum 2013 contains the following propositions:

(a) Retinal haemorrhages are an important sign of paediatric AHT, present in an estimated 85 per cent of cases.[144]

(b) The extensive clinical experience of paediatric ophthalmologists suggests that isolated raised intracranial pressure rarely causes retinal haemorrhage, and that retinal haemorrhage due to raised intracranial pressure is limited to the peripapillary retinal haemorrhages sometimes seen with papilledema.[145]

(c) The haemorrhage pattern observed in Terson syndrome, which is thought to arise from a sudden increase in intracranial pressure due to intracranial haemorrhage, is primarily that of preretinal and vitreous haemorrhage and rarely resembles the patterns of haemorrhage seen in AHT.[146]

292 Against this background, Professor Eriksson rejected the proposition that the pattern of retinal haemorrhages may indicate whether the baby has been shaken or whether the baby has haemorrhages for another reason. This, he says, was confirmed by 'one of the greatest proponents of specificity of retinal haemorrhages, Binenbaum, who has written several papers supporting Levin's standpoint before'.

293 Professor Eriksson told the Court that Dr Binenbaum changed his position 'at a meeting' in April 2021:

> I can tell you that we recently had a case, a triad only case in Sweden, where the clinicians and especially the paediatric ophthalmologist identified these allegedly highly specific haemorrhages and patterns in the retina of the child. And was one hundred per cent sure that the baby had been shaken. But the correct diagnosis in that case was revealed later, and the correct diagnosis was a benign external hydrocephalus, a disease process.
>
> …
>
> Binenbaum now says that — well I don't — I cannot quote exactly word by word but he says something like the overlap between intentional and accidental trauma regarding patterns of retinal haemorrhages that is greater than previously appreciated. And that is also my opinion. There is so much overlap. You cannot use the pattern of retinal haemorrhages.

294 This is the 'refutation' to which Dr Squier referred in her brief review of Thiblin 2021.

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143 Ibid e432–3.
144 Ibid e431.
145 Ibid.
146 Ibid e433.

295 It appears that the presentation in question was given by Dr Binenbaum in April 2021 at the virtual annual meeting of the American Association for Pediatric Ophthalmology and Strabismus. Only a brief report of that presentation[147] and a conference abstract[148] were available at the time of hearing.

296 The report describes Dr Binenbaum as proposing a 'slightly new way' of interpreting retinal haemorrhages, although it also refers to him suggesting a 'new paradigm that would identify retinal haemorrhage patterns that would indicate trauma, as well as distinguish between trauma and medical causes' rather than 'trying to diagnose abuse based on number, type and location of retinal haemorrhages'.[149]

297 Dr Binenbaum is quoted in the report as follows:

> There's good evidence that retinal haemorrhage is associated with increasing likelihood of abusive rather than accidental head trauma, which is why it's important to describe in detail what you see on your examination. But this paradigm is incomplete … Retinal haemorrhage severity is not perfectly correlated with abuse. There are missing useful patterns. And it doesn't address medical causes of retinal haemorrhages. With regard to severity, there is generally more overlap than appreciated between abusive and accidental retinal haemorrhages.[150]

298 In substance, Dr Binenbaum is reported to be seeking to find patterns to distinguish retinal haemorrhages caused by abuse and those caused by accidental trauma. However, medical causes of retinal haemorrhage are also mentioned: the report of the presentation describes 'traumatic patterns' identified by Dr Binenbaum and 'medical patterns'. Dr Binenbaum is then quoted as follows:

> The question becomes, is there a pattern of haemorrhage that tells us this is trauma instead of a medical cause of retinal haemorrhage? If a traumatic pattern is identified, only then would we secondarily consider the severity of retinal haemorrhage along with the severity of non-ocular findings as a gauge for the severity of trauma that can be considered by the child abuse team as they consider the history provided by the caregivers.
>
> The key points are to think about diagnosing trauma and not to think about diagnosing abuse.
>
> Look for a pattern of retinal haemorrhage that tells you this is trauma and then you can make that statement clearly. Then simply describe the severity of the retinal haemorrhage you see to the child abuse team so that they can gauge the severity of trauma and make a determination of the likelihood of abuse.

299 The reported extracts from Dr Binenbaum's presentation are plainly not contiguous and it seems unlikely that the proposed 'slightly new way of interpreting' retinal

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147 See Patricia Nale, 'Recognising Retinal Haemorrhage Pattern Aids in Diagnosing Abuse', *Ocular Surgery News* (Web Page, 13 April 2021) <https://www.healio.com/news/ophthalmology/20210413/recognizing-retinal-hemorrhage-patterns-aids-in-diagnosing-abuse> ('Nale').

148 See Gil Binenbaum et al, 'Retinal hemorrhage patterns: a new paradigm' (2021) 25(4) *Journal of American Association for Pediatric Ophthalmology and Strabismus* e3.

149 Nale.

150 Nale.

haemorrhages involves a marked departure from Dr Binenbaum's earlier position that retinal haemorrhages caused by trauma are distinguishable from retinal haemorrhages caused by intracranial pressure or other disease processes. Certainly Dr Squier understood the relevant issue to be distinguishing between retinal haemorrhages caused by accidental and non-accidental head trauma.

300 The abstract provides a little more clarity. It refers to a study carried out by Dr Binenbaum, Dr Levin and others directed to developing 'a new framework for interpreting retinal findings in child abuse evaluations'. It is convenient to set out the abstract in full:

> **Introduction**: The current paradigm for interpretation of retinal hemorrhages (RH) focuses on diagnosis of abusive head trauma based on severity of RH, for which classifications have been published. While RH severity has value, there is overlap in severity between accidental and abusive injuries, some patterns are diagnostic even if RH are not severe, and medical diagnoses are not addressed. We sought to develop a new paradigm for RH interpretation using patterns that distinguish medical from traumatic causes of RH.
>
> **Methods:** Three masked ophthalmologists reviewed 188 fundus photographs of RH from many causes in an iterative process to identify patterns that distinguish medical from traumatic causes. Based upon these patterns, a new framework for interpreting retinal findings in child abuse evaluations was developed.
>
> **Results:** Distinguishing patterns were defined. Traumatic patterns included 'peri-macular', 'central macular sparing', 'midperipheral sparing', 'cherry hemorrhages', 'too numerous to count hemorrhages with nonradiating areas', and 'carpeting'. Medical patterns included sectoral distribution, numerous RH in radiating pattern, superficial peripapillary RH with disk swelling, and primarily peripapillary preretinal or vitreous hemorrhage. Presence of focal white lesions, lipid, or disk swelling also supported a medical cause.
>
> **Conclusion/relevance:** In a newly developed paradigm, diagnostic interpretation of RH should first involve ophthalmologist identification of these patterns to distinguish between a medical and traumatic cause, not to diagnose abuse. Once a traumatic pattern is identified, the severity of RH and non-ocular injuries can be used by the child abuse team to evaluate the plausibility of the history provided by caregiver.

301 Like the report, the abstract emphasises the need to distinguish between medical and traumatic causes of retinal haemorrhages before diagnosing abuse. Medical and traumatic patterns of retinal haemorrhages are distinguishable. Once traumatic patterns have been identified, it is then necessary to distinguish between accidental and non-accidental trauma having regard to both the retinal haemorrhages and 'non-ocular' injuries.

302 There is nothing in the report or the abstract to suggest that Dr Binenbaum has moved away from the propositions in Binenbaum 2013 set out at [291] above.

303 It was put to Dr Tully, and she agreed, that a major finding reported in the 2021 Binenbaum presentation was that there was more overlap than was previously appreciated between retinal haemorrhages caused by AHT and those caused by

accidental trauma. Useful diagnostic patterns had been missed in this regard. Dr Tully gave evidence that the understanding of retinal haemorrhage patterns was advancing all the time and that she understood Dr Binenbaum to be referring to an increased understanding of the patterns of retinal haemorrhage in circumstances where there is overlap between patterns of retinal haemorrhages that can occur in accidental head trauma and those that can occur in inflicted head trauma. She did not believe that Dr Binenbaum was referring to the discrimination between, for example, raised intra-cranial pressure, CPR and the numerous other possible causes of retinal haemorrhages.

304 Dr Tully said that it had been understood for some time that significant accidental head trauma could in rare cases cause the same pattern of retinal haemorrhages as AHT. The pattern is seen in crush injuries of the head, as occurs in high-velocity motor vehicle accidents. She said that significant occipital impact with severe retinal haemorrhages had to be very carefully evaluated.

305 We observe that there is no suggestion that Kaleb suffered from any kind of accidental injury of the kind described.

306 For his part, Professor Eriksson dismissed the relevance of particular patterns of retinal haemorrhage to the diagnosis of AHT by referring to a 2011 study of 'blinded' ophthalmologists by Alan Mulvihill et al.[151] This study, Professor Eriksson said, clearly shows that the uninformed ophthalmologist cannot differentiate between the causes of retinal haemorrhages.

307 As the title suggests, Mulvihill et al is concerned with the interpretation of images. Four examiners[152] were shown 142 retinal haemorrhages on high quality RetCam photographs. The individual images/haemorrhages were selected from a database of over 100 children with accidental and abusive head injury and other encephalopathies. Specified haemorrhages from each image were classified by each examiner as a particular type according to their clinical understanding. The examiners were not called upon to identify the *cause* of the retinal haemorrhage, but which category the haemorrhage fell into: vitreous, pre-retinal, nerve fibre layer, intra-retinal/sub-retinal, schisis cavity or indeterminate.

308 According to the study's authors, this exercise demonstrated that the clinical classification of RetCam images of retinal haemorrhages in children, based on the generally held defining features of haemorrhages in different retinal layers, lacks consistency between examiners and even on re-examination by the same examiner.[153]

309 In the case of Kaleb, extensive retinal haemorrhages were observed directly and, following his death, his eyes were macroscopically examined and sectioned at St Vincent's Hospital in Sydney and the sections were examined and reported by a specialist. The summary of those findings was that there were bilateral optic nerve

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151 Alan O Mulvihill et al, 'An Inter-Observer and Intra-Observer Study of a Classification of RetCam Images of Retinal Haemorrhages in Children' (2011) 95(1) *British Journal of Ophthalmology* 99 ('Mulvihill et al').

152 The examiners were two experienced paediatric consultant ophthalmologists, a paediatric ophthalmology fellow and a consultant paediatric neurologist with experience in non-accidental head injury.

153 Mulvihill et al, 101.

sheath haemorrhages present and extensive bilateral retinal haemorrhages involving all layers of the retina. There was no question of miscategorising the retinal haemorrhages identified in Kaleb in the manner described in Mulvihill et al.

310 Professor Wester dealt with retinal haemorrhaging at a number of points in his oral evidence. Unlike Professor Eriksson, he did not address the Levin paper.

311 Professor Wester relied on a very short paper published in 2017 by Dr Joseph Scheller, neurologist, responding to papers in an issue of *Acta Paediatrica* debating the scientific basis for the diagnosis of AHT.[154] Dr Scheller listed 10 cases in which infantile retinal haemorrhages were found in the absence of brain or bodily injury.[155] Of the 10 cases, four had unilateral retinal haemorrhages, with the eye findings noted to be on the same side as a small subdural or subarachnoid haemorrhage, which Dr Scheller considered to be consistent with the mechanisms of abrupt increases in intracranial pressure and venous stasis.[156] Dr Scheller suggested that in the absence of brain injury, macrocephaly and chronic subdural hygroma may make the child more prone to developing retinal haemorrhages.[157] It was also considered possible that when subdural hygroma is present, an event that triggers a small acute subdural or subarachnoid haemorrhage can also cause unilateral or bilateral retinal haemorrhages. Dr Scheller concluded:

> [E]xtensive multilayer retinal haemorrhages occur in infants who have not suffered any apparent brain injury. Clinicians should reassess the importance of retinal haemorrhages in the setting of suspected abusive head trauma.[158]

312 Dr Wester also relied on a case study published in 1999 by Dr Joseph Piatt Jr, entitled, 'A Pitfall in the Diagnosis of Child Abuse: External Hydrocephalus, Subdural Hematoma, and Retinal Hemorrhages',[159] which described the case of an infant with external hydrocephalus who developed retinal as well as subdural haemorrhages after sustaining a minor head injury. The author warned that although retinal haemorrhage in infancy had been considered virtually pathognomonic of child abuse, in the setting of external hydrocephalus a more cautious interpretation might be appropriate.[160]

313 According to Professor Wester, high intracranial pressure produces retinal haemorrhages because the pressure in the cranial vault is transferred along the optic nerve sheath to the retina. The retina is connected to the brain by a 'direct waterway', so increased intracranial pressure will also appear as increased intra-retinal pressure and cause bleedings there. Upon being questioned about Dr Tully's view that BESS is a 'benign' condition that cannot cause retinal haemorrhages, Professor Wester referred to Thiblin 2021, which he said showed that multi-layered retinal haemorrhages are associated not with shaking but with high intracranial pressure. According to Professor

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154 Joseph Scheller, 'Infantile Retinal Haemorrhages in the Absence of Brain and Bodily Injury' (2017) 106(12) *Acta Paediatrica* 1902 ('Scheller').

155 The infants selected for the study had one or more of the following symptoms: large head circumference, alteration of consciousness, emesis or irritability. There was no suggestion of abuse.

156 Scheller, 1904.

157 Ibid 1903.

158 Ibid 1904.

159 (1999) 7(4) *Neurosurgical Focus* 4:1–8.

160 Ibid 1.

Wester, if you have an intracranial condition giving rise to increased intracranial pressure, multi-layered retinal haemorrhages will follow.

314 Professor Wester was cross-examined about the retinal haemorrhages suffered by Kaleb. He maintained that the haemorrhaging was not the result of trauma, but rather 'external hydrocephalus [BESS] complicated with a subdural haematoma with re-bleedings'. Professor Wester accepted that in his left eye Kaleb had multiple retinal haemorrhages in all retinal layers extending to the retinal periphery and that in his right eye Kaleb had multiple retinal haemorrhages extending from the optic nerve through to the macula to the retinal periphery. He also accepted that Dr Rodriguez, the neuropathologist who carried out dissections at the request of Dr Iles, had described extensive bilateral retinal haemorrhages extending from the optic nerve to the ora serrata involving all layers of the neural retina, sparse haemosiderin-laden macrophages, multifocal sub-hyaloid and subretinal haemorrhages. When asked whether this pattern of retinal haemorrhaging was consistent with AHT Professor Wester again referred to Thiblin 2021:

> Well, as I told you we had just published an article with allegedly shaken babies [and] only the ones with increased intracranial pressure had this picture ... including all layers of the retina and everything, whereas those that were only shaken without any intracranial pathology did not have these retinal haemorrhages, indicating that it is not the shaking or hitting the head that causes these retinal haemorrhages, it is the increased intracranial pressure that is mediated through the optic nerve sheath to the retina and causes the bleeding there.
>
> No one has ever provided solid scientific evidence that shaking causes these retinal haemorrhages. It is an assumption … but no one has ever provided solid, scientific evidence that that is — that it is possible or that it is pathognomonic for shaking that they have these retinal haemorrhages. In all layers, just as described in Kaleb. And we know that he had a very high intracranial pressure that caused his death eventually.

315 We pause to record our understanding that the information available for Thiblin 2021 did not include any detail about the extent or distribution of the retinal haemorrhages found to be present in the study cases, and as a result it did not specify 'all layers of the retina and everything'.

316 Professor Wester summarised his position in relation to Kaleb as follows:

> My position is that if you have external hydrocephalus complicated with a subdural haematoma with re-bleedings in as we have discussed, at some time the [intracranial] pressure must and will be or may be so high that it causes retinal haemorrhage.

317 Professor Wester also maintained that 'quite a few publications … show that cardiac compression when you resuscitate a child can cause retinal haemorrhage'. He went on to list some other possible non-AHT-related causes of raised intra-cranial pressure: 'the birth process …, squeezing the head[,] … external hydrocephalus/macrocephaly, high risk deliveries and acute life-threatening events'.

318 Professor Wester explained why he, too, had used Binenbaum 2013 to support his position. He said that Binenbaum 2013 showed that retinal haemorrhage is dependent on raised intracranial pressure. The degree of raised intracranial pressure corresponds with the presence of retinal haemorrhage. He said:

> It's just that Binenbaum has shown that the increased intracranial pressure is sort of the cause of any retinal haemorrhage and why shouldn't a moderately increased intracranial pressure only give a moderate amount of retinal haemorrhage, that's my point.
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> … [A]ny retinal bleeding indicates an increased intracranial pressure. The higher the pressure the more pronounced retinal bleedings will be. And just the fact that cardiopulmonary resuscitation can cause a retinal haemorrhage when you in addition have an increased intracranial pressure, that may easily explain [the] extensive retinal haemorrhage in Kaleb and in many other children too.
>
> I mean these observations are all indications that the retinal haemorrhage is associated with increased intracranial pressure, the so-called Terson syndrome.

319 We find it difficult to understand how it can be said that Binenbaum 2013 shows that increased intracranial pressure is the cause of retinal haemorrhages in infants. This entirely ignores the findings that we have set out in para [291] above.

320 Professor Högberg considered whether the raised intracranial pressure seen in Kaleb on 14 January 2016 and on 23 January 2016 could have caused the retinal haemorrhages findings at autopsy. He observed that increased intracranial pressure may lead to stasis and congestion in venous retinal vessels, which is consistent with reports of subdural haemorrhages and retinal haemorrhages in infants with reported falls, benign external hydrocephalus, and hygromas. Professor Högberg thought it unlikely that the diagnosed extensive bilateral retinal haemorrhages and haemorrhages in Kaleb's optic nerve sheaths were related to birth. However, based on current knowledge, it could not be claimed that the haemorrhages were caused by SBS/AHT . It was more probable that the extensive bilateral retinal haemorrhages and haemorrhages in the optic nerve sheaths were caused by the intracranial pathology with increased intracranial pressure.

321 Of the Scandinavian witnesses, Professor Högberg's oral evidence dealt with retinal haemorrhages the least. He was not questioned at all about the Levin article. Professor Högberg repeated his view that he did not believe that retinal haemorrhages were a good indicator of AHT.

(5) Alternative diagnosis: Benign Enlargement of the Subarachnoid Space (BESS)

322 Before describing this evidence and outlining the points of difference between the experts, it is convenient to briefly restate what was known about the state of Kaleb's brain prior to his collapse on 23 January 2016.

323 As we have recounted, when Kaleb presented to Casey Hospital and MMC for the first admission on 14 January 2016, he had been vomiting, he had an increased head circumference and his mother described his fontanelle as raised and firm. An ultrasound was conducted at Casey Hospital, with the radiographer concluding that Kaleb had

'bilateral lateral ventricular dilation with prominent extra-axial spaces bilaterally'. He was referred for an MRI which was conducted on 15 January 2016 at MMC. The MRI noted 'mild prominence of bilateral frontal extra–axial spaces measuring [up to] 8 mm on the right and 10 mm on the left. Within this area are bilateral frontal … collections measuring 4 mm in maximal depth on the left and 2 mm on the right'.

324 The MRI report concluded:

> Mild ventricular dilation, aetiology unknown. Small bilateral frontal subdural hygromas. No intra-axial or extra-axial haemorrhage.

325 The neurosurgical team reviewed the MRI and noted, 'MRI brain — front extra-axial CSF (subarachnoid and subdural) no hydrocephalus'.

(a) Professor Wester's evidence supporting a diagnosis of BESS

326 Professor Wester was asked whether he was able to provide an opinion as to an alternative cause of Kaleb's death.[161] He concluded that the cause of Kaleb's death was a global ischemia caused by the cardiopulmonary arrest resulting in a lack of oxygenated blood supply to the brain on the evening of 23 January 2016. However, the underlying cause was a rapidly developing external hydrocephalus (ie BESS)[162] complicated by a subdural haematoma that most probably elicited an Acute Life-Threatening Event ('ALTE').

327 According to Professor Wester, Kaleb had all the known predisposing and epidemiological factors associated with the development of BESS as well as the clinical and radiological features typical of BESS complicated by subdural haematoma. The predisposing factors were his sex and his moderately premature birth and low birthweight.

328 It is Professor Wester's opinion that BESS is known to predispose the sufferer to spontaneously occurring assemblies of subdural fluid containing blood and it may also cause retinal haemorrhages due to extensively increased intracranial pressure and Terson syndrome. Retinal haemorrhages are a known complication of any condition that causes a very high and very sudden increase in intracranial pressure and they cannot be taken as evidence of SBS/AHT.

329 According to Professor Wester, Kaleb experienced the formation of chronic subdural haematomas/hygromas associated with BESS. The ultrasound and MRI performed on 14 and 15 January 2016 respectively (during the first admission) showed bilateral chronic subdural fluid collections with the typical appearance of BESS and were features indicating a rapid development of increased intracranial pressure. When the subdural collections were finally tapped on 26 January 2016, three days after Kaleb's acute worsening with cardiac and respiratory arrest, the drained fluid was described by the surgeon as 'dark (old) blood stained [cerebrospinal fluid]' ('CSF'). According to

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161 Among other things, Professor Wester was provided with: copies of evidence given by Dr Tully and Dr Iles at committal and at trial as well as their reports; Professor Ditchfield's report and those of Dr Collins and Professor Duflou; and all reports relating to Kaleb's medical treatment along with four digital files of Kaleb's medical records, a copy of MR scans and images and an electronic photobook.

162 Which we will refer to simply as 'BESS'.

Professor Wester, this is a description of a chronic rather than an acute assembly of blood, which fits with the original signal of subdural fluid on the MRI 11 days earlier. If the cause of Kaleb's acute worsening was an acute event on the evening of 23 January 2016 while in the care of the applicant, the drained blood-stained fluid would be expected to be fresh and red, not dark and old.

330 Professor Wester described BESS as a special form of hydrocephalus with a marked male preponderance. He said that despite being described as 'benign', BESS can cause severe complications such as epilepsy, subdural haematomas, psychomotor delay and discognition. It is characterised by large amounts of CSF not only in the ventricles, but also in the fluid compartment outside the brain. This causes increased intracranial pressure and, consequently, 'a too-rapid growth of the head circumference'. According to Professor Wester, the increased 'CSF brim' is known to predispose the infant to an oozing of blood into the CSF, often referred to as subdural haematoma, even though the amount of blood components in the fluid is quite low. Usually this oozing of blood occurs spontaneously and without head trauma.[163]

331 According to Professor Wester, external hydrocephalus is clinically characterised by a large and/or rapidly growing head, often associated with symptoms of delayed development and elevated intracranial pressure such as irritability, feeding problems and epilepsy. Radiologically, it is characterised by an increasing amount of CSF in the subdural compartment during the first weeks or months after birth in addition to an increasing amount of CSF in the ventricles of the brain. The first sign is an increase in the distance between the two cerebral hemispheres frontally, followed by an increased fluid layer in the subdural compartment and, finally, increased fluid in the ventricles.

332 Professor Wester considered there to be very striking epidemiological similarities between BESS, subdural haematoma and SBS/AHT in infancy, having regard to gender and age. There are also similarities between MR images claiming to be typical examples of SBS/AHT and those published as examples of BESS complicated by subdural haematoma.

333 Professor Wester gave evidence that he and a neuroradiologically competent colleague went through the relevant literature on SBS/AHT published between 2008 and 2018 and found 40 publications with a total of 68 illustrations that claimed to show SBS/AHT. Of these, 78 per cent were compatible with BESS complicated by chronic subdural haematoma. Only 18 per cent were compatible with what one would expect to find after an acute event (ie shaking).

334 Professor Wester further opined that bilateral retinal haemorrhages are not specific or pathognomonic for SBS/AHT and stated that for more than a century, increased intracranial pressure has been known to cause retinal haemorrhages. Retinal haemorrhages can be seen in infants in circumstances unrelated to abuse, for example, in a large number of healthy newborns, in infants with macrocephaly/BESS, after high-

163 Based on CT images, Professor Wester distinguishes between fresh, acute and coagulated blood, which is referred to as an acute subdural haematoma, and a chronic subdural haematoma, where the blood elements were caused by oozing into the subdural compartment several days or even weeks before it shows up on the CT imaging.

risk deliveries, following ALTEs with dysphagic choking, and after cardiopulmonary resuscitation.

335 Turning to Kaleb specifically, Professor Wester noted the abnormally rapid rise in Kaleb's head circumference from birth to eight days before his sudden deterioration on 23 January which, together with the features revealed in the first MRI on 15 January, he considered strongly indicated the development of BESS. Consistently with this diagnosis, the MRI showed moderately enlarged ventricles and an increased distance from the brain surface to the inner skull.

336 Professor Wester based his radiological identification of BESS on the images generated by the MRI performed on Kaleb on 15 January (during the first admission), which were reproduced and discussed in Professor Ditchfield's statement. The MRI showed bilateral subdural fluid collections and the lateral ventricles at the upper range of normal in size.

337 Professor Wester noted that the subdural haematomas that were diagnosed on the CT scan on 23 January (during the second admission) were described as 'bilateral acute subdural haematomas … in the background of chronic subdural hygroma'. According to Professor Wester, what is described as a 'chronic subdural hygroma' is in fact a chronic subdural haematoma based on Professor Ditchfield's opinion that there was blood in the subdural fluid. Kaleb already had a small chronic subdural haematoma on 15 January, the only possible explanations for which were trauma to the head weeks before 15 January or that the chronic subdural fluid collection/haematoma was a complication of BESS.

338 Importantly for Professor Wester's thesis, he noted that during the autopsy Dr Iles found membranes in the subdural compartment indicating organisation (resolution) of a subdural haemorrhage preceding Kaleb's final collapse. Dating the changes was imprecise, but they appeared to be some weeks of age. According to Professor Wester, the disclosure of subdural membranes is of the utmost importance as organised membranes only occur in chronic subdural haematoma and take a long time to develop. This must mean that Kaleb had a chronic subdural haematoma, not a hygroma, for a long period prior to his collapse. According to Professor Wester, an acute subdural haematoma on the background of a chronic subdural collection (haematoma or hygroma) is the typical appearance of a subdural haematoma as a complication to BESS.

339 There are many points of disagreement between Professor Wester on the one hand and Drs Tully and Iles and Professor Ditchfield on the other, concerning the diagnosis and consequences of BESS, both generally and in relation to Kaleb specifically.

340 Generally, there is disagreement as to whether subdural haemorrhages are 'strongly' associated with BESS and, more significantly, as to whether BESS can be the cause of retinal haemorrhages and, specifically, widespread multi-layered retinal haemorrhages. There is also a disagreement as to whether BESS is a serious condition capable of causing dramatic collapse and death.

341 More specifically, there is disagreement as to whether Kaleb had BESS, based on whether or not he had the necessary radiological signs, and whether BESS was capable (through the production of intracranial pressure or otherwise) of causing the bleeding

on the brain and the retinal haemorrhages found on autopsy, and the respiratory and cardiac collapse that ultimately killed Kaleb.

342 Professor Wester's report dealt with the evidence given by Dr Tully and Dr Iles at trial, as well as Professor Ditchfield's 2018 statement. In their evidence, Dr Tully and Professor Ditchfield considered the possibility that Kaleb suffered from BESS, but rejected that diagnosis as a realistic possibility.

343 Apart from the fault that he finds with the methodology and epistemological basis for Dr Tully's diagnosis of AHT, Professor Wester says Dr Tully is wrong to state that the typical MRI features of BESS were not present in Kaleb's case and that BESS does not usually cause symptoms of vomiting, drowsiness and poor feeding. As for the absence of typical MRI features, Professor Wester states that Professor Ditchfield's conclusion in this statement that Kaleb did not have BESS is based on incorrect measurements of the intracranial distances showing on the MRI scan of 15 January.

344 Professor Wester also says that Dr Tully is wrong to state that babies with BESS are normally born with heads that are average to large, she is wrong to say that infants with BESS do not have an increased risk of subdural haemorrhage and she is wrong when it comes to retinal haemorrhages. Infants with BESS have been diagnosed with retinal haemorrhages. Retinal haemorrhages have also been demonstrated following ALTE and after cardiopulmonary resuscitation. Kaleb most likely had a BESS condition that caused an ALTE and he was resuscitated. He therefore had three independent reasons for developing retinal haemorrhages.

345 Professor Wester took further issue with Dr Tully's statement that Kaleb seemed at the relevant time 'to be his normal self (as he had been for the preceding month)', as Kaleb had not been well for the preceding month. According to Professor Wester, Kaleb had exhibited symptoms and findings compatible with hydrocephalus and increased intracranial pressure since 28 December 2015.

346 It was Professor Wester's overall contention that Dr Tully discussed and excluded the wrong sequence of causalities. The correct sequence, according to Professor Wester, is that BESS may cause subdural haematomas, which have been associated with retinal haemorrhages and epileptic fits (because of both increased intracranial pressure and blood components irritating brain cells in the cortex, causing seizures), and seizures may cause respiratory arrest (apnoea).

(b) Dr Tully's and Professor Ditchfield's response to BESS diagnosis

347 Dr Tully and Professor Ditchfield opined that Kaleb did not have BESS as the radiological signs of BESS were not present in the MRI scan conducted on 15 January. Furthermore, any rebleed of a subdural haematoma could not account for the catastrophic collapse and resulting injuries suffered by Kaleb on 23 January.

348 The experts agreed that whether Kaleb had BESS was to be determined by reference to both radiological and clinical signs. However, Dr Tully and Professor Ditchfield considered that without radiological evidence, the clinical signs alone were not determinative of BESS.

349 As discussed, Professor Ditchfield was asked in August 2018 to give his opinion as to whether there was any radiological evidence of BESS.

350 Professor Ditchfield answered this question by reference to the MRI scan performed on 15 January, and had regard to both axial and coronal images. He identified subdural collections on both sides of Kaleb's brain. He then measured the subarachnoid space on both axial and coronal planes, making seven measurements in total. Those measurements did not include sinocortical or inter-hemispheric measurements. The measurements were taken from the surface of the brain to the edge of the subdural collection on each side. They were almost all less than 5 mm, with an occasional measurement at 5 mm.

351 In considering whether there was any radiological evidence of BESS, Professor Ditchfield:

- defined BESS as an enlargement of the subarachnoid space that occurs in infants;
- commented that the diagnosis is often made by a subjective assessment of the extra-axial spaces; and
- stated that there is no agreed depth of the subarachnoid space required to make the diagnosis, but that most authors in the literature use a definition of greater than 5 mm to make this diagnosis (referring to Kleinman 3rd Edition[164]). He then said:

> My subjective assessment is that the subarachnoid space is within the normal range for his age. By measurement … the subarachnoid space largely measures less than 5 mm with an occasional measurement being at 5 mm. My assessment is that this MRI demonstrates a subarachnoid space at the upper range of normal and does not demonstrate BESS.

352 To repeat, Professor Ditchfield said that while there is no agreed depth of subarachnoid space required in order to make a diagnosis of BESS, most authors use a definition of greater than 5 mm and, despite this measurement often being within the normal range, Professor Ditchfield himself utilised 5 mm as the minimum required in order to ensure that even a remote suggestion of BESS could be properly identified.

353 In his report, Professor Wester agreed that the radiological criteria for the diagnosis of BESS differed between publications, depending on the imaging modality, the age of the child, and the selection of static population with regards to head circumference. He stated that the upper limit above which craniocortical width ('CCW') is likely to be abnormal ranges from 4–10 mm and the corresponding ranges for sinocortical width ('SCW') are 2–10 mm. For interhemispheric distance, the range is 6–8.5 mm. However, no validated normal values exist and thus the cut-off phase may differ between radiologists.

354 Notwithstanding this, Professor Wester criticised Professor Ditchfield's conclusions regarding BESS on the ground that they were based on a false premise. According to

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164 See Paul K Kleinman, *Diagnostic Imaging of Child Abuse* (Cambridge University Press, 3rd ed, 2015) 422.

Professor Wester, Professor Ditchfield did not measure the distances from the cerebral cortex to the inside of the skull as required.

355 There was disagreement between Professor Wester and Professor Ditchfield as to how the measurements of the subarachnoid space should be made for the purpose of deciding whether Kaleb met the radiological criteria for BESS. While Professor Wester agreed that the assessment of whether the subarachnoid space is abnormal is a subjective one, he said that it was necessary to measure the SCW, the CCW and the interhemispheric distance ('IHD'), and emphasised the importance of capturing the CCW from the cerebral cortex to the inside of the skull rather than to the arachnoid membrane.

356 As a result, Professor Wester opined that Professor Ditchfield's measurements for CCW, which were from the cerebral cortex to the arachnoid membrane (that is, to the edge of but not including the subdural collection) rather than to the inside of the skull, were incorrect. In Professor Wester's view, the subdural compartment had to be included in the measurement. According to Professor Wester, Professor Ditchfield should also have taken a SCW measurement from the cortex to the sagittal sinus and, further, that the measurement should have been taken on the other side and at the point of the largest distance, which would have given a measurement above 10 mm. Had measurements been taken of the proper distances, the SCW and IHD would have been above 10 mm and the CCW would be close to 10 mm.

357 Professor Ditchfield pointed out that in his evidence, Professor Wester repeatedly conflated or confused the subdural space (created by the subdural collections) with the subarachnoid space. BESS, as the name suggests, is the enlargement of the subarachnoid space. Nevertheless, in the course of his oral evidence, Professor Ditchfield sought to demonstrate that even when measuring as Professor Wester had suggested, none of the measurements would be above 10 mm. Professor Ditchfield carried out a number of measurements in the courtroom to make this point. We accept that these measurements were accurate, even though they were conducted on a laptop computer in the awkward confines of a small witness box.

358 Professor Wester's critique of Professor Ditchfield raised as an issue the application of data from a study of subarachnoid spaces in infants carried out in 2001 known as the 'Lam study'. The Lam study measured the subarachnoid spaces in 278 healthy infants in order to produce normative data about the subarachnoid space in infants. Measurements were taken using ultrasound on the coronal plane at the level of the foramen of Monro.[165] Mean widths were then plotted against age. The Lam authors proposed the normal upper limit of the subarachnoid space to be the 95th percentile of the regression curve. In other words, below the 95th percentile was not considered to be pathological.

359 In his rebuttal report, and in response to Professor Wester's criticism that he had not used Lam measurements and data, Professor Ditchfield measured Kaleb's IHD and SCW using the MRI taken on 15 January: the IHD, measured at the narrowest point between the hemispheres, was 4.9 mm; the SCW was measured at 3.3 mm. These values, along with a measurement of 4.5 mm for the CCW, were then plotted on what

165 The foramen of Monro is a short conduit between the paired lateral ventricles and the third ventricle of the brain.

Professor Ditchfield described as 'graphs of normative data' — which were the graphs produced by Lam — to demonstrate that Kaleb's CCW and SCW were below the mean for his age and his IHD was minimally above the mean.

360 Nonetheless, there remained a significant disagreement between Professors Ditchfield and Wester as to:

(a) whether subdural haematomas should be included in the measurement of the subarachnoid space;

(b) the meaning of the Lam data and how it can be used; and

(c) when and how to diagnose BESS.

361 As Kaleb had subdural collections (haematoma or hygroma), a key point of difference between Professor Wester and Professor Ditchfield was as to whether the subdural collections should be included in the measurement. Professor Ditchfield agreed that in the absence of a subdural collection, the measurements would be taken from the cerebral cortex to the inside of the skull, because the subdural space in that case is negligible. However, where a subdural collection is present, including the subdural compartment will artificially inflate the measurements and BESS will be diagnosed inappropriately. Children with subdural haematomas will be diagnosed with BESS when there is no abnormality in the subarachnoid space.

362 Professor Ditchfield relied upon an image reproduced in one of the scientific papers referred to by Professor Wester, a paper by McNeely et al,[166] to demonstrate that even where the subdural collection is on one side only, it can have an impact on the subarachnoid space on the other side, causing the size of that space to nearly double. This is because a subdural collection impedes the reabsorption of CSF. Professor Ditchfield also gave evidence that the IHD may be affected by the presence of a subdural collection elsewhere. In fact, he identified a collection in the interhemispheric space in Kaleb's head.

363 When it was put to Professor Wester that the subdural collection would artificially widen the IHD at the point that he proposed for measurement, Professor Wester denied there was any subdural haematoma or hygroma between the hemispheres in Kaleb's brain.

364 Professor Wester maintained his position that the measurement of the space on the right hand side of Kaleb's MR image should have continued through the hygroma/haematoma from the cerebral cortex to the skull. He said that if the distance had been altered by the subdural haematoma, it would have compressed the surface of the brain and the subarachnoid space so that the CSF would have been squeezed out of the subarachnoid space. This did not occur because there was an even distribution of intracranial pressure inside and outside the brain. The arachnoid membrane is very thin and when it stretches, it is easy for the CSF to leak out into the subdural space.

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166 P D McNeely et al, 'Subdural Hematomas in Infants with Benign Enlargement of the Subarachnoid Spaces Are Not Pathognomonic for Child Abuse' (2006) 27(8) *American Journal of Neuroradiology* 1725.

365 Professor Ditchfield responded that such a leak of fluid outside the usual anatomical compartment into another anatomical compartment would be pathological and that the CSF ceases to be benign once it leaves the subarachnoid space. He did not accept that the distance to be measured would be the same because the fluid had simply moved from one extra-axial space to another.

366 There was a further difference of opinion between Professor Ditchfield and Professor Wester as to where the IHD measurement should be made.

367 Professor Wester was particularly critical of Professor Ditchfield's measurement of the IHD (at 4.9 mm), arguing that the measurement needed to be taken at a wider point between the brain hemispheres and on the coronal plane. According to Professor Wester, measurement of the IHD taken at the widest point (1 cm lower than the point at which Professor Ditchfield's measurement was taken) would have complied with the Lam study and would have doubled or even trebled the measurement that Professor Ditchfield obtained.[167] Professor Ditchfield responded that he had measured the IHD where it is usually measured. Measurement at the point he chose is more reproduceable because it involves two convex surfaces as opposed to the more complex anatomy further down between the hemispheres. Lam only measured the IHD further down because when using ultrasound (which is less accurate than MR imaging), the subarachnoid space was virtually unmeasurable at the higher location. Most current publications measure the IHD higher up and Professor Ditchfield pointed out that Professor Wester had done the same in his report.

368 In his oral evidence on the *voir dire*, Professor Ditchfield agreed that he had not carried out the IHD measurement 'according to the Lam protocol'. He then measured the IHD at the lower point at 6.92 mm.

369 Professor Ditchfield's evidence was that the application of Lam measurements in this instance, and more generally when using more sophisticated imaging technology than the ultrasound available at the time of the Lam study, is inherently problematic and that Lam data has to be used in a qualified way. Lam contributes to the literature; it does not set international standards. While there has been nothing to replace the Lam data, that does not mean that it sets 'the' standard.

370 Professor Ditchfield outlined the limitations of the Lam charts of normative data. The data are relatively old (measurements were taken from 1996 to 1998) and the technology has improved to include MR imaging that allows measurements to be taken from different points, as there is less artifact from the imaging process. According to Professor Ditchfield, the normative data taken from MR images will be significantly greater than the normative data derived from ultrasound. Plotting the data from MR imaging onto the Lam graphs is therefore flawed: it is not quite 'comparing apples and oranges' in that there is still a normative curve, but it is necessary to take into account that the Lam data will set the threshold too low for abnormality and overstate the incidence of BESS. Furthermore, there is no validity to applying the Lam measurements

[167] He also stated that the measurement should have been taken on the coronal plane and not the axial plane.

to patients with subdural haematomas as the Lam data was derived from patients without subdural collections.

371 According to Professor Ditchfield, using the Lam data in this case is problematic, but even if the Lam data are used, Kaleb's measurements are less than the threshold set by Lam for abnormality. Professor Wester did not use the Lam data in a way that they should be used scientifically. The findings of Lam are that a normal CCW (in an infant of Kaleb's age) can be up to 9 or 10 mm. Professor Ditchfield explained that in his medico–legal statement (his 2018 statement) he used the conservative measurement of 5 mm, which Kaleb did not reach. Lam itself sets the threshold low for when the subarachnoid space is above the 95th percentile. Even using the Lam data and the low threshold, Kaleb's subarachnoid space did not fall within the 95th percentile.

372 In his evidence, Professor Ditchfield referred to a paper by Libicher and Tröger[168] from 1992 (published 10 years before the Lam paper) to illustrate how improvements in technology can affect the data and how it can be used. Because artifact was greater in the technology used in 1992, the normative data and the threshold that Libicher established for the 95th percentile was even lower than Lam — its threshold for the 95th percentile was about 6.3 mm, which is much lower than the Lam threshold. If Libicher's normative data is used, the 95th percentile is reached at 6 mm, which is not appropriate when modern imaging technology is used. Professor Ditchfield's point was that the literature varies (and dates) and the radiologist must use his or her expertise to determine where the threshold should be set having regard to the information available. For Kaleb, Professor Ditchfield set the threshold low, but even using a low threshold, Kaleb did not have the radiological signs of BESS.

373 Professor Ditchfield agreed that where a measurement is compared with Lam data, it should be taken on the coronal plane. However, MRI measurements will usually also be taken on the axial plane. This provides a more holistic assessment, as more of the subarachnoid space can be considered. Furthermore, MR imaging is better than ultrasound at demonstrating the difference between the subdural and subarachnoid spaces.

374 However, Professor Ditchfield denied that he took a number of the measurements on the axial plane while purporting to apply Lam. He said that he took a number of measurements on the axial plane in his initial statement, which made no mention of Lam. He did not call them measurements of the CCW but measurements of the subarachnoid space. Professor Ditchfield emphasised that he did not purport to apply the Lam data in his 2018 statement.

375 As to whether, if Kaleb did have BESS, it was capable of causing his death, Dr Tully opined that BESS would not explain Kaleb's earlier symptoms, his sudden collapse and the wide-spread retinal haemorrhages that were found. It explained neither his symptoms prior to his admission nor his clinical presentation upon admission on 23 January. Dr Tully opined that Kaleb did not die as a result of his head 'growing too big' and he did not suffer his collapse on 23 January as a result of his head growing

168 Martin Libicher and Jochen Tröger, 'US Measurement of the Subarachnoid Space in Infants: Normal Values' (1992) 184(3) *Radiology* 749.

faster than it should. According to Dr Tully, Kaleb's collapse was a new event. No pre-existing condition was responsible for his collapse.

376 Dr Tully opined that BESS does not generally cause clinically significant symptoms and does not result in fatality. Furthermore, there is no good evidence that BESS is associated with raised intracranial pressure, wide-spread retinal haemorrhages or fatalities. In her rebuttal report, Dr Tully said that BESS could not explain the presence of 'subarachnoid and parenchymal haemorrhages in addition to wide-spread multi-compartment [subdural haemorrhages]'. Nor could it explain a 'sudden and very rapid accumulation of intracranial fluid and [the] very rapid onset of raised [intracranial pressure]'. Further, BESS could not explain brain swelling, or cardiorespiratory arrest caused by an ALTE.

377 According to Dr Tully, Kaleb's final presentation was not a result of chronic raised intracranial pressure, which could not cause death or collapse. The raised intracranial pressure that he suffered was a result of brain swelling consequent upon 'hypoxic ischemic brain injury'. Any raised intracranial pressure suffered by Kaleb on his first admission was a result of the presence of a subdural hygroma. The raised intracranial pressure seen on 23 January involved a different process.

378 Dr Tully said that while babies with BESS have an increased risk of subdural haemorrhages, they are not at risk of neurological collapse, retinal haemorrhages and multi-compartment subdural haemorrhages. Subdural haemorrhages associated with BESS are generally bilateral, at the front and generally small and asymptomatic. Dr Tully's understanding of Kaleb's situation was that he had 'multi-compartment subdural haemorrhage' that included the cranial fossa, which is not typical of BESS. She said that if a child had seizures and an MRI showed an enlarged subarachnoid space, then she might diagnose BESS. However, if the MRI was otherwise normal, she would continue to look for alternative causes for the seizures, because to diagnose BESS alone would be 'negligent practice'. Kaleb had bilateral multi-compartment subdural haemorrhages which were acute and recent. Further, he had areas of recent subarachnoid haemorrhage and recent intraparenchymal haemorrhage (ie in the brain tissue itself). Additionally, he had multi-layered, bilateral retinal haemorrhages that were too numerous to count. According to Dr Tully, there is no evidence that raised intracranial pressure could have caused those injuries.

379 Dr Iles agreed with Dr Tully's opinion. In her rebuttal report she stated that 'BESS is considered to be a benign condition that does not result in catastrophic collapse, multi-layered retinal haemorrhages and death in infants'. She said she was not aware of any convincing evidence that BESS causes death in infants.

380 To similar effect, Professor Ditchfield said that there was no evidence that a rapidly developing external hydrocephalus in the setting of BESS could be complicated by subdural haematomas and lead to an ALTE.

381 Professor Wester disagreed with the opinions of Dr Tully, Dr Iles and Professor Ditchfield that BESS complicated by a subdural haematoma would not result in fatality. He referred to 'quite a lot of evidence' to the contrary, and to 'many examples in the literature of external hydrocephalus complicated with subdural haematomas that cause

death'. In re-examination, the applicant tendered a series of articles and literature, some of which were said to refute the notion that BESS is a benign condition.

(6) Alternative diagnosis: Rebleed of hygroma

382 The objective of Professor Högberg's report was to address the question of whether there may have been circumstances during Kaleb's foetal period, birth, neonatal period or early infancy that could be related to the symptoms and findings of the subdural hygroma diagnosed during his admission between 14 and 17 January 2016 and his final collapse.

383 Among other things, Professor Högberg examined: Kaleb's neonatal record and 'green book'; maternal and child health care notes; charts of his head circumference, length and weight for age; his MRI records; the radiographic skeletal survey and whole-body CT examination carried out post-mortem; Dr Iles' autopsy report; the confidential medical report prepared by Drs Tully and Collins; and Professor Ditchfield's statements. However, he did not have access to Kalen's antenatal records or records concerning Kaleb's birth, including ultrasound measurements during pregnancy and the delivery records. Thus he was unable to assess risk circumstances preceding those revealed at birth.

384 In relation to Kaleb's birth, Professor Högberg reported an induced labour due to symptoms of decreased foetal movements. The records indicated that Kaleb's condition late in the foetal period was fragile and he did not sustain normal labour contractions. However, he was term-born and showed no asphyxia at birth. He was small for his gestational age, indicating that he had suffered from intrauterine growth retardation. At three months, Kaleb's weight and length had increased from the 5th percentile to the 75th percentile, representing catch-up growth. His head circumference had increased from the 5th percentile to the 95th percentile, which was pathological.

385 Dr Högberg stated:

> It is likely that Kaleb's growth retardation at birth contributed to [his having] symptomatic subdural hygromas with increased intracranial pressure, as seen on 14.01.16, and respiratory arrest on 23.01.16, due to subdural and subarachnoid haemorrhages with increased intracranial pressure, encephalopathy and retinal haemorrhages.

386 He also stated:

> Subclinical, asymptomatic [subdural haemorrhages] are very common in new-borns. Occasionally, they can remain as subdural hygromas. The localisation of [a subdural haemorrhage] cannot be used to distinguish between [a subdural haemorrhage] that has arisen during birth and one that has arisen later. Current knowledge on [subdural haemorrhages] indicates that they generally disappear, but there are exceptions, and more research is needed.
>
> Kaleb's symptomatic subdural hygroma, diagnosed on 14.01.16, may have been birth-related. Kaleb's rapid deterioration on 23.01.16 may have been a re-bleeding from the underlying subdural hygromas. Such bleeding could occur spontaneously or due to minor trauma.

387 Professor Högberg concluded that no certain answer could be given as to whether the forces caused by traumatic shaking on their own could lead to subdural haemorrhage and, if so, after how much time. He agreed with the assessment in the SBU Report that current knowledge is uncertain. Kaleb did not have injuries to the neck, cervical vertebrae or spinal cord, or grip marks on the ribcage, which disaffirmed that traumatic shaking had occurred.

388 Professor Högberg opined that Kaleb's symptomatic subdural hygromas may have been related to birth. Current knowledge indicates that it is probable that Kaleb's intrauterine growth retardation was a strong risk factor for developing subdural hygromas, giving symptoms of increasing intracranial pressure. He continued:

> It is likely that his growth retardation at birth contributed to have symptomatic subdural hygromas with increased intracranial pressure, as seen on 14.01.16, and respiratory arrest on 23.01.16, due to subdural and subarachnoid haemorrhages caused by increased intracranial pressure, encephalopathy and retinal haemorrhages.
>
> Current evidence-based knowledge indicates that there is limited scientific support for [subdural haemorrhage] after traumatic shaking, and that there is insufficient scientific support for assessing the diagnostic accuracy of identifying traumatic shaking based on [subdural haemorrhage], retinal haemorrhage, and encephalopathy.
>
> Based on current knowledge, it cannot be claimed that Kaleb's symptomatic subdural hygromas with increased intracranial pressure and collapse one week later with subdural and subarachnoid haemorrhages with increased intracranial pressure, encephalopathy, and retinal haemorrhages were caused by SBS/AHT. Kaleb did not have injuries to the neck, cervical vertebrae, or spinal cord, or grip marks on the ribcage, which also disaffirms that traumatic shaking had occurred.
>
> It is not likely that the diagnosed extensive bilateral retinal haemorrhages and haemorrhages in the optic nerve sheaths of Kaleb could be related to birth. Based on current knowledge, it cannot be claimed that the retinal haemorrhages were caused by SBS/AHT. It is more probable that the extensive bilateral retinal haemorrhages and the haemorrhages in the optic nerve sheaths were caused by the intracranial pathology with increased intracranial pressure.

(a) Dr Tully's and Dr Iles' response to the rebleed diagnosis

389 Dr Tully described Professor Högberg's proposition that a birth-related subdural haemorrhage rebled almost four months after Kaleb's birth resulting in his sudden catastrophic collapse and death as 'speculative', without pathophysiological basis and unsubstantiated by the evidence.

390 The theory underpinning rebleeding of subdural haemorrhages is that during the process of resolution of an acute subdural haemorrhage, neomembranes may form within which can be seen a network of very fine blood vessels. These frequently bleed spontaneously or from very minimal trauma resulting in small areas of acute haemorrhage in the same location as the original subdural haemorrhage. According to Dr Tully, the best available evidence suggests that the rebleeding of a subdural haemorrhage is not associated with

symptoms and does not cause catastrophic collapse. If these features are present, any areas of recent subdural haemorrhages in association with areas of chronic (older) subdural haemorrhages are more likely to have been caused by a significant episode of recent trauma than be a spontaneous rebleed from membranes associated with a pre-existing chronic subdural haemorrhage.

391 According to Dr Tully, if Professor Högberg is correct, the outcome he describes might be expected to occur with regularity and would most likely have been previously described. In addition, so Dr Tully maintains, Kaleb's subdural and subarachnoid haemorrhages did not put pressure on his brain. His raised intracranial pressure resulted from brain tissue swelling. According to Dr Tully, rebleeding does not explain Kaleb's catastrophic neurological deterioration, severe brain swelling, and recent intraparenchymal and subarachnoid haemorrhages. These features indicate significant brain injury as a result of direct trauma to brain tissue.

392 Dr Iles agreed that birth-related subdural haemorrhages are common. The majority resolve quickly; some leave residual thin membranes. Rebleeding from microscopic-calibre delicate capillaries in organising (ie healing) subdural haematomas is recognised to occur. On rare occasions this can result in clinical symptomology in the setting of space-occupying acute on chronic subdural haematomas.

393 However, Dr Iles opined that in the absence of a space-occupying subdural haematoma, there is no pathophysiological link between low-pressure rebleeding from capillaries in a subdural membrane and catastrophic collapse resulting in irreversible and fatal brain injury. She referred to a study by Bradford et al[169] which reported that, of 105 children with traumatic subdural haemorrhages, 17 (16 per cent) were found to have rebleeding; and the rebleeding identified was small in amount, only identified on imaging, and none of the children had symptoms.

394 Furthermore, according to Dr Iles, there is no evidence that rebleeding from organised (ie healed) birth-related subdural haemorrhages is in any way clinically significant, if it occurs at all. Spontaneous bilateral rebleeding from capillary-size vessels is exceedingly unlikely, and if this did occur, would be of limited clinical consequence, and would not precipitate Kaleb's sudden collapse and respiratory arrest. In addition, bleeding from a chronic subdural membrane would not result in subarachnoid haemorrhages.

(7) What is the consequence of the new/fresh evidence?

395 The evidence submitted by the applicant to justify an acquittal or to require a re-trial of the applicant can be described as follows:

(a) Professor Eriksson's evidence about the SBU Report and its consequences for diagnosing AHT. Professor Wester and Professor Högberg also gave evidence about the pitfall of circular reasoning and the epistemological problems associated with relating the triad injuries to shaking or AHT;

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169 Ray Bradford, Arabinda K Choudhary and Mark S Dias, 'Serial Neuroimaging in Infants with Abusive Head Trauma: Timing Abusive Injury' (2012) 12(2) *Journal of Neurosurgery: Paediatrics* 110.

(b) The studies conducted after the SBU Report reviewing cases in light of the epistemological failings identified by the SBU Report, being principally Thiblin 2020, Wester 2021 and Thiblin 2021. Andersson 2021 also falls into this category;

(c) Professor Wester's evidence that Kaleb most likely died from complications caused by BESS; and

(d) Professor Högberg's evidence that Kaleb may have died from the rebleeding of a subdural hygroma or haematoma.

(a) New or fresh evidence

396 The only parts of this evidence that post-date the applicant's trial are the studies conducted as a consequence of the SBU Report (and the asserted elimination of AHT as a possible explanation for the clinical features that make up the 'triad') that reviewed recorded cases in light of the epistemological failings identified by the SBU Report and the need to find alternative causes for the symptoms and outcomes described.

397 The SBU Report itself was widely available at the time of the trial. By 2017, the SBU Report had generated the work carried out for the Royal College paper. The Consensus Statement was prepared in the same year, albeit not in response to the SBU Report. Furthermore, at the time of the applicant's trial it was well understood that the connection drawn between the components of the triad and AHT was based on indirect evidence and inferential reasoning, as it is impossible to carry out prospective empirical studies to directly confirm the causal nexus between those clinical features and AHT. Over decades there had been, for that reason, an assortment of legal challenges to the validity of evidence of the triad injuries as evidence of AHT.[170] A simple search of the internet would have readily brought up information about those challenges.

398 More specifically, the evidence given by Professor Eriksson about the methodology and findings of the SBU Report was available at the time of the trial and he could have been called to give that evidence to challenge Dr Tully's and Dr Iles' evidence about the cause of Kaleb's death. Professor Wester and Professor Högberg could also have been called to explain to the jury why they contend that the presence of subdural haemorrhages, retinal haemorrhages and encephalopathy cannot be used to diagnose AHT. This would have presented a direct challenge to Dr Tully's diagnosis of AHT, although, as Dr Tully pointed out, 'isolated shaking' and AHT are not co-extensive, and what the applicant admitted to doing to Kaleb did not involve isolated shaking (or was not limited to shaking).

399 The applicant chose not to go down that path. As we observed when considering ground 5, the applicant's defence at trial was that, while Kaleb's death may have been causally related to the manner in which the applicant handled him prior to his collapse on the evening of 23 January, the applicant's conduct (as described by him in his record of interview), while vigorous, did not amount to an unlawful and dangerous act or to

170 See, eg, *R v Harris* [2006] 1 Crim App R 55; *Henderson v The Queen* [2010] EWCA Crim 1269; *Lutze v Sherry*, 392 F 455 (6th Cir, 2010); *SS v Department of Human Services (NSW)* [2010] NSWDC 279; *R v Kumar* [2011] ONCA 120; *Flick v Warren*, 465 F 461 (6th Cir, 2012); *R v Laverdiere* [2014] ABQB 161; *Chief Executive of the Ministry of Social Development v Tilo* [2017] NZFC 2593.

criminal negligence. Kaleb had a pre-existing condition that made him particularly vulnerable to serious injury from 'rough' handling of a kind that fell short of being unlawful and dangerous or criminally negligent.

400 For the reasons that we have stated in our discussion of ground 5, it was open to the jury to reject this argument and to be satisfied beyond reasonable doubt of the applicant's guilt.

401 Furthermore, as we have noted, prior to trial the applicant obtained his own expert reports on the cause of Kaleb's death from Professor Duflou and Dr Collins upon which he ultimately chose not to rely. Professor Duflou's first report (dated 23 February 2017) contains the following commentary on AHT and 'the triad':

> Dr Iles refers to the 'triad' of encephalopathy, subdural haemorrhage and retinal haemorrhage in cases of craniocervical trauma in infants who have been shaken, and correctly refers to controversy in relation to the mechanism whereby these pathological process are produced, as well as whether an impact is required. I agree with those comments. Indeed, considerable controversy remains in relation to mechanisms whereby these pathological processes are produced, whether impact alone can cause such changes, and whether pre-existing pathology, natural or otherwise, can modify the extent and prognosis of injuries. I note that Professor Cordner and others of the Victorian Institute of Forensic Medicine have provided a detailed review of the various issues, and I refer the reader to this important paper commissioned by the Goudge Inquiry into Pediatric Forensic Pathology in Ontario, Canada.
>
> Cordner et al in their report to the Goudge Inquiry refer to the different approaches and views of paediatricians, forensic pathologists and biomechanical engineers. This is also reflected in recent research by Narang et al, indicating that forensic pathologists and biomechanical engineers have views on mechanisms of traumatic head injury in children which appear to differ from those of clinical medical practitioners in multiple respects. Reflecting the unsettled science and general uncertainty in relation to mechanisms and its actual existence as a diagnosis, I note that the Narang study finds that amongst the pathologist respondents less than half accepted the validity of shaken baby syndrome as a diagnosis, a further 30% responded with 'don't know' or 'unsure' and a further 30% did not accept the validity of the diagnosis.[171]

402 The applicant's legal team made a forensic decision not to call Professor Duflou or Dr Collins to give evidence or, apparently, to investigate these matters further.

403 Thus, it was not contended at trial that the extensive bleeding in Kaleb's brain and eyes could not be used as evidence that he had suffered head trauma. We consider this forensic decision to be soundly based. The applicant was confronted at trial with two highly qualified prosecution expert witnesses who, for the reasons we have outlined, concluded that Kaleb was the victim of some form of AHT, although they were uncertain of the forces required to produce the symptoms, conditions and injuries observed. Superimposed upon this formidable prosecution body of evidence was the fact that the applicant's own experts generally agreed with Drs Tully and Iles that Kaleb's death was due to a head injury, with no plausible alternative cause of death, but

171 Citations omitted.

left open the question of the forces required to produce that head injury, as had (at least to some extent) Drs Tully and Iles. The case concept adopted by the defence was, in our view, an entirely appropriate strategy to pursue at trial, and provides a cogent explanation as to why it was not then disputed that the extensive bleeding in Kaleb's brain and eyes was evidence of some form of head trauma. Adducing the Scandinavian evidence at trial would have involved pursuing an entirely different defence case concept.

404 As we have noted, the line between 'fresh' and 'new' evidence is drawn by reference to whether the evidence could have been discovered by the 'reasonable diligence' of the accused.[172] The question is, was the evidence 'evidence of which the accused was unaware and of which he could not have become aware by the exercise of reasonable diligence in preparing his defence'?[173] That is an evaluative exercise and one in which, as Barwick CJ observed in *Ratten*, 'great latitude must … be extended to an accused in determining what evidence by reasonable diligence in his own interest he could have had available at his trial, and it will probably be only in an exceptional case that evidence which was not actually available to him will be denied the quality of fresh evidence'.[174]

405 However, that is not to deny that, as Barwick CJ also observed, an accused must bear the consequences of his or her own decisions as to the evidence to be adduced at trial:

> A trial does not become an unfair trial because the accused of his own volition has not called evidence which was available to him at the time of his trial, or of which, bearing in mind his circumstances as an accused, he could reasonably have been expected to have become aware and which he could have been able to produce at the trial.[175]

406 In this case, we consider that the applicant could with reasonable diligence have obtained the evidence upon which he now seeks to rely. There is no suggestion that the Scandinavian witnesses were unavailable or unwilling to give evidence at the applicant's trial in 2018.

407 The evidence of the Scandinavian witnesses made reference to later studies launched from the epistemological platform provided by the SBU Report. Thiblin 2020 and 2021, and Wester 2020 came into existence after the applicant's trial. Whatever new learning they contain was not available at the time of the trial and, insofar as it constitutes admissible evidence, it might be described as 'fresh'. In our view, however, the expert evidence of Professors Eriksson, Wester and Högberg is not 'fresh' evidence just because they refer to the later studies. Their evidence may arguably be buttressed by the later studies, but the fundament of the evidence before us could have been given at trial. Professor Wester had given evidence to the same or similar effect in the appeals described in the twins paper[176] by the time the applicant's trial took place.

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172 See, eg, *Rodi* (2018) 265 CLR 254, [28] (Kiefel CJ, Bell, Keane, Nettle and Gordon JJ); [2018] HCA 44.

173 *Lawless* (1979) 142 CLR 659, 664 (Barwick CJ).

174 *Ratten* (1974) 131 CLR 510, 517 (McTiernan J agreeing at 524, Stephen J, Jacobs J agreeing at 533); [1974] HCA 35.

175 Ibid.

176 See above [223].

408 Approached on this basis, in order for this 'new' evidence to be consequential, it would have to show the applicant to be innocent, or raise such a doubt about his guilt in the mind of the Court that the verdict should not be allowed to stand. In our view, the Scandinavian evidence does not meet this standard.

409 In this case, there was cogent evidence at trial, which the jury was entitled to accept, that the extensive subdural, subarachnoid and intradural haemorrhages, the extensive bilateral multi-layered retinal haemorrhages and the encephalopathy found during Kaleb's second admission and on autopsy were evidence that he had suffered trauma to the head. There was no suggestion that the trauma was accidental. On appeal, there was comprehensive and, we consider, persuasive, evidence from Drs Tully and Iles, and from Professor Ditchfield, that Kaleb did not have BESS, and that, even if he did have BESS, it would not have caused the major brain bleeds, the extensive retinal haemorrhages and the severe hypoxic ischaemic encephalopathy described. There was persuasive evidence from Drs Tully and Iles that these injuries, in the particular distributions and patterns identified, indicated inflicted head injury.

410 Even if the evidence of the Scandinavian witnesses represents a respectable body of scientific opinion, which we doubt, it would do no more than stand against another respectable body of scientific opinion in the form of the evidence of Drs Tully and Iles and Professor Ditchfield. It would be open to a hypothetical future jury to accept the latter, which would involve rejecting the Scandinavian evidence.

411 The applicant was convicted by a properly instructed jury after an unimpeachable trial. In our view, the Scandinavian evidence, viewed in the context of the evidence led by the prosecution at trial, does not establish that an acquittal would be inevitable if a jury were to consider this evidence. In this regard the observations of Mason J in *Lawless* are apposite:

> [T]here must be powerful reasons for disturbing a conviction obtained after a trial which has been regularly conducted. No such reason for disturbing a conviction presents itself if all that emerges is that the accused has deliberately chosen not to call evidence or that he has failed to search out evidence with reasonable diligence, unless the evidence not called at the trial demonstrates that the accused should not have been convicted of the offence charged. If the evidence newly adduced falls short of establishing that the accused should not have been convicted, there is no overwhelming reason why the conviction, regularly obtained after a fair trial should not be allowed to stand. After all, in a criminal appeal uncomplicated by the existence of newly adduced evidence it is not a ground for the setting aside of a conviction and the ordering of a new trial that the appellate court itself considers that it was unlikely on the evidence that the jury would have convicted. If there was evidence on which the jury could reasonably convict, the verdict must stand, for in such a case there is no miscarriage of justice. So it is when evidence not called at the trial, not being fresh evidence when considered with the evidence given at the trial, leads to the conclusion that the jury could reasonably convict, though it appears to the appellate court that it would be unlikely to do so. There is then no miscarriage of justice because the jury has arrived at a verdict which is unimpeachable and the new evidence produced on the appeal falls short of establishing that the accused should not have been convicted, it being the fault of the accused that

the new evidence was unavailable at the trial.[177]

412 We consider that the Scandinavian evidence falls well short of establishing that the applicant should not have been convicted and that there has been a miscarriage of justice.

413 We observe that the SBU Report makes the valid point that the evidence upon which the nexus between the elements of the triad and AHT is based, being documented cases of children found or suspected to have been subjected to non-accidental (abusive) head trauma, will in many instances contain unstated assumptions about the plausibility of the care-giver's account of the cause of the injury to the infant. In other words, we accept, as do Drs Tully and Iles, that there is some substance to the 'circular reasoning' criticism. That is not to say, however, that the large number of case studies collected over many years across a large number of countries and cultures should be completely cast aside. This is especially so where there is evidence of other physical injuries to the infant consistent with abuse. In many cases the child protection team will have been correct in its assessment that there has been abuse. While caution must be exercised when using the triad injuries for diagnosis, we see no justification for simply discarding all of the evidence, painstakingly built up over decades, of the relationship between the clinical features comprising 'the triad' and AHT. Even with the limited (and arguably idiosyncratic) search terms used by the SBU project team, they identified over 3,700 studies in their review. These, as we have explained, were pared back to only two on the basis of strict inclusion criteria that ignored the difficulties of conducting controlled trials to identify the symptoms of AHT in live infants.

414 Furthermore, as Dr Tully pointed out, there is a body of evidence that does not rely on the kind of reasoning identified in the SBU Report. This body of evidence provides information about the relationship between the triad injuries and head trauma, such as retrospective epidemiological studies as well as advances in clinical, pathophysiological and biomechanical knowledge and understanding informed by animal studies, computational modelling, improved biomechanical modelling and advances in radiological techniques. It is not the case, as the SBU Report and its advocates maintain, that this entire body of knowledge has been simplistically built up on a base of circular logic.

415 We accept the evidence of Dr Tully and Dr Iles that the diagnostic process for AHT is complex and multifaceted and it is to mischaracterise the process to suggest that it involves a near mechanical determination of abuse simply based on the presence of the triad injuries.

416 In short, we consider that the SBU Report is of little assistance in determining whether there has been a miscarriage of justice in this case.

417 As for the evidence concerning the radiological signs of BESS, we accept Professor Ditchfield's evidence that there is uncertainty as to when BESS should be diagnosed, as this accords with both what Professor Wester himself said about the subjective nature of the evaluation and the evidence before us about natural variations in the subarachnoid space in infants, the nature of the available normative data and the adjustments required

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177 *Lawless* (1979) 142 CLR 659, 675–6; [1979] HCA 49.

by advances in imaging technology. Professor Ditchfield's evidence was complicated by his attempt to show that even when using the Lam data, Kaleb's subarachnoid space did not meet the threshold for BESS. That gave the impression that his previous use of the 5 mm figure as the threshold was arbitrary. We do not consider this to be the case: the 5 mm threshold was taken from a well-known textbook and Professor Ditchfield used it because he considered that it reflected a conservative approach to the question he had been asked. We are not persuaded that his decision to use the 5 mm threshold in his 2018 statement can be criticised.[178]

418 Professor Ditchfield did not refer to the Lam data in his 2018 statement, and he told the Court that he did so in his rebuttal report in response to Professor Wester's criticism. In his oral evidence, he repeatedly said that to use the Lam data where a subdural collection was present is 'flawed', as it is based on measurements of healthy infants. In his oral evidence, Professor Ditchfield clearly outlined the limitations of the Lam data and supported his conclusion that the normative data derived from MR images will be significantly greater than the normative data derived from the ultrasound technology used by Lam.

419 Professor Ditchfield concluded that, despite the limitations of the Lam data and the fact that MRI is likely to produce greater measurements than the technology used by Lam, Kaleb was still at the point where approximately 50 per cent of children of his age would have measurements larger than his. This meant that his measurements were not pathological. He remained under the 95th percentile (at around the 75th or 80th percentile) on the Lam charts even with the measurement of IHD taken at a lower point than Professor Ditchfield considered optimal.

420 We do not accept the applicant's criticisms of Professor Ditchfield, whom we consider to be a conscientious and highly qualified expert witness. Professor Ditchfield is a clinical paediatric radiologist who has reported ultrasound, CT and MRI studies of children with head injuries/BESS in tertiary paediatric hospitals on a daily basis for 26 years. In particular, we do not accept that the Lam-based criticisms of his methodology in measuring CCW, SCD and IHD are well founded. As we have said, Professor Ditchfield did not use Lam data in his first report, was criticised for not doing so, and so referred to it in his rebuttal report, but was anxious to point out the limitations of using this data in his oral evidence.

421 We consider Professor Ditchfield's evidence to be persuasive and we accept it. His evidence, in conjunction with the evidence of Drs Tully and Iles on this issue, in our view eliminates as a reasonable possibility that Kaleb died of complications from BESS.

422 Further, and in any event, we consider that a diagnosis of BESS would not negate the powerful evidence that Kaleb suffered a fatal inflicted head injury. Even on the evidence

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178 One of the papers referred to by Professor Ditchfield to justify the position he used for the IHD measurement referred to the three measurements used in the Lam study — CCW, SCW and IHD — and gave a series of thresholds below 5 mm for when subarachnoid spaces are said to be enlarged. However it also referred to a 'more flexible range of measurement' of these parameters as IHD: 6–8.5 mm, SCW: 2–10 mm and CC: 4–10 mm. The paper in question concerned enlarged subarachnoid spaces in a seven-month-old infant, and the measurements were derived using ultrasound imaging, not MRI: Pradeep Raj Regmi et al, 'Benign Enlargement of Subarachnoid Space in Infancy (BESS)' (Case No 16696, European Society of Radiology, 15 April 2020).

of Professor Wester, in order to explain Kaleb's extensive multi-layered retinal haemorrhages (akin to the types of retinal haemorrhages seen following high impact motor accidents), any abnormal enlargement of Kaleb's subarachnoid space would have had to have resulted in the very sudden and severe increase in intracranial pressure associated with Terson syndrome. In other words, Kaleb would need to have suffered from both BESS and the relatively rare condition known as Terson syndrome. Insofar as this requires some kind of intervening event — an 'ALTE' — it demands acceptance of a further level of hypothesis unsupported by empirical evidence.

423 Professor Wester's evidence that the complication of a 'spontaneously occurring subdural hygroma/haematoma' could cause such a chain of events was roundly rejected by Professor Ditchfield, who stated in his rebuttal report that 'there is no evidence that a rapidly developing external hydrocephalus in the setting of Benign Enlargement of the Subarachnoid Space (BESS) can be complicated by subdural haematomas and lead to an Acute Life-Threatening Episode'. In addition, Dr Tully made the point that Kaleb had recent subdural, subarachnoid and parenchymal brain haemorrhages (that is, his brain haemorrhages were not all subdural) and that the presence of subarachnoid and parenchymal haemorrhages in addition to multicompartment subdural haemorrhages suggested a common traumatic cause, not BESS. Moreover, the causal chain posited by Professor Wester completely ignores the striking temporal coincidence of the applicant's admitted 'rough handling' of Kaleb and Kaleb's sudden collapse.

424 It is, we consider, highly speculative to propose such a chain of events in the absence of any evidence of deterioration in Kaleb's condition, let alone dramatically increasing intracranial pressure, in the hours leading up to his collapse. As we know, Kaleb spent the day in the company of a number of people engaging in the types of very ordinary activities that might be undertaken with a baby. He played with his grandfather, he was taken to the shops for lunch. His mother, obviously an intelligent and caring person who was highly sensitive to changes in Kaleb's condition, was sufficiently comfortable to go to work and leave him with the applicant.

425 We note that Dr Tully was questioned about the level of impact that might be necessary to cause retinal haemorrhages in the context of BESS, and she conceded that the effect of a pre-existing brain injury — in terms of brain vulnerability — on later injury was not known and that it was therefore possible that there was an effect. However, she said she was not sure there was any evidence to suggest that the same applied to the eyes. When it was suggested to her that if there was no evidence, then it could not be impossible (excluded), she agreed, but referred to the fact that it was not known whether Kaleb had retinal haemorrhages on admission on 14 January (the first admission). She appeared to be saying that if Kaleb did have retinal haemorrhages on the first admission, it was not known if this could impact on the pattern of retinal haemorrhages found following his admission on 23 January.

426 We do not understand this evidence to be inconsistent with Dr Tully's evidence that BESS would not cause retinal haemorrhages of the severity and distribution found in Kaleb. Kaleb had retinal haemorrhages meeting the criteria described as highly specific for AHT: multi-layered, too numerous to count, extending to the retinal edge and

occurring with retinoschisis.[179] In relation to the last of these criteria, we note that Dr Rodriguez found widespread separation of the neural retina from the retinal pigment epithelium and retinal fragmentation.

427 Dr Tully's evidence about the possibility that Kaleb was vulnerable to retinal haemorrhages might be relevant, if at all, to the defence case concept run at trial, namely, that Kaleb's pre-existing condition made him particularly vulnerable to 'rough handling'. That was a case concept in respect of which Professor Duflou might have given useful evidence, given that he had considered the possibility that Kaleb had BESS. However, it is not how we were asked to evaluate the Scandinavian evidence. The causal chain postulated by Professor Wester did not include rough handling. The applicant relies on the Scandinavian evidence in order to raise as a reasonable possibility that there was an organic cause for Kaleb's death.

428 In our view, the alternative diagnoses proposed by Professors Wester and Högberg are so nebulous and speculative in character as to leave undisturbed the diagnosis of AHT. Their analyses flow from the contestable proposition that the triad injuries are not diagnostic of AHT. As there is no suggestion of significant accidental trauma, it was necessary for them to propose an organic explanation. That explanation is, as we have found, both nebulous and speculative in character and in our view entirely insufficient to displace the evidence of Dr Tully and Dr Iles as to the cause of Kaleb's death.

429 It follows that we do not consider that the evidence of the Scandinavian witnesses would have resulted in an acquittal had it been led at trial or that the applicant has otherwise established that he has suffered a substantial miscarriage of justice as a result of his trial being conducted without the Scandinavian evidence.

(b) No substantial miscarriage of justice

430 It will be apparent that the first part of our conclusion in the preceding paragraph is expressed in the language of the 'new evidence' test, because nearly all the evidence that we have considered fits comfortably within that category. We are mindful that there are 'no absolute hard and fast rules' in considering belatedly acquired evidence and that there will be cases in which the practical guidelines are unjust or unworkable.[180] As we have observed earlier in these reasons, these common law tests are not rigid or inflexible rules and they cannot replace the statutory text of s 276 of the *CPA*. The ultimate question that this Court must assess under this ground is, having considered all the evidence (that is, the trial evidence and the evidence adduced in this application), has a substantial miscarriage of justice been shown to have occurred? For the reasons we have expressed under this ground, the applicant has failed to establish this. In short, we are not satisfied that the evidence called and tendered on the applicant's behalf on the appeal is of the quality asserted. Further, we consider the evidence called and tendered by the prosecution at trial, and by the respondent on this application, to be cogent and reliable — both scientifically and more generally.

431 We are also troubled by the volte-face adopted by the applicant's legal team on this application. It will be an unusual case indeed where an applicant will be able to

179 Retinoschisis refers to the separation of the layers of the retina.

180 *Gallagher* (1986) 160 CLR 392, 395 (Gibbs CJ); [1986] HCA 26. See above [152].

demonstrate a substantial miscarriage of justice by mounting an entirely different case on appeal than was mounted at trial. As we have observed repeatedly, the applicant was competently represented at trial and pursued a sound defence strategy that did not involve challenging the 'triad' hypothesis as part of the causation chain. The Scandinavian evidence, if called, would have been inconsistent with that case concept and, we infer, the applicant 'of his own volition has not called evidence which was available to him at the time of his trial'.[181]

432 Leave for an extension of time in which to file notice of application for leave to appeal will not be granted under this ground, as we do not consider it to be meritorious.

GROUND 4

433 This ground alleges that a substantial miscarriage of justice occurred due to the admission of 'evidence as to the "triad"'. It is submitted that the probative value of this evidence was outweighed by the danger of unfair prejudice to the applicant and thus its admission should have been prevented by s 137 of the *Evidence Act 2008*.

434 Section 137 reads as follows:

> **Exclusion of prejudicial evidence in criminal proceedings**
>
> In a criminal proceeding, the court must refuse to admit evidence adduced by the prosecutor if its probative value is outweighed by the danger of unfair prejudice to the accused.

435 In order to assess the merit of this ground it is first necessary to identify with some measure of specificity (which the applicant does not do) what is meant by 'evidence as to the "triad"'; that is, precisely what evidence the applicant says ought not to have been admitted. As we have seen, the term 'triad' refers to the constellation of findings on which the diagnosis of AHT can be based — subdural haemorrhage or haemorrhages, retinal haemorrhages and encephalopathy. It is not disputed that all three of these symptoms were identified in Kaleb at the time of his death. The applicant presumably does not submit that these medical observations were not admissible evidence; it is therefore presumed that the evidence the applicant says should not have been admitted was the evidence of Dr Tully[182] that these findings supported a diagnosis of AHT in Kaleb's case.

436 The applicant contends that any connection between the triad injuries and the diagnosis of AHT is based on 'junk science', and that the expert evidence that Dr Tully gave 'appears to have qualities of "science" but, in fact, is not "scientific"'. This contention is based largely upon the criticisms of the conventional diagnostic method for AHT made in the SBU Report and which we have outlined in our discussion under ground 3. In brief, those criticisms are that the scientific studies underpinning the perceived diagnostic utility of the triad injuries are beset by circular reasoning and methodological constraints which prevent the scientific validation of the causal connection between

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181 *Ratten* (1974) 131 CLR 510, 517 (Barwick CJ); [1974] HCA 35. See above [405].

182 As well as, presumably, that of Dr Iles, though the applicant does not advert to her evidence at any point in his written submissions under this ground.

forceful shaking and the triad injuries, meaning that the use of that constellation of findings to diagnose AHT lacks a proper scientific basis.

437 It followed, so the applicant contended, that Dr Tully's evidence as to the mechanism causing Kaleb's death had 'very low' probative value, which was outweighed by the danger of unfair prejudice resulting from the jury overvaluing the worth of this evidence.

(1) Consideration

438 In our view, the probative value of the evidence of Dr Tully going to the significance of the triad injuries to the diagnosis of AHT (which was corroborated by the evidence of Dr Iles, though she deferred to the more relevant expertise of Dr Tully on that question) was very high indeed. The applicant mischaracterises the longstanding, established body of scientific literature underlying this evidence as 'junk science'; for the reasons we have given in detail in our consideration of ground 3, it is nothing of the sort.

439 As we have said, the applicant's attack on the scientific validity of the triad simply reproduces that of the SBU Report, which criticises the use of the triad in identifying AHT on the basis of a misrepresentation of its role in the diagnostic process. It will be recalled that the authors of the SBU Report, and the applicant in this application, contend that the existing diagnostic theory is based on a flawed circular process that results in a robotic conclusion of AHT once the three triad injuries are identified.

440 This contention does not address the evidence that was before the jury at trial, which was that a diagnosis of AHT followed from a careful, multifaceted process, which drew on multiple medical disciplines and investigations to eliminate all other causes. The presence of the combination of findings known as 'the triad' is just one aspect of that process. This was the evidence of Dr Tully at trial:

> [AHT is] a diagnosis that needs obviously to be made with care, and with rigorous attention to detail, and arises as the result of a constellation — so a combination of findings — that *include* subdural haemorrhage *of a particular pattern, distribution and location* within the subdural space, … plus evidence of damage to the brain itself, plus retinal haemorrhages … that, again, are in a *particular pattern and distribution, plus exclusion of all alternative causes*'.[183]

441 Once the applicant's attack on the scientific validity of the evidence adduced as to the use of the triad injuries in the diagnosis of AHT is dismissed, the very high probative value of that evidence is clear. It provides a causal link, explained by qualified experts drawing on an established body of scientific literature, between the conduct admitted by the applicant and the fatal injuries sustained by Kaleb. The unfair prejudice alleged by the applicant — that 'a jury may overestimate the potency of this evidence because it is clothed in [a suspect] scientific discourse' — can be dismissed for the same reason.

442 We observe that no objection was made at trial to this aspect of Dr Tully's evidence, or for that matter, to any evidence of Dr Iles as to the role of the triad in diagnosing AHT.

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183 Emphasis added.

While this does not prevent the applicant from raising a ground of appeal based on the admissibility of that evidence now, in the absence of any suggestion of incompetence of trial counsel, that failure to object renders the applicant's task in making out this ground significantly more onerous.

443 Further, it is unsurprising that no such objection was made at trial. As we have outlined earlier in these reasons, the applicant's case at trial focused largely on putting the prosecution to their proof that he had committed any act towards Kaleb which could constitute an unlawful and dangerous act or criminal negligence. Trial counsel's highly competent cross-examination of both Dr Tully and Dr Iles engaged fully with the science underpinning the 'triad-based' diagnostic process for AHT, and to this end established from both witnesses that the requisite level of force is uncertain, especially in the context of Kaleb's pre-existing condition. An objection on the basis of s 137 to the admission of evidence as to the diagnostic connection between the triad injuries and AHT would have been antithetical to the way the case was run at trial. As we have observed in our consideration of ground 3, the purpose of an appeal against conviction is to prevent the perpetuation of a substantial miscarriage of justice. It is not to provide an applicant with an opportunity to run a revised defence honed with the benefit of hindsight.

444 Leave for an extension of time in which to file a notice of application for leave to appeal will not be granted under this ground, as we do not consider it to be meritorious.

GROUND 2

445 The effect of ground 2 is to contend that there was a substantial miscarriage of justice as a consequence of:

(a) Dr Tully denying at trial that there was a scientific controversy as to the utility of 'the triad' as a diagnostic tool in the determination of the existence of AHT when, in fact, there is such a controversy; and

(b) Dr Tully asserting that there was a consensus in the scientific community that the 'triad' can be used to determine the existence of AHT when, in fact, there is no such consensus.[184]

446 These assertions were alleged to be 'incorrect and contrary to [Dr Tully's] obligations as an expert witness'.

(1) Background

447 The applicant contends that the controversy addressed in this ground was exposed by evidence sought to be adduced in this application. This evidence includes the evidence of Professors Högberg, Wester and Eriksson as to the utility of the 'triad' as a diagnostic tool,[185] and PowerPoint slides used by Dr Tully in two lectures given to paediatric

184 Ground 2, and its particulars, are set out in full at [3] of these reasons.

185 This evidence is set out in our consideration of ground 3. We shall refer to it but not repeat it.

trainees and paediatricians on the issue of child abuse.[186] Dr Tully's rebuttal report and oral evidence on this application also bear upon this ground.

(a) Dr Tully — At trial

448 In Dr Tully's cross-examination at trial she stated that the term 'the triad' was a term embracing the three 'features' — encephalopathy, subdural haemorrhages and retinal haemorrhages. This term was not used by her (and others) in a medical context but was used more in a 'legal setting'. She accepted unequivocally that there was 'controversy as to the required level of force to produce this triad of injuries because no biofidelic model can be created'.

449 Later in cross-examination the following exchange occurred:

> I know you'll be able to tell me whether you agree or disagree with any of these propositions quite competently. The pre-existing conditions that the deceased had in this case made him more susceptible to the head injury that he received than a normal healthy infant; correct?---It made him more — possibly made him more susceptible to subdural haemorrhage.
>
> And agree or disagree that as to the mechanism required to produce the triad there is controversy? The triad of injuries, the three things we've been talking about?---This is the problem when you use the term 'triad'. When you have the pattern of subdural haemorrhage, retinal haemorrhage and encephalopathy seen in Kaleb, then I do not believe there is a medical controversy about that diagnosis, no.
>
> Agree or disagree that there is further controversy as to whether impact alone can cause these changes?---Impact alone?
>
> M'mm?---Yes, I don't know that we're clear that impact alone can cause this.
>
> And agree or disagree that there is further controversy as to whether the degree of existing pathology, natural or otherwise, can modify the extent and prognosis of these injuries?---As we've talked about, we don't know the magnitude or degree of force that's required. I think it is possible that, in a child who has a pre-existing enlarged extra-axial space, then we don't know the effect of that on the degree of force required to cause subdural haemorrhage; that's because the bridging veins are theoretically more stretched, because they're going through a bigger space. There's alternative opinion that says they are better buffered by all of the fluid in the space. We simply don't know. What we do know is that a small number of them can develop small subdural haemorrhages without any symptoms either spontaneously or with trivial forces. However, enlarged extra-axial spaces in your head don't affect your eyes, and we know that to cause widespread multilayered retinal haemorrhages requires significant forces.

450 A little later in cross-examination counsel returned to this theme:

> I think where we were was: is it right to say that you can't exclude or can you exclude birth-related subdural haemorrhage in this case?---Yes, I can exclude it.

---

186 These lectures were part of Dr Tully's teaching duties at VFPMS.

> Do you know whether any of the doctors stated words to Erin Baylis-Clarke in or around the 14th to the 17th that a cause of Kaleb's condition may have been birth-related?---Sorry, did I — was I aware of that?
>
> Did you read anything in the medical records that indicate[s] that the doctors may have mentioned the possibility of a birth-related issue?---I can't recall.
>
> If I said to you that there was unsettled science and uncertainty in relation to the mechanisms and the existence of shaken baby syndrome as a diagnosis, you wouldn't accept that?---No, I wouldn't.

451 In re-examination the prosecutor asked Dr Tully to elucidate this last answer:

> There was a question also put as to whether the science in relation to what's described as 'shaken baby syndrome' is unsettled. What opinion do you offer in relation to that proposition?---I don't think there is a medical controversy about — in relation to an infant who presents with very specific features of what has been described in court as the triad when [a] rigorous and accurate medical diagnostic pathway is followed. And, last year there was a very important, we believe, consensus statement that was published by a number of colleges and Royal Colleges throughout America, and Europe and Japan, that provided support in relation to the diagnosis of abusive head trauma. Now, we don't call it shaken baby syndrome, that was something that was — a term that was used years ago, still is used colloquially, but the term that is used is 'abusive head trauma', and that's because of the idea that this constellation or combination of findings is thought to occur as a result of forceful shaking, but also maybe associated impact. So, we don't use the term shaken baby syndrome because it's so specific; we use an umbrella term, abusive head trauma, and there is very well recognised medical consensus opinion that, when done properly, that is a valid diagnosis.

(b) The PowerPoint slides

452 The PowerPoint slides were generated in 2017 and 2019. Neither set of slides was disclosed before trial. For reasons that we shall express, we shall treat both sets of slides as 'fresh' evidence.

453 The full sets of slides can be found in Annexure A to these reasons. In substance, the 2017 slides contained the following statements relevant to this proposed ground:

> Slide 3:
>
> Triad of[:]
>
> - SDH[187]
> - RH[188]
> - Encephalopathy
>
> Slide 20:

187 Subdural haematoma.

188 Retinal haemorrhage.

The controversies surrounding AHT

- The triad cannot be caused by shaking alone
- Forces required to cause injury would damage neck
- Legal perspective — If an infant is shaken what injuries would occur IN THIS case?
- Geddes 'unifying hypothesis' — pathogenesis of SD + RH was hypoxia ischaemia not trauma
- Squires [sic]
- Duhaime and early biomechanical studies

454 There are two citations at the bottom of slide 20:

> Geddes, J.F. et al **Dural haemorrhage in non-traumatic infant deaths: Does it explain bleeding in 'shaken baby syndrome'?** *Neuralpathol Appl Neurobiol* 2003 29:14–22
>
> Squier W **Shaken baby syndrome: The quest for evidence** *Dev Med Child Neurol* Jan 2008 50:10–14

455 The ultimate 2017 slide contains this summary:

- AHT poses many challenges to the clinician
- Careful consideration of other possible aetiology is required
- High quality evidence exists but the area is also plagued by 'non-believers'!
- Strategies to aid prevention are essential

456 The 2019 slides contained the following relevant statements:

Slides 10 and 11

How common is the problem?

- Likely to be a significant **under-estimate**

…

- Unseen, unreported or unknown — these are the ones we know about …

How many are we missing and why are we missing them?

- How many might be below the water? … Denver study
- 31% cases presenting to hospital were 'missed'
- 1 in 3 of these go on to be re-injured
- 4 out of 5 deaths might have been prevented
- Why are we missing them?
- Vague/non-specific symptoms
- No story from carers or child
- Other stories — parental shifting of blame

- Clinicians unwilling to believe

Slides 34 and 35:

The controversies — what the [defence] make of AHT

Does SBS exist

Can only shaking injure an infant's brain

Are the SDH's [sic] and RH's [sic] caused by trauma if there is no other objective evidence

Why is the neck not damaged

The controversy continues …

- 2003 — Geddes: 'unified hypothesis' pathogenesis of SDH + RH was hypoxic ischaemic damage not trauma
  - 'only ever meant to be a theory'
- Dr Waney Squier — need impact
  - Struck off the medical register for misleading the courts
- Biomechanical studies
- The Swedish study
- Confession statements — are they all systematically lying
- Consensus statement

457 The following citation is set out at the bottom of the slide:

> Squier W, Adams L.B. The triad of retinal haemorrhage, subdural haemorrhage and encephalopathy in an infant associated with evidence of physical injury is not the result of shaking, but is most likely to have been caused by a natural disease *J. Prim Health Care* 2011:3(2) 159–163

458 The penultimate 2019 slide contains this summary:

- AHT poses many challenges to the clinician
- Careful consideration of alternative causes
- High quality evidence exists but the area is also plagued by 'non-believers'!
- Recent consensus statement 2018
- Importance of specifics of findings plus supporting 'evidence' especially neck/spine
  - Eye exam EARLY
  - Spinal imaging important

(c) Evidence heard on this application

(i) Professors Wester, Högberg and Eriksson

459 We shall not repeat the evidence of Professors Wester, Högberg and Eriksson, which has been set out extensively under proposed ground 3. It will be recalled that their position was that there was no scientific basis for the association between the presence of the triad injuries and AHT.

(ii) Dr Tully's evidence on this application

460 We have set out significant portions of Dr Tully's evidence in this application in our consideration of proposed ground 3. We shall avoid repetition where possible. In evidence-in-chief, Dr Tully stated that she maintained the opinions that she had expressed in evidence at the trial. This of course included the 'no scientific controversy' evidence that we have set out at [96]–[99] of these reasons.

461 In cross-examination Dr Tully stated that she considered it unnecessary to include in her CV all the lectures that she had delivered. She stated that she had hundreds of presentations on her computer and taught very often, which was part of her role with VFPMS. She considered it unreasonable to expect that she would include all of her many PowerPoint presentations in a CV. She noted that VFPMS held three-day seminars and that she would often conduct 10 or 11 lectures over the course of a seminar, as well as other interactive classes. It did not cross her mind that she may have been under some obligation to provide her teaching sessions, but had she been asked for 'them' (referring to the PowerPoint slides) she would have provided them.

462 Dr Tully stated the following:

- The 'controversies' referred to in the slides 'include[d] the fact that there are perceived controversies within the legal forum and sometimes reflected in the media in relation to … alternative theories around the causation of the findings that are seen in infants with head trauma including inflicted head trauma'.
- The reason she referred to these controversies was that paediatricians may need to appear as expert witnesses in court, and it would be remiss of VFPMS not to acknowledge those alternative theories so that paediatricians can 'understand and therefore fulfil their obligations as … expert witness[es]'.
- She used the term 'non-believers' in quotation marks to indicate that she was quoting others and to capture her audience's attention in the context of a long seminar. It is well acknowledged that there are a small number of researchers and clinicians who do not support the consensus view that shaking or shaking combined with impact can damage an infant's brain.
- She accepted that Dr Squier was removed from the medical register but was subsequently reinstated.

463 Insofar as her trial evidence about the existence or otherwise of a scientific or medical controversy is concerned, Dr Tully was asked on this application this awkwardly expressed question: 'But in terms of the controversy, you now describe there being in effect no controversy with being a medico–legal one, is that right?' She replied:

I don't say there's no controversy. It is my view that there is not a valid[,] legitimate debate in relation to the medical diagnosis of abusive head trauma when the process is done as it should be done. So I … think that's a little different. I acknowledge that there are alternative theories of it but I think what — what we're talking about here is [is] there unsettled science, is there legitimate debate about an issue and so if you use the example of biomechanical data, then there is unsettled science because you have a body of biomechanical research that … suggests that forcible shaking can generate forces that do exceed the tolerance of the bridging veins and neck. You have an alternative … evidence base that suggests maybe that that isn't the case. So that in my view is … unsettled.

In relation … to, can forcible shaking an infant with or without impact result in injury or result in findings that have been described as the triad, then I don't believe there's unsettled science about [those] mechanics because you have a very large body of evidence that supports that association through multiple [disciplines] across the globe in thousands of cases. I am not aware of, and I'm happy to stand corrected, but I'm not aware of any studies, well-designed large studies[,] that are able to refute that association, so the way I interpret that is that there isn't unsettled science.

[COUNSEL:] So at the committal hearing … you were cross-examined and asked this:

> To put it a little more specifically, would you agree that there's controversy over the mechanism that would be required to produce what is called the triad?

You say:

> I don't know about the mechanism but degree of force, yes. Well, yes, actually yes. I mean there is some controversy in the literature. You have to acknowledge controversy in the literature around shaken baby syndrome. However, there is a very large body of well-supported evidence, and a collective forensic paediatric approach to suggest that the triad that we see or the constellation of features that we see is as a result of significant acceleration/deceleration and rotational forces.

That's what you said at the Magistrates' Court at the committal hearing in this case?---Yeah, I accept that.

Yes, and that was on 15 March 2017 and would you accept that now, is that your position that there is this controversy in the literature?---No I think what I'm saying there is that there is controversy about the biomechanical data. I have consistently said there is controversy and unsettled science in relation to biomechanical data.

464 Dr Tully then clarified that, when referring to controversy concerning the biomechanical data, she was referring to the degree of force required to produce the triad injuries.

465 Towards the end of cross-examination, leading counsel for the applicant sought to develop the theme that Dr Tully was a partial advocate who had offered opinions that

had been expressed in near-identical words to opinions expressed by other paediatricians. The following exchange then took place:

> [COUNSEL]: And then if we go to the Daubert analysis, [page] 574:
>
> > As has long been validated, both medically and legally, through the diagnosis of battered child syndrome, if a clinician determines injuries are at variance with the history given regarding the occurrence of trauma, then the clinician can diagnose AHT, not accident[al] trauma with a reasonable degree of medical certainty.
>
> You've adopted that test and put that in your rebuttal report?---No, I mean, I did say when you asked me, is this my opinion and mine alone, I did say this is the consensus view, that from those of us who work in the field and have particular qualifications in this area, that when this process is done correctly, rigorously and following the correct process, then this, the medical diagnosis of inflicted head trauma can be made. And that is what I have, that is my opinion and that is what I have put in the rebuttal report.
>
> I want to suggest, Dr Tully, that what is occurring here is an echo chamber between your research in terms of picking authors or selecting things from people that agree with you. What do you say?---I don't think so. I think I'm … just practising as a clinician, as many other clinicians practise. I don't think there's an echo chamber.
>
> And in my submission you have become an advocate for one side of the debate. What do you say?---I don't think I'm an advocate. I, doing this work … I have to say I would choose not to make this diagnosis. I have sat in front of many families and had to have very difficult conversations. I'm not an advocate for diagnosing inflicted head trauma. I'm a clinician who has expertise in forensic paediatrics and expertise in determining and considering the cause, timing, mechanism of injuries or other things that are … difficult to explain. I don't just work in the field of physical abuse. I also consider children with sexual abuse, neglect, emotional maltreatment in the broad spectrum. So I have no vested interest in diagnosing inflicted head trauma. I am not an advocate for inflicted head trauma. I simply believe that unfortunately child abuse is common, and probably commoner than … a lot of people believe and that we have a need[,] unfortunately, to do this work with a high level of integrity and with a high attention to detail, such that we can provide opinions that best support children and families that are in these difficult situations. So I refute the suggestion that I am an advocate for this condition.

466 Cross-examination concluded with leading counsel asking Dr Tully about her obligations as an expert witness under Practice Note SC CR 3: 'Expert Evidence in Criminal Trials':[189]

> [T]he first [thing] is in the expert practice note from the Supreme Court, expert evidence in criminal trials, at 6.2, Dr Tully, it says:
>
> > Where an expert is aware of any significant and recognised disagreement or controversy within the relevant field of a specialised knowledge, which is

[189] Supreme Court of Victoria, *Practice Note SC CR 3: Expert Evidence in Criminal Trials*, 30 January 2017 ('Practice Note').

> directly relevant to the expert's ability, technique or opinion, the expert must disclose the existence of that disagreement or controversy.
>
> I want to suggest in this case that there was an obligation upon you to recognise and inform [sic] in your report the nature of what I suggest is a significant dispute between scientists and clinical practitioners about this issue. What do you say?---I agree that if I believed there was a … valid controversy about the medical diagnosis or the diagnosis I had made that I would need to include that. I think I've … referred in my report to … the fact that the degree of force [required] to cause certain injuries is … not known but given that I don't think there is … unsettled science in relation to the medical diagnosis of inflicted head injury, then I don't think I'm … under an obligation to do so and that isn't something that is … acknowledged routinely in VFPMS medical legal reports. Where … I don't know, I must say, and I believe that that's what I do. If I don't know the cause[,] mechanism, timing of an injury, [I] say that and that is what I would do.
>
> I suppose my point is those seminars you present indicate that there is a controversy. You might say the other side is providing, you know, inappropriate, insufficient [evidence] but there is I want to suggest a proper controversy which is reflected in your own papers or seminar papers?---[T]he PowerPoint presentation has a … slide entitled, 'Controversies', and I think we visited that before in terms of the reason that is there and I do think it is important to understand when you do this work that there are, as I've said, alternative — a small minority of individuals who offer alternative views but that these are not at current times substantiated by any evidence. So we have a large body of evidence that consistently, reliably and repeatedly demonstrates this association including information that has been gained from people who said what they've done. We don't as yet have any well-designed large studies that refute that association. So I don't think I'm under an obligation where there isn't a study that I can refer to, to … include that information in my [medico–legal] report. I've made a medical diagnosis and that is what is in my report.

(2) Submissions

467 The applicant contended that Dr Tully's evidence in denying a scientific controversy concerning 'the diagnostic utility of the triad' was demonstrably incorrect by reference to the evidence of the Scandinavian witnesses and the PowerPoint slides. The applicant further contended that Dr Tully's reference at trial to a 'consensus' within the scientific community as to this diagnostic utility was incorrect. The product of these two overlapping pieces of evidence was said to be a substantial miscarriage of justice. Within this broad argument the applicant developed a number of themes:

- The PowerPoint slides should have been disclosed prior to trial.
- The reference to a 'consensus statement' in Dr Tully's trial evidence was misleading and required clarification as to the epistemological status of a consensus statement.
- Dr Tully had become an advocate for the 'triad'-based diagnosis of AHT as demonstrated by the reference in the slides to 'non-believers' and her expressed opinion that AHT was likely more common than was detected.

468 In reply to these contentions the respondent submitted that Dr Tully was under no obligation to disclose the PowerPoint slides and that she quite fairly conceded the existence of various 'controversies' associated with the diagnosis of AHT, including as to what level of force was required to produce the triad injuries and whether underlying pathologies could modify that level of force. The statement made by Dr Tully that there was not a medical or scientific controversy concerning the use of the triad injuries as a diagnostic tool was confined to the particular circumstances of this case, and so understood the statement itself is 'uncontroversial'. The respondent further contended that even if the Court were to conclude that Dr Tully had failed to concede a controversy where one existed, this was unimportant in the context of the conduct of the trial, and manifestly insufficient to constitute a substantial miscarriage of justice. The respondent's submissions were made on the basis that all of the evidence relevant to this ground, including the PowerPoint slides, the evidence of the Scandinavian witnesses and Dr Tully's evidence on this application, was 'new' rather than 'fresh' evidence.

(3) Consideration

469 We are of the view that the applicant has failed to demonstrate a substantial miscarriage of justice under this ground.

470 We are not satisfied that Dr Tully was under any obligation to disclose the impugned PowerPoint slides. As referenced at [466] of these reasons, the Supreme Court has issued practice notes on expert evidence in criminal trials. Expert witnesses have an overriding duty to assist the Court by giving impartial and unbiased opinions on matters within their specialised knowledge.[190] Relevantly, an expert *must disclose any significant or recognised disagreements or controversies within their field which he or she knows of and which are relevant to the expert's ability, technique or opinion*.[191]

471 The Practice Note gives 'the other party' (in this case the representatives of the accused) the right to inspect an expert's instructions and material, notes made by the expert in connection with preparing his or her report and a record of any examination, measurement, test or experiment that provided the basis for the expert's opinion.

472 We consider that it is unreasonable to expect Dr Tully to have provided the PowerPoint slides pursuant to the above-cited obligations. Both sets of slides set out topics for discussion at a lecture conducted by Dr Tully that is part of a three-day seminar offered by VFPMS. As we have observed, Dr Tully stated that she had hundreds of 'presentations' on her computer and taught very often. She considered it unreasonable to expect that she would include all of her many PowerPoint presentations in the CV annexed to her report. We agree with this perspective. The slides are not peer-reviewed statements of professional opinion. They are aids to classroom teaching and set out points for elaboration and discussion. Dr Tully explained that paediatricians may be required to appear as witnesses in court proceedings and need to be across theories that may be advanced in the court setting. She further stated that her use of the term 'non-

190 Practice Note, [4.1]–[4.4].

191 Ibid [6.2].

believers' was in quotation marks to indicate that it was a term others had used in relation to a media-driven controversy.

473 Whilst we do not consider that the PowerPoint slides were required to be disclosed prior to trial, it is plain that, in this application, they constitute 'fresh', as opposed to 'new', evidence. We do not consider that the exercise of reasonable diligence by the applicant's practitioners before trial would have disclosed the existence of the slides. These were private teaching tools, neither published nor used in any public way. The defence had no hint of their existence. We also accept that the evidence Dr Tully gave on this application concerning the fresh evidence of the slides must also be characterised as fresh evidence.

474 It follows that, under this proposed ground, the issue devolves to this: If the PowerPoint slides had been before the jury at the trial, together with Dr Tully's evidence concerning them, is there a 'significant possibility' that the evidence would have led a reasonable jury to acquit the applicant?[192] If there is such a possibility, the applicant will have demonstrated a substantial miscarriage of justice.

475 It is a central component of the applicant's submissions under this proposed ground that Dr Tully was not an impartial expert witness but was in fact an 'advocate' for 'the triad' hypothesis, unprepared to admit there was a legitimate disagreement as to its validity as a diagnostic tool. We shall ignore the applicant's assertions that Dr Tully was an advocate for the diagnosis of child abuse in a broader sense. The contention that Dr Tully's 'interviewing' the applicant and Erin Baylis-Clarke and her assistance to police while Kaleb was in hospital meant that she was 'embedded in the police investigation at an early stage' (and, impliedly, aligned with the prosecution) is simply empty rhetoric and does not describe conduct that deviates in any way from that which would be expected of a clinician undertaking a standard diagnostic process in these tragic circumstances. Considering only, then, Dr Tully's alleged advocacy for the triad-based diagnosis of AHT as a scientific theory, it was submitted that, had these slides been in the possession of the defence at trial, a skilful cross-examination could have exposed this.

476 It is unnecessary to indulge in speculation as to how Dr Tully would have dealt with this skilful cross-examination at trial because we observed in this application a skilful cross-examination on this very topic. It is reasonable to assume that, had the slides been in evidence at the trial, Dr Tully would have explained them in a similar fashion, namely:

- There are controversies, but there is no valid, legitimate debate in relation to the *medical* diagnosis of AHT when a rigorous process of investigation and examination is undertaken.
- The lectures were directed to current and future paediatricians who may be required to give evidence in a court setting in future and who would need to be across theories commonly advanced in *that* setting.

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192 See *Bowden* (2017) 54 VR 135, 144 [36] (Priest JA); [2017] VSCA 46; *Lawless* (1979) 142 CLR 659, 675–6 (Mason J); [1979] HCA 49.

- ‘Non-believers’ is a term other people have used, which is why it appears in quotation marks in the slides. It is responsive to a perceived controversy which is media-driven rather than having any significance in the scientific or medical community.
- She was not aware of any well-designed, large studies that are able to refute the association between findings described as the ‘triad’ and the forcible shaking of an infant with or without impact.
- On the other hand, there is a very large body of evidence that supports this association involving multiple studies. There is no unsettled science concerning this association.

477 The same approach can be taken to the applicant’s contention that the new and fresh evidence would have cured any misunderstanding on the jury’s part of the scientific significance of the Consensus Statement when it was referred to by Dr Tully in her evidence at trial. Defence counsel had every opportunity to adduce this evidence from Dr Tully at trial regardless of the availability of the slides or the Scandinavian evidence. If this had occurred, any necessary clarification would have been given. To understand this, we need only look to the answer Dr Tully gave to the applicant’s counsel on this application when challenged on the potential for the term ‘consensus’ to mislead when not placed into a context to understand this:

> At trial though what you tend to do in your evidence is to — when you’re challenged about the science, you refer to this consensus opinion and the fact that it’s been signed, it’s in all these countries with no further qualification or elaboration, is that right?---Well, that’s just an example. It’s … an example of exactly what I’m saying, that there is a very strong medical consensus about this … diagnosis and I think that that is valid because that consensus statement exists and that’s what that says.
>
> But you don’t explain to the jury that a consensus opinion in terms of scientific evidence is at the lowest in terms of science?---If I’d been asked that question, I would have explained … where consensus opinion sits in some rankings and I would have explained my view of the consensus statement but I wasn’t asked that question.

478 As to the contention that the slides would have exposed Dr Tully’s partial advocacy for the triad theory because they disclosed a belief that AHT is more common than is detected or diagnosed, we do not consider that the statements to that effect included in the slides would have led the jury to that conclusion. Those statements simply cite studies and data which support the hypothesis that the AHT is underdiagnosed both in Australia and overseas. They also suggest hypotheses as to why there may be a tendency to misdiagnose cases of AHT presenting to hospitals. In the context of a seminar given to paediatricians and trainee paediatricians, the sharing of such knowledge and analysis does not, in our view, disclose an eagerness to ‘achieve’ more diagnoses of AHT, but rather an effort to assist paediatricians to identify more accurately AHT when it does present. In any event, the evidence Dr Tully gave on this application again provides an answer to what impact the slides may have had in this regard on the trial:

> I simply believe that unfortunately child abuse is common, and probably commoner than … a lot of people believe and that we have a need[,]

> unfortunately, to do this work with a high level of integrity and with a high attention to detail, such that we can provide opinions that best support children and families that are in these difficult situations. So I refute the suggestion that I am an advocate for this condition.

In our view Dr Tully did not argue that AHT is more common than is currently diagnosed in an effort to simply increase the rate of AHT diagnoses, but in order to increase its rate of detection. We think it likely that a jury would have reached the same conclusion, had it considered the slide evidence.

479 We considered Dr Tully to be an impressive witness and we accept her evidence concerning the 'fresh' PowerPoint slides. We do not consider that her expertise or independence were challenged successfully in any meaningful way in this application, either by reference to the slides or (for the reasons we have stated under proposed ground 3) to the 'new' evidence of Professors Wester, Högberg and Eriksson.

480 Further, it is important to understand that in her trial evidence Dr Tully at no stage asserted that there was no controversy as to the diagnosis of AHT based on the existence of triad injuries. Repeatedly and explicitly, she said that there is no *valid scientific or medical* controversy of this nature when a rigorous diagnostic pathway is followed. By qualifying the word 'controversy' in this way, Dr Tully's evidence was a confined expression of expert opinion that, in our view, remained undisturbed by either the Scandinavian evidence, the PowerPoint slides, or the combination of both pieces of evidence.

We are compelled once again to refer to the defence strategy at trial. The existence or otherwise of a controversy in relation to the medical diagnosis of AHT using the triad was irrelevant to the defence case concept. That concept admitted the presence of the triad injuries in Kaleb and their use as a diagnostic tool, but contended that Kaleb's pre-existing intracranial condition impacted the degree of force necessary to produce those injuries. As we have observed, given the opinions expressed by Professor Duflou and Dr Collins, the admissions made by the applicant in his police interview, and the evidence of both Drs Tully and Iles, that strategy was forensically sound. And it was supported, in part, by the concessions made by Drs Tully and Iles that there *was* controversy attached to the degree of force required to produce the triad injuries, particularly in the context of Kaleb's underlying intracranial condition.

It follows that we do not consider there was any significant possibility that, had the PowerPoint slides been in evidence at the trial, together with Dr Tully's explanation for them (as given to this Court), it would have changed the trial outcome. We do not consider that Dr Tully's credibility would have been undermined, and even if it had been, the applicant would still have had to contend with the untarnished and cogent evidence of Dr Iles, his own admissions, and the manner in which the defence case had been conducted.

481 Leave for an extension of time in which to file notice of an application for leave to appeal will not be granted under this ground, as we do not consider it to be meritorious.

(4) Conclusion

482 We decline to grant an extension of time in which to file notice of application for leave to appeal. We consider that none of the grounds of appeal proposed by the applicant are meritorious.

WALKER JA:

(1) Introduction and summary of conclusions

483 This case concerns the tragic death of Kaleb Baylis-Clarke, an infant aged three-and-a-half months. The Crown case was that the applicant, Jesse Vinaccia, who was caring for Kaleb at the time of his collapse, had shaken Kaleb with sufficient force to cause his death. Cases of this kind are not unknown to the law, both here and in the United Kingdom. In *Henderson v The Queen*[193] the United Kingdom Court of Appeal heard appeals concerning babies alleged to have died as a result of forceful shaking. In allowing one of the appeals, the Court said this:

> There are few types of case which arouse greater anxiety and controversy than those in which it is alleged that a baby has died as a result of being shaken. … The controversy to which such cases gives rise should come as no surprise. A young baby dies whilst under the sole care of a parent or childminder. That child can give no clue to clinicians as to what has happened. Experts, prosecuting authorities and juries must reconstruct as best they can what has happened. There remains a temptation to believe that it is always possible to identify the cause of injury to a child. Where the prosecution is able, by advancing an array of experts, to identify a non-accidental injury and the defence can identify no alternative cause, it is tempting to conclude that the prosecution has proved its case. Such a temptation must be resisted. **In this, as in so many fields of medicine, the evidence may be insufficient to exclude, beyond reasonable doubt, an unknown cause. … [E]ven where on examination of all the evidence, every possible known cause has been excluded, the cause may still remain unknown.**[194]

484 As will become apparent, in my opinion this case is of such nature.

485 On the evening of 23 January 2016, Kaleb was found unresponsive in his cot by the applicant, who was caring for Kaleb at the time, alone. The applicant called 000 and administered CPR. When the ambulance arrived Kaleb was resuscitated by ambulance officers and taken to hospital. However, he died seven days later. Medical examination revealed that, while Kaleb had no external injuries, no bruising other than that attributable to medical intervention, and no fractures, he had very serious brain injuries (including subdural haemorrhages) and bleeding in both retinas. Expert medical opinion was to the effect that these injuries must have been caused by violent shaking causing rapid acceleration and/or deceleration of his head, with or without impact. The applicant

193 [2010] EWCA Crim 1269 ('*Henderson*').

194 *Henderson* [2010] EWCA Crim 1269, [1] (Moses LJ for the Court) (emphasis added).

denied having shaken Kaleb, but admitted that he had picked Kaleb up 'with a bit of force' and put him down in his cot 'pretty rough'.

486 There was also evidence that Kaleb had been unwell in the weeks prior to 23 January 2016 and that his head circumference had been rapidly expanding. He had been admitted to hospital on 14 January 2016, where an ultrasound and an MRI found various abnormalities in Kaleb's brain. He was discharged on 17 January 2016.

487 The applicant was convicted of child homicide. The prosecution case at trial was that Kaleb had died of abusive head trauma, inflicted by the applicant by forceful shaking. The prosecution case was based on expert medical evidence from two experts, Dr Tully and Dr Iles. Their evidence at trial was that the presence of subdural haemorrhages, retinal haemorrhages of a particular nature and pattern, and hypoxic ischemic encephalopathy (often referred to as the 'triad'), coupled with the exclusion of alternative causes, led to a diagnosis of abusive head trauma.

488 The applicant engaged an expert, Professor Duflou, but chose not to call him at trial. The applicant now seeks leave to appeal his conviction, in part on the basis of different expert evidence, from Professors Eriksson, Högberg and Wester, who were not called at trial. This Court received written and oral evidence from those witnesses, and further written and oral evidence from Drs Tully and Iles, as well as written and oral evidence from Professor Ditchfield, who had not given evidence at trial, but whose opinion had underpinned part of Dr Tully's evidence at trial.

489 The applicant has advanced four proposed grounds of appeal, set out in the judgment of Forrest and Emerton JJA. In summary, those grounds are as follows:

(a) Ground 2: that Dr Tully 'gave evidence that was incorrect and contrary to her obligations as an expert witness'. The particulars to this ground focused on Dr Tully's oral evidence at trial that there was no 'scientific controversy, or dispute, in the scientific community' about the diagnostic utility of the 'triad' in diagnosing non-accidental head trauma.

(b) Ground 3: that new expert evidence — that is, the evidence of Professors Eriksson, Högberg and Wester — should be admitted on the appeal and that that evidence demonstrates the applicant's innocence or creates a reasonable doubt as to the applicant's guilt because it suggests an alternative cause of death, namely benign enlargement of the subarachnoid space ('BESS').

(c) Ground 4: that Dr Tully's and Dr Iles' evidence at trial concerning the 'triad' should not have been adduced because the probative value of that evidence was outweighed by its unfair prejudice.

(d) Ground 5: the verdict was unsafe and unsatisfactory or cannot be supported by the evidence. The particulars to this ground were that the prosecution had not excluded the reasonable possibility that Kaleb's death was caused by a pre-existing medical condition, independent of the acts of the applicant; and that the prosecution had not excluded the possibility that the acts of the applicant, as described in his record of interview, did not amount to an unlawful and dangerous act or criminal negligence.

490 I have had the advantage of reading in draft the reasons for judgment of Forrest and Emerton JJA. I gratefully adopt their recitation of the facts and the evidence at trial. I also gratefully adopt their description of the evidence given by Professors Eriksson, Högberg and Wester on the appeal, as well as the rebuttal evidence of Drs Tully and Iles and Professor Ditchfield. However, I will add some further comments of my own in relation to the evidence adduced on the appeal and, as will become apparent, I disagree with the conclusions their Honours have reached concerning the characterisation and effect of that evidence.

491 I consider it to be in the interests of justice that the extension of time sought by the applicant be granted.[195] I would thus grant that extension and grant leave to appeal. I would reject ground 5, for the reasons given by Forrest and Emerton JJA. However, I would uphold grounds 2 and 3. In light of my conclusion with respect to grounds 2 and 3, it is not necessary for me to deal with ground 4.

(a) Summary of conclusions on ground 3

492 In summary, I would uphold ground 3 for the following reasons.

493 *First*, in light of the new evidence, I consider that the Crown had not excluded the possibility that Kaleb suffered from a pre-existing condition, BESS, at the time of the events in question. That is because, while there was a controversy about that issue as between Professor Wester and Professor Ditchfield, I have concluded that the flaws in Professor Ditchfield's evidence were such that it would not have been open to the jury to conclude that BESS had been excluded. Once that is accepted, Dr Tully's evidence at trial is necessarily undermined, because she had relied on Professor Ditchfield's evidence as the basis for excluding BESS.

494 *Secondly*, I have concluded that, if BESS is not excluded, then it would not have been open for the jury to conclude that the only explanation for Kaleb's injuries was that he was shaken with significant force. That is because both the Crown experts and the applicant's experts accepted that BESS provides an explanation for the subdural haemorrhages observed in Kaleb; the question then was whether there was an explanation for the retinal haemorrhages observed in Kaleb. In my view the evidence revealed that that was uncertain. In particular:

(a) Professor Wester opined that retinal haemorrhages are not specifically characteristic or indicative of abusive head trauma, and can be caused by other events, including raised intracranial pressure. His evidence was that Kaleb's retinal haemorrhages were attributable to BESS, because of raised intracranial pressure, which is a symptom of BESS, and which Kaleb had prior to the events of 23 January 2016.

(b) Dr Tully accepted that it was 'not impossible' that Kaleb's retinal haemorrhages might have been attributable to a pre-existing medical condition, and to some extent accepted a degree of uncertainty about the cause of Kaleb's retinal haemorrhages (which was not consistent with the evidence she gave at trial).

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195 *Madafferi v The Queen* [2017] VSCA 302, [11] (Priest, Hansen and Coghlan JJA); *Kentwell v The Queen* (2014) 252 CLR 601, 613–14 [30]–[32] (French CJ, Hayne, Bell and Keane JJ); [2014] HCA 37.

(c) Finally, the academic literature concerning the causes of retinal haemorrhages of the nature and pattern observed in Kaleb was equivocal as to whether such retinal haemorrhages could be caused by BESS (or by raised intracranial pressure). While the literature reflected the consensus that such haemorrhaging is rare in non-abused children, it did not exclude the possibility of such a cause. Further, some of the literature was to the effect that the severity of retinal haemorrhages is not perfectly correlated with abusive head trauma, and that other events may mimic the retinal haemorrhages typically associated with abusive head trauma.

495 Thus, in my opinion, the evidence before this Court concerning the connection of retinal haemorrhages with abusive head trauma was not sufficient to support Dr Tully's definitive position at trial that the retinal haemorrhages must have been caused by the application of significant force.

496 *Thirdly*, I consider that, had the jury heard the new evidence, and noting the uncontroverted evidence concerning Kaleb's ill-health prior to 23 January 2016, it would not have been open to the jury to conclude, beyond a reasonable doubt, that the applicant had committed child homicide, either by an unlawful and dangerous act or by criminal negligence.

497 I have also concluded that, even if I am wrong in concluding that the new evidence was such that it would not have been open to the jury to convict the applicant, I would in the alternative conclude that the new evidence demonstrated that a substantial miscarriage of justice had occurred, on the basis that, had the new evidence been before the jury, and accepted by them, there is a reasonable possibility that they would have acquitted the applicant. As I explain later in my reasons, I do not consider that the authorities that distinguish between fresh and new evidence preclude me from reaching that conclusion.

498 I note that I make no positive finding either that Kaleb's death was caused by BESS, or as to the manner in which the applicant handled Kaleb and the level of force involved. Nor do I make any positive finding that the applicant did not shake Kaleb with significant force. Ultimately, when the new evidence is considered, this is a case where the applicant's guilt has not been proved beyond a reasonable doubt. That is because the cause of Kaleb's injuries is uncertain and because, even if caused by the applicant, the level of force required to produce Kaleb's injuries is also uncertain. Thus I do not consider that the necessary elements of child homicide were proved beyond a reasonable doubt. It may be *likely* that the applicant shook Kaleb with such force that he caused his injuries. It may also be *likely* that he did so either by an unlawful and dangerous act or by criminal negligence. But that is not sufficient.

499 It is also important to emphasise that I have not concluded that a diagnosis of abusive head trauma, based on the presence of subdural haemorrhages, retinal haemorrhages of the relevant nature and pattern, and hypoxic ischemic encephalopathy, cannot be made or is inherently unreliable or otherwise inadmissible. That is, I have not concluded that the evidence of Professors Eriksson, Högberg and Wester concerning the reliability of a diagnosis of abusive head trauma based on the 'triad' is to be preferred over the evidence of Dr Tully and Dr Iles on this issue. Rather, my conclusion is based on the new evidence concerning BESS and retinal haemorrhages. That is, my conclusion is squarely based on Kaleb's particular medical history and pre-existing conditions.

(b) Summary of conclusions on ground 2

500 In summary, I have concluded that ground 2 should succeed based on the evidence before this Court of Dr Tully's PowerPoint slides (which were not adduced at trial and had not been disclosed to the applicant). That evidence was fresh evidence, thus the question is whether, had that evidence been before the jury, there is a 'significant possibility' that the jury, acting reasonably, would have acquitted the applicant. In my opinion, there is such a possibility. That is, I consider that there is a significant possibility that, had Dr Tully's PowerPoint slides been in evidence, the jury would have accepted that there is a real (medical) controversy concerning the diagnostic utility of the 'triad'. Dr Tully's certainty as to her diagnosis, and her credibility, could have been undermined. Dr Tully's evidence was the critical evidence that provided a pathway for the jury to convict. Had the defence had available to it material that potentially undermined Dr Tully's evidence, whether as a matter of substance or as a matter of credibility, the prosecution may not have successfully excluded a reasonable hypothesis consistent with innocence.

501 Thus, in my opinion, had the PowerPoint slides been available to the defence at trial, there is a significant possibility that the jury would have acquitted the applicant. In that sense, I am satisfied that there has been a substantial miscarriage of justice, and ground 2 is made out.

(c) Appropriate orders

502 In consequence of my conclusion on ground 3, I would grant leave to appeal, allow the appeal, set aside the applicant's conviction and order that an acquittal be entered.

GROUND 3

503 It is convenient to commence with ground 3. I note at the outset that the reception of some eight days of expert evidence, from six experts, together with the tender of numerous articles and studies published in medical journals, made this a very unusual case. This has resulted in an unusual level of detail being required in order to address ground 3. The nature of the issues has required an unprecedented examination of highly technical issues concerning medical opinions about the cause of Kaleb's symptoms and, ultimately, his death.

(1) 'New' vs 'fresh' evidence

504 As discussed above, ground 3 turns on the evidence of Professors Eriksson, Högberg and Wester, which has been described in detail in Forrest and Emerton JJA's judgment. That evidence was not led at trial. Further, in so far as this evidence was directed to identifying an alternative cause of death, namely BESS, the defence had obtained expert evidence directed to that question from Professor Duflou and had decided not to call him at trial. Ground 3, as originally drafted, was that this evidence should be admitted because it demonstrates the applicant's innocence or creates a reasonable doubt as to his guilt.

505 A preliminary issue in relation to ground 3 is whether the evidence of Professors Eriksson, Högberg and Wester is properly characterised as 'fresh' evidence or 'new' evidence.

506 The courts have long drawn a distinction between fresh evidence and new evidence for the purposes of appeals. Evidence is fresh if it was not available to the defence at trial in the exercise of reasonable diligence. Evidence is new if it was available to the defence at trial in the exercise of reasonable diligence, but was not led. The distinction has been used as the basis for different approaches to the reception and treatment of such evidence on an appeal. The 'test' for allowing an appeal on new evidence is different, and more stringent, than the 'test' for allowing an appeal on fresh evidence. Thus, as a general proposition:

(a) on an appeal against conviction on the basis of fresh evidence, the court may find a miscarriage of justice if there is a *significant possibility* that the evidence, if believed, would have led *the jury, acting reasonably, to acquit*;[196]

(b) in contrast, if the evidence is new evidence (that is, it could reasonably have been obtained for the trial), the appeal will only be allowed if the new evidence shows that the appellant is innocent, or raises for the appellate court a reasonable doubt about the appellant's guilt.[197] The appellate court must review the whole of the evidence (including the new evidence) and decide whether, if the new evidence had been before the jury, the *jury must have entertained a reasonable doubt* about guilt.[198]

507 That is, the difference between fresh evidence and new evidence is that, when evidence is 'fresh', the court will consider whether the jury *might* have entertained a reasonable doubt, if the evidence had been led. In contrast, the test for new evidence is whether the jury *must* have entertained a reasonable doubt, if the evidence had been led.[199]

508 For the reasons that follow, I consider that, regardless of whether the evidence of Professors Eriksson, Högberg and Wester is properly characterised as new or fresh, ground 3 is made out. That conclusion is primarily based on the evidence of Professor Wester that Kaleb had a pre-existing condition — BESS — that caused or contributed to his death, together with the evidence of Dr Tully and Professor Ditchfield on the appeal.

(2) The evidence concerning BESS

509 A key issue raised by the evidence before us on this appeal was whether Kaleb had BESS (also known as benign external hydrocephalus, or 'BEH') and, if so, whether that

196 *Bowden (a pseudonym) v The Queen* (2017) 54 VR 135, 144 [36] (Priest JA, Maxwell P and Kidd AJA agreeing at 137 [1]); [2017] VSCA 46 ('*Bowden*'); *Visser v DPP (Cth)* [2020] VSCA 327, [189]–[194] (McLeish, Emerton and Osborn JJA) ('*Visser*'); *R v Nguyen* (1998) 4 VR 394, 401 (Kenny JA, Winneke P and Callaway JA agreeing at 395).

197 *Bowden* (2017) 54 VR 135, 146 [40] (Priest JA); [2017] VSCA 46.

198 *Bowden* (2017) 54 VR 135, 137–8 [2]–[6] (Maxwell P and Kidd AJA); [2017] VSCA 46; *Visser* [2020] VSCA 327, [189]–[194] (McLeish, Emerton and Osborn JJA).

199 *Bowden* (2017) 54 VR 135, 138 [6] (Maxwell P and Kidd AJA); [2017] VSCA 46; *Visser* [2020] VSCA 327, [186]–[187] (McLeish, Emerton and Osborn JJA).

could have been either a cause of his death or a condition that made him susceptible to injury from handling which fell short of forceful shaking. As explained in Forrest and Emerton JJA's reasons, Professor Wester's evidence was that in his opinion, Kaleb did have BESS and that this was the likely cause of Kaleb's death. I will discuss that evidence in further detail below. First, however, it is necessary to understand the evidence concerning BESS at trial and the underlying basis for that evidence, including the report of Professor Ditchfield, which was not in evidence at the trial.

(a) The BESS evidence at trial

510 At trial, very little was said of BESS. However, Dr Tully gave evidence that she had excluded all possible medical causes of Kaleb's injuries. Thus, in relation to the 'working diagnosis' of the treating doctors on the 23 January 2016, she said this in her evidence in chief (emphasis added):

> **So, that's a diagnosis that needs obviously to be made with care, and with rigorous attention to detail, and arises as the result of a constellation – so a combination of findings** – that include subdural haemorrhage of a particular pattern, distribution and location within the subdural space, and that is across the surfaces of the brain, especially around the deep groove, into the deep groove and across the tentorium, which was that fibrous layer that went between the top and the bottom bit of the brain, plus evidence of damage to the brain itself, plus retinal haemorrhages, so haemorrhages at the back of the eyes that, again, are in a particular pattern and distribution, **plus exclusion of all alternative causes**.

511 Later, she said this:

> So, I think the combination of these findings, when an infant has been fully investigated for any other medical reason and there's no history of significant trauma, then we don't have another diagnosis other than inflicted head trauma.

512 And later she said that she had 'ruled out any alternative medical cause'.

513 In cross-examination Dr Tully gave evidence that she was reliant on other medical specialists to interpret and report on their findings and that her opinion was partly based on their findings; in particular, she accepted that she was reliant on radiologists, ophthalmologists and pathologists. As is clear from her second report dated 30 August 2018, one of those specialists was Professor Ditchfield (a paediatric radiologist).

514 In relation to Kaleb's pre-existing condition, Dr Tully gave evidence in chief to the effect that 'Kaleb's head circumference was increasing or had increased more than it should have done', and that on 14 and 15 January 2016 the ultrasound and MRI showed 'enlargement of the space between his brain and the skull and fluid in the subdural space that shouldn't be there, as well as those spaces in the centre — fluid-filled spaces in the centre of his brain being a little bit bigger'. She was then asked whether Kaleb died 'as a result of his head growing too big', or 'as a result of his head growing faster than it should have'. She answered 'no'. The following exchange then occurred concerning the increase in his head circumference:

> [I]s that head circumference attributable to the internal trauma to the child's head? You mentioned for example, haemorrhaging and so on?---No, that big

head circumference is likely to be attributable to something that's happened before. So that fluid that was identified on 14/15th January is almost certainly the reason why Kaleb's head circumference was big and had been enlarging. There was something going on prior to his presentation on the 23rd which meant he had a bigger head and that was the fluid inside his head that shouldn't be there.

What do you say about the collapse on 23 January; is that a supervening new event or is that a continuation of something else?---That's a new event. Something has happened in the interval, almost certainly just before he collapsed, to cause that.

515 In cross-examination on this issue, Dr Tully's evidence was as follows:

Your review of the medical records would be consistent with this: that between 14 and 17 January Kaleb was presenting with a situation of raised intracranial pressure; that's right, isn't it?---He had symptoms that suggested that, yes.

HIS HONOUR: Which were those symptoms?---Which were the bulging fontanel, the vomiting and irritability or crying.

What about the fact that you combine those things, including the bulging fontanel, with the increasing head circumference over time?---In terms of?

What does it tell you is going on or might be going on?---That there is a process happening inside the skull that is pushing outwards.

MR CASEMENT: I'll just ask you, p.356 of the previous trial, 27 March 2019, lines 9–11. This is a question I asked you and I'll ask you again whether this is the answer that you gave: 'And you had a situation from discerning of your medical records a position of potential raised intracranial pressure; correct?' And you answered: 'Yes'?---Yes.

…

Mr Gibson asked you some questions about the significance of the head circumference. You'd accept that at the time of at least his admission on 14 January 2016, that that was a concerning aspect of his disposition; that's right?---Yes.

…

If there was no tap performed on Kaleb Baylis-Clarke between 14 and 17 January 2016, there's a reasonable possibility that raised intracranial pressure may have persisted beyond that date; correct?---Yes.

…

You accept that Kaleb Baylis-Clarke was a patient who had pre-existing conditions, correct, prior to his presentation on 23 January 2016?---Yes.

You'd go this far, wouldn't you, that the pre-existing conditions that Kaleb Baylis-Clarke had may have predisposed him to subdural haemorrhage; correct?---So, what we know about babies with enlarged extra-axial spaces, is that, a small number of those — and the literature suggests around 5 per cent —

> can develop a subdural collection of fluid and that may sometimes contain blood, and that may occur either spontaneously or with what we would call trivial forces. So, yes, I accept that.
>
> But you would not accept as a reasonable possibility that he may have been more vulnerable to his final presentation by reason of his pre-existing condition; correct?---Yes, I — I don't — I'm not of the opinion that he was more vulnerable, and I can explain why that is if required.

516 Dr Tully was not asked expressly about BESS as a possible pre-existing condition, but she was asked about hydrocephalus, and other questions also raised matters that could be related to a diagnosis of BESS.

517 In relation to Kaleb's increasing head circumference, in cross examination Dr Tully accepted that the movement from between the 3rd and 10th centile at age two weeks, to the 95th centile at age three and a half months, was a concern, and that it was something 'you would have to look into … more'. She then gave evidence in relation to Kaleb's admission to hospital between 14 and 17 January 2016, as follows:

(a) at his admission he had a raised fontanel and a rapidly enlarging head size;

(b) scans found fluid on his brain;

(c) the fluid may, or may not, have had blood in it;

(d) no diagnostic tap or surgical intervention was conducted;

(e) Kaleb's eyes were not checked;

(f) his head circumference was described as being in the 90th centile;

(g) the discharge sheet stated that Kaleb may have had hydrocephalus, although the opinion of the neurosurgeons was that he did not;

(h) that raised intracranial pressure can be a feature of hydrocephalus;

(i) that the discharge summary said that Kaleb had sun-setting eyes, which are a sign of quite significant raised intracranial pressure; and

(j) that Kaleb had subdural hygromas and fluid in the subdural spaces.

518 Dr Tully also accepted in cross-examination that Kaleb's pre-existing conditions made him more susceptible to subdural haemorrhage. Later, when asked about the force necessary to cause Kaleb's injuries, she said this:

> the evidence base would suggest that, with this combination of findings even in the presence of an enlarged extra-axial space, that the retinal haemorrhages and the severe damage to the brain would indicate significant force. The subdural haemorrhage, I accept, maybe it's less — we don't know, it's unclear.

519 That is, it is clear that Dr Tully accepted that although Kaleb might have been more susceptible to subdural haemorrhage, he was not more susceptible to the retinal haemorrhages or the hypoxic ischaemic injury to the brain.

520 The evidence she gave at trial was based on her two written reports, and also on a report prepared by Professor Ditchfield who, as I explain further below, had been engaged to provide an opinion as to whether Kaleb had BESS.

(b) Dr Tully's written reports

521 In early February 2016, shortly after Kaleb's death, Dr Tully had prepared her first written report concerning Kaleb's death (her second report, prepared in August 2018, being an addendum to her first report). In her first report she considered BESS, and said as follows (emphasis added, footnotes omitted):

> Benign enlargement of the subarachnoid space ('BESS') is a relatively common condition seen in infants and can be confused with subdural hygromas. It is characterised by macrocephaly (a large head circumference) and enlarged subarachnoid spaces containing CSF [cerebrospinal fluid]. It can be associated with mild ventricular enlargement although the ventricles are usually normal in size. These features were all noted in Kaleb's case. However BESS does not usually cause symptoms of vomiting, drowsiness and poor feeding. The typical MRI features of BESS were not present in Kaleb's case.
>
> BESS results from an imbalance between production and absorption of CSF due to immaturity and resolves spontaneously over the first couple of years of life. It has been suggested that infants with BESS might develop intracranial bleeding after minor head trauma, although long-term observation of infants with BESS have not shown an increased risk of subdural haemorrhage. Infants with BESS do not develop retinal haemorrhages and neurological collapse as result of minor head trauma. **Although it is possible that Kaleb had BESS, this would not explain his preceding symptoms, his sudden collapse and widespread retinal haemorrhages.**
>
> Kaleb's initial ultrasound scan on 14th January 2016 reported mild ventricular dilatation and 'hydrocephalus'. This finding was confirmed on subsequent imaging (MRI and CT). No cause was identified. Hydrocephalus is the term used for an abnormal accumulation of CSF within the cavities of the brain (ventricles).
>
> …
>
> It was the opinion of the neurosurgical team that the accumulation of CSF within Kaleb's lateral ventricles was not sufficient to use the label hydrocephalus. It is possible that the mild dilation of Kaleb's lateral ventricles and increased CSF resulted from previous subdural haemorrhages, the disruption of CSF flow and formation of subdural hygromas. **It is possible that these findings were a result of BESS. Other causes are possible. The precise aetiology of this finding has not been determined.**

522 She summarised her conclusion on BESS as follows (emphasis added):

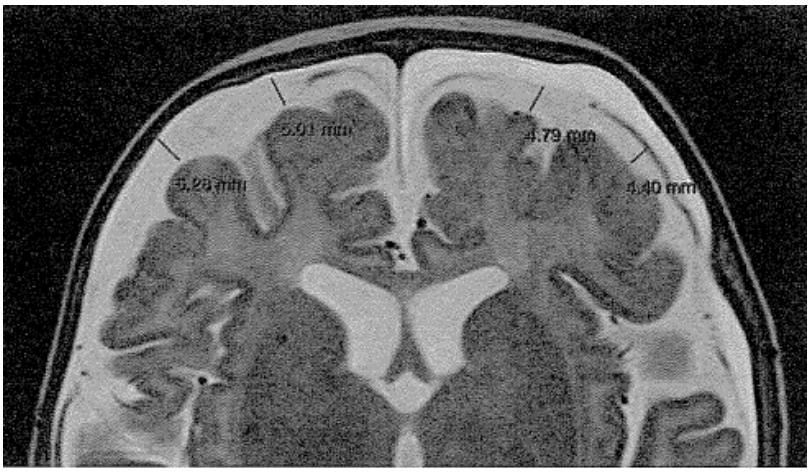
> In summary, although Kaleb might have had a condition known as BESS, this does not explain his symptoms prior to admission or his clinical presentation on 23rd January. The presence of chronic subdural hygromas in Kaleb's case suggests the possibility of at least one previous episode of trauma to the head although it is noted that there are non-traumatic causes of chronic subdural

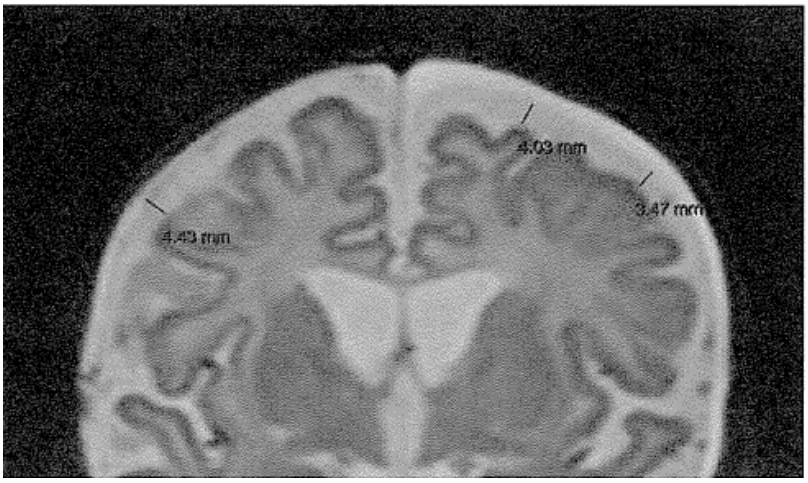
hygromas in infancy. **The finding of mild hydrocephalus is not associated with subdural haemorrhages and retinal haemorrhages**.

(c) Professor Ditchfield's initial report

523 Professor Ditchfield provided an initial report, prior to trial, in which he excluded BESS as a condition to which Kaleb was subject. That report was procured after the committal, as a consequence, it seems, of questions Dr Iles was asked at the committal, and because BESS had been raised by Professor Duflou.[200]

524 Professor Ditchfield is a paediatric radiologist with 22 years of experience. He prepared a report on 29 August 2018. He was asked to express a view in relation to Kaleb about whether there was any radiological evidence of BESS. He considered five images from the MRI performed on 15 January 2016 (ie during Kaleb's first admission, and prior to his collapse on 23 January 2016). Of particular relevance were the following two images presented at Figure 4, namely an axial T2 weighted image (the first image) and a coronal T2 weighted image (the second image):





200 At the committal Dr Iles was asked if she could exclude BESS and she started her answer to the question with the statement 'I'm not a radiologist', and pointed out that she had not seen the initial imaging of Kaleb's brain, indicating that she could not express a definitive view. However, she stated that her findings on the autopsy correlated with a diagnosis of chronic subdural hygromas, not BESS.

525 The text with Figure 4 stated that these images demonstrated 'measurements of the subarachnoid space, **almost all, less than 5 mm in measurement,** with an occasional measurement being at 5 mm' (emphasis added).

526 As to whether there was any radiological evidence of BESS, Professor Ditchfield said as follows (emphasis added):

> BESS (Benign Enlargement of the Subarachnoid Space) by definition is an enlargement of the subarachnoid space that occurs in infants. The diagnosis is often made by a subjective assessment of the extra axial spaces. **There is no agreed depth of the subarachnoid space required to make this diagnosis. Most authors in the literature use a definition of greater than 5mm** to make this diagnosis (Reference 1).
>
> In relation to the MRI performed on 15 January 2016 on Kaleb … **my subjective assessment is that the subarachnoid space is within the normal range** for this age. By measurement (Figure 4) **the subarachnoid space largely measures less than 5mm, with an occasional measurement being at 5mm**. My assessment is that this MRI demonstrates a subarachnoid space at the upper range of normal and does not demonstrate BESS.

527 I note, at this point, that one of the measurements of the subarachnoid space in the images was 5.01 mm, and another was 5.28 mm. 'Reference 1' was a textbook by Kleinman.[201]

528 Professor Ditchfield's initial report was thus not entirely consistent with Dr Tully's first written report, in which she had opined that it was 'possible' Kaleb had BESS.

(d) Dr Tully's second report

529 Following the receipt of Dr Ditchfield's initial report, Dr Tully prepared a second report, in which she offered the following revision to the opinion provided in her first report (emphasis in original):

> There is no radiological evidence of BESS on the MRI performed on 15 January 2016. In combination with Kaleb's clinical presentation on 14th January 2016 with a full fontanelle and vomiting on the background of being 'not himself' for several weeks previously, I conclude that Kaleb did not have BESS.

530 She also stated that the fluid in the subdural space identified in the MRI scan was likely to be the result of a prior subdural haemorrhage, that was most likely caused by trauma. She said that the opinion she had expressed in her first report — that the presence of the subdural haemorrhages and the widespread multilayered retinal haemorrhages, in combination with Kaleb's catastrophic collapse, were 'not in keeping with "trivial" forces' — had not changed in light of the new information provided by Professor Ditchfield.

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201 Paul Kleinman, *Diagnostic Imaging of Child Abuse* (Cambridge University Press, 3rd ed, 2015) 422 ('Kleinman').

(e) Professor Wester's evidence

531 Professor Wester gave evidence that, in his opinion, Kaleb had BEH (being another term for BESS). As Forrest and Emerton JJA explain, Professor Wester concluded that the cause of Kaleb's death was a global ischemia caused by the cardiopulmonary arrest resulting in a lack of oxygenated blood supply to the brain on the evening of 23 January 2016. That is consistent with the conclusion reached by Dr Tully as to the immediate cause of death. However, in his opinion the underlying cause was a rapidly developing external hydrocephalus, complicated by a subdural haematoma that most probably elicited an Acute Life–Threatening Event ('ALTE').

532 According to Professor Wester, the ultrasound and MRI performed on 14 and 15 January 2016 showed 'bilateral chronic subdural fluid collections with the typical appearance of BESS'. Professor Wester opined that these, together with Kaleb's 'dramatically increasing head circumference', vomiting, and tense fontanelle, were features indicating a rapid development of increased intracranial pressure.

533 Professor Wester gave evidence that BESS is clinically characterised by a large and/or rapidly growing head and is often associated with symptoms of delayed development and elevated intracranial pressure, such as irritability, feeding problems and epilepsy. Radiologically, BESS is characterised by an increasing amount of cerebrospinal fluid in the subdural compartment during the first weeks or months after birth, as well as an increasing amount of cerebrospinal fluid in the ventricles of the brain. The first sign is a frontal increase in the distance between the two cerebral hemispheres, followed by an increased fluid layer in the subdural compartment and, finally, increased fluid in the ventricles.

534 In relation to Kaleb, Professor Wester noted the abnormally rapid rise in Kaleb's head circumference from birth (when his head circumference was around the 3rd percentile) to eight days before his sudden deterioration on 23 January 2016 (when his head circumference was around the 90th percentile). That, in his opinion, together with the features revealed in the first MRI on 15 January 2016, strongly indicated the development of BESS. Consistently with this diagnosis, Professor Wester observed that the MRI showed moderately enlarged ventricles and an increased distance from the brain surface to the inner skull. Professor Wester based his radiological identification of BESS on the images generated by the MRI performed on Kaleb on 15 January 2016 at Monash Medical Centre (the 'first admission'), which were reproduced and discussed in the statement of Professor Ditchfield.

535 Professor Wester also concluded that the hygromas observed at the first admission were in fact chronic subdural haematomas, based on Professor Ditchfield's evidence that there had most likely been blood in the subdural fluid seen on the MRI of 15 January 2016, and on Dr Iles' finding of membranes in the subdural compartment, indicating a subdural haemorrhage at a time preceding Kaleb's final collapse.[202]

536 Ultimately, Professor Wester concluded that it was highly probable that the underlying cause of the cardiopulmonary arrest that caused Kaleb's death was a rapidly developing external hydrocephalus complicated by a spontaneously occurring subdural

202 Dr Tully and Dr Iles accepted that the membranes were indicative of a pre-existing haematoma, rather than a hygroma.

hygroma/haematoma causing increased intracranial pressure that most probably elicited an ALTE with cardiopulmonary arrest. An injury to the head is not necessary to explain this series of events.

537 In Professor Wester's report he expressed the view that Dr Tully was wrong to state in her report that the typical MRI features of BESS were not present in Kaleb's case, and that Professor Ditchfield was wrong to exclude BESS.

538 As to Professor Ditchfield, Professor Wester stated that Professor Ditchfield was correct to say that a diagnosis of BESS is based on subjective assessment. He further stated that a diagnosis of BESS is not based on radiological criteria alone; the radiological criteria must be used in combination with clinical signs, in particular a 'too large head circumference', or one that grows too fast.

539 Professor Wester's opinion was that Professor Ditchfield had used incorrect measurements of intracranial distances on the MRI of 15 January 2016. Professor Wester's evidence was that the measurements of the sinocortical and craniocortical widths are measurements of the distance from the cerebral cortex to the inside of the skull/superior sagittal sinus, not the distance from the cortex to the arachnoid membrane. He also stated that the radiological criteria for diagnosing BESS differ between publications, so that the upper limits above which the measurements are likely to be abnormal fall into a range:

(a) for craniocortical width, a range from 4 mm to 10 mm;

(b) for sinocortical width, a range from 2 mm to 10 mm; and

(c) for interhemispheric distance, a range from 6 mm to 8.5 mm.

He observed that 'no validated normal values exist and thus the cut-off values may differ between radiologists'.

540 In Professor Wester's view, Professor Ditchfield had measured the wrong distances, having measured the distance from the cerebral cortex to the arachnoid membrane, rather than the distance to the inside of the skull. He also stated that some of the lines that Professor Ditchfield measured did not cover the entire distance that Professor Ditchfield claimed they covered.

541 Professor Wester's opinion was that if the craniocortical width had been measured correctly, it would have shown distances 'way above all radiological criteria for BESS'. He gave coarse estimates, based on Kaleb's MRI of 15 January 2016, that both sinocortical width and interhemispheric distance were above 10 mm and that craniocortical width was close to 10 mm. Professor Wester also observed that Professor Ditchfield did not make any attempt to measure sinocortical width or interhemispheric distance.

(f) Professor Ditchfield's rebuttal report

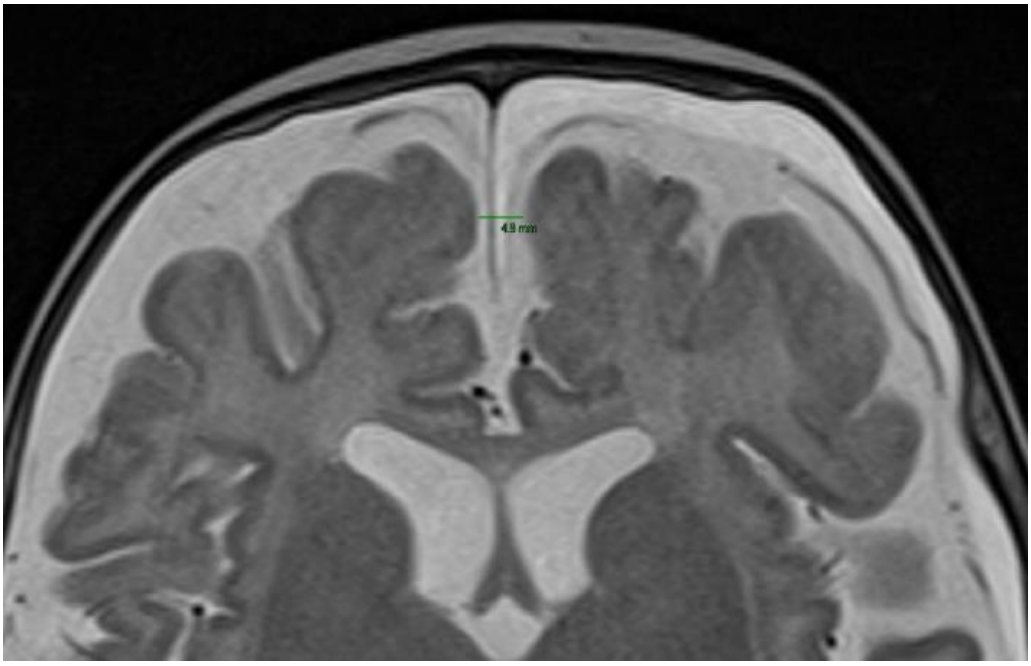
542 Professor Ditchfield provided a rebuttal report in which he responded to Professor Wester's report. Relevantly, Professor Ditchfield expressed the following opinions:

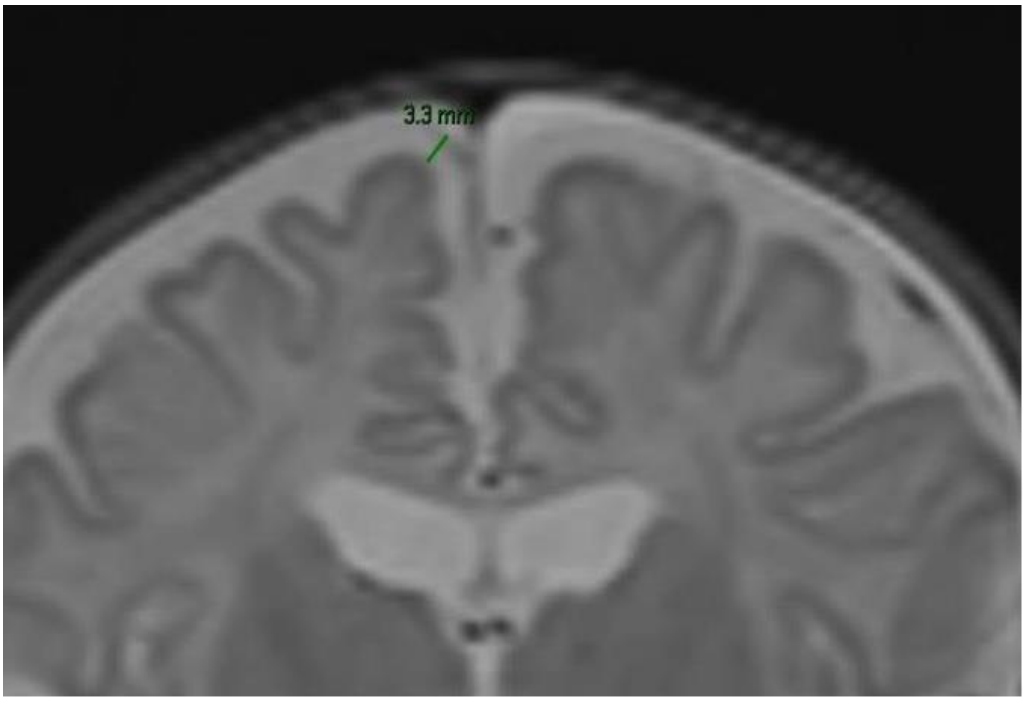
(a) In his report, Professor Wester had confused the subarachnoid and subdural spaces; BESS is the enlargement of the subarachnoid space, not of the subdural compartment, as Professor Wester's report stated.

(b) Professor Wester's statement that 'the diagnosis BESS/BEH is however not based on radiological criteria alone' is correct, 'however if the radiological criteria for BESS are not met then this diagnosis is not present'.

(c) In the diagnosis of BESS the subarachnoid space needs to be measured. In the absence of a subdural collection, this measurement will be from the cerebral cortex to the inside of the skull, as Professor Wester proposes, because the subdural space is negligible. However, in the presence of a subdural collection the measurement must exclude the subdural collection, otherwise the measurement will be considerably greater and BESS will be diagnosed inappropriately. He stated that Professor Wester had not provided scientific justification for including a subdural collection in his measurement of BESS.

543 In his rebuttal report Professor Ditchfield included two further images, reproduced below, where he measured the sinocortical width and the interhemispheric distance (without commenting on Professor Wester's statement that he had previously failed to measure them). In the first image he identified an interhemispheric distance of 4.9 mm. In the second he identified a sinocortical width of 3.3 mm.





544 Following the images, Professor Ditchfield extracted three charts of normative data on which he plotted the measurements he had taken. These charts were taken from an article by Lam et al.[203] This, he said, demonstrated that Kaleb's craniocortical width and sinocortical width were below the mean for his age and that the interhemispheric distance was minimally above the mean. He also observed that the measurement of the latter (which was normal) included the subdural collection and was therefore artificially greater than the true measurement of the subarachnoid space. In his opinion, the size of the subarachnoid space was normal and there was no evidence of BESS.

(g) Professor Wester's oral evidence on the appeal

545 In his oral evidence on the appeal Professor Wester responded to Professor Ditchfield's rebuttal report. Of particular significance was his response to Professor Ditchfield's new measurements of the sinocortical width and interhemispheric distance. As to sinocortical width, Professor Wester said that Professor Ditchfield should have measured from the cortex to the sagittal sinus, but had not done so. As to the interhemispheric distance, Professor Wester said as follows:

> You see he has tried to make it the distance between the two hemispheres. He has chosen the shortest, not the widest distance as Lam recommends. If he had moved his — his, ah, measuring — his ruler — a centimetre further down — he would have doubled or even perhaps trebled the — the measure he got. So he has done it wrong, and his calculation is therefore wrong.

546 Professor Wester's evidence was that because Professor Ditchfield had not measured the distances recommended by Lam, it was not correct to conclude, based on the normative data provided by Lam, that Kaleb's measurements were normal. He said that the Lam report included a sketch of the brain and where to measure. The Lam report

203 Wendy Lam et al, 'Ultrasonographic Measurement of Subarachnoid Space in Normal Infants and Children' (2001) 25(5) *Pediatric Neurology* 380 (the 'Lam report').

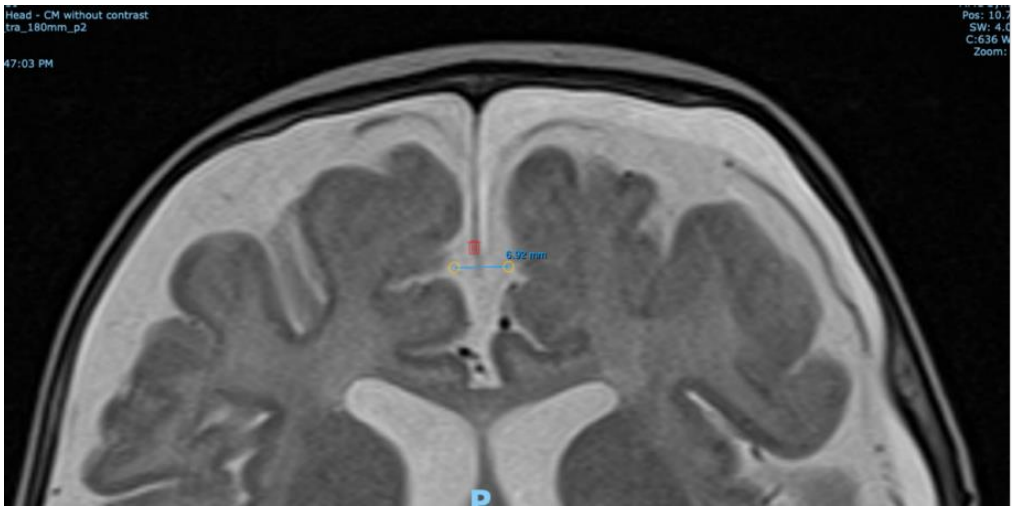
was put into evidence, and a diagram on the second page confirmed what Professor Wester had said.

547 In cross-examination Professor Wester was asked whether the existence of a subdural hygroma meant that one did not measure the interhemispheric distance strictly in the way described by Lam, because that would capture the subdural collection, which would artificially widen the measurement. Professor Wester disagreed with that proposition, because he said that there is no subdural collection between the two hemispheres. That area, he said, is just subarachnoid space with cerebrospinal fluid. He pointed out that in the images one could see the subdural collection, and that it did not extend down in between the hemispheres. He also rejected the proposition that the subdural collection 'pushes down upon the subarachnoid space, changing its shape'.

548 Professor Wester also pointed out that the Lam report recommends measuring on the coronal section of the scan,[204] whereas Professor Ditchfield had performed his measurements on the axial section. He observed that 'if you switch between the axial sections and coronal sections, you leave the recommendations of Lam'. He said that the contribution of the Lam report is that it has 'sort of standardised the measurements and where to do them'.

(h) Professor Ditchfield's oral evidence

549 Professor Ditchfield gave evidence after Professor Wester. Having watched Professor Wester give evidence, Professor Ditchfield produced to the Court a further MR image, on which he measured the interhemispheric distance at a lower point that he had previously measured it, showing a measurement of 6.92 mm, rather than 4.9 mm:



550 When that figure was plotted on one of the charts from the Lam report that Professor Ditchfield had used in his rebuttal report, he said that it showed that Kaleb was between the 75th and 80th percentiles — not at or close to the mean (as his rebuttal report had said).

551 That image was tendered as an exhibit in the course of Professor Ditchfield's oral evidence. In his oral evidence about that new MRI image, Professor Ditchfield said that Professor Wester was correct that Lam had done the measurement at the lower, wider

204 Referring to the paragraph below figure 1 in the Lam report, 381.

point of the interhemispheric distance, and so in his new MRI image he measured the distance 'according to Lam'. However, he maintained that 'most of the modern literature' measures it at the location he had originally provided, and papers were tendered in support of that proposition.[205] He also explained that the children the subject of the Lam report were 'normal' children, in that they did not have subdural collections. Thus, he said, applying the Lam measurements 'to patients with subdurals … there is no validity to that … and I've demonstrated that by showing you that other case where a subdural will expand that measurement'.

552 Further, Professor Ditchfield said that the Lam data was based on ultrasound images, that are less precise than MR images. Thus, he said, 'even though we're using the data from … [the Lam] paper … this paper does not represent normative data for MR and the normative data for an MR will be significant greater'.

553 He also gave evidence that, if a subdural collection is included in the craniocortical width, that will artificially widen the subarachnoid space, such that every child with a subdural would be diagnosed with BESS. He demonstrated that proposition by reference to an image in a paper by Rooks et al,[206] with his own measurements added to it. In so far as the interhemispheric distance is concerned, he gave evidence that in Kaleb's case the subdural extended into the interhemispheric space and, like a wedge, would have pushed apart the hemispheres and artificially expanded that measurement of 6.92 mm. In that regard, the following exchange occurred:

> WALKER JA: The measurement that you took, staying with this interhemispheric width, the 4.8 you said is measured across I think the haematoma or hygroma the subdural collection that extends down into that interhemispheric space, the measurement at the wider part, forgive me for not using the technical term, would that be below the ---?---So, on this image, it doesn't look like the subdural space has extended to that point but if you imagine putting a wedge between the two hemispheres, the subarachnoid space will widen below it.

554 Professor Ditchfield was asked why, in his rebuttal report, he had chosen to measure at the higher location for the interhemispheric distance. His response was that 'that's where we usually measure it now', because the anatomy is more predicable in that location. When asked why he would then use Lam, he said that that was an error, and that he should have measured in a different location. He said he had not realised that Lam had measured at the lower point. When asked how it was then appropriate to use the Lam charts, he said that they are 'the most current that we have … the best data that we have'. However, he said that because Lam had used ultrasound, Lam was not measuring what is now measured on MR. Thus 'the average that they're obtaining from their data would be less than if we did normative data using MR now'. Ultimately Professor Ditchfield said that plotting data from an MRI onto the Lam charts is 'flawed

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205 Sook Kyung Yum et al, 'Enlarged Subarachnoid Space on Cranial Ultrasound in Preterm Infants: Neurodevelopmental Implication' (2019) 9(1) *Scientific Reports* 19072 ('Yum et al'); Pradeep Raj Regmi et al, 'Benign Enlargement of Subarachnoid Space in Infancy' (2020) *Eurorad* 16696: 1–7 ('Regmi et al'); Liza van Eijk et al, 'Automating Quantitative Measures of an Established Conventional MRI Scoring System for Preterm-Born Infants Scanned Between 29 and 47 Weeks' Postmenstrual Age' (2021) 42(1) *American Journal of Neuroradiology* 1870.

206 Veronica Rooks et al, 'Prevalence and Evolution of Intracranial Haemorrhage in Asymptomatic Term Infants' (2008) 29 *American Journal of Neuroradiology* 1082.

without any doubt'. In his opinion the consequence of the flaw is an overestimate of the diagnosis of BESS. However, he later described the Lam report as the best publication that he relies on in his work, and observed that 'in medicine we often use … data that we know is flawed but it's … what we have and … we know the limitation of that data … and we work with that'.

555 As to the selection of greater than 5mm as the measurement to make the diagnosis, as set out in his original report, Professor Ditchfield said that that is 'arbitrary'. He later explained that 'there is a large observer variability of where you measure it from, what you include, and so forth', and thus 'in trying to set an absolute number … and we're using data that is, you know, 20-odd years ago – it's majorly flawed and we're trying to overlay that where half of children will have a number greater than … five millimetres – and so … when we're talking about BESS, the definition you know is just so arbitrary'. Later this exchange occurred (emphasis added):

> --- When you've got — when you're — so — and by measuring over all of these spaces for Kaleb, his measurement's around five, and not six.
>
> But didn't your report say five millimetres?---Yes, it did. Well I had to come up with something — a number.
>
> …
>
> EMERTON JA: But are you today saying it would need to be above ten or around ten for a BESS diagnosis?---Well no, the honest — the honest answer is BESS has not been well-defined, so what is BESS? Like when is it pathological, so ---
>
> … So we see this variability in the subarachnoid space where in children of Kaleb's age, so at 16 weeks, the subarachnoid space will vary between 4.7 and 9.3 or whatever it is. And so my report is not that this child has BESS, it's that this child has prominence of the extra-axial space which is usually a normal finding at this age, and that's true, because 95 per cent of children will be below 9.5 millimetres at that age, and — and — and in my report, I was using what is used in the literature, and the literature is flawed by all of these things; the age of when the literature was produced, but all we have to go with is Lam, ah, which does set the threshold low, ah, for diagnosing, um, ah, when it's — when it's above the 95th percentile. But even using his data with that lower threshold, Kaleb's doesn't come to that.
>
> …
>
> WALKER JA: Now, what I'm asking you is … if it was at the upper range of normal and we change the interhemispheric width measurement so that it's even higher, are we over the line or are you just maintaining that your subjective view hasn't changed, even [though] the measurements might?---No, 'cause if we go back to — so, two points, I just want to clarify now. So, the first one is, um, ah, so when — **when do we call BESS. And I don't have — I don't have a good answer to that and I don't think you can answer that because it's not well defined.** It's within the range of normal, um, so what I've done in the report is follow what is in the literature. What I'm sharing with you now is, um, is a depth higher than that — that the literature is flawed.

556 He went on to say that BESS is not a term he uses in clinical practice because he considers it to be misleading, in that it is giving 'a diagnosis for something that's normal', because 50 per cent of children will have a cerebrospinal fluid space that is between 5 mm and 10 mm, depending on age. He said that is why it is referred to as 'benign' enlargement of the subarachnoid space — it is not a clinical or pathological condition. (In contrast, a subdural collection is pathological.) Professor Ditchfield's opinion was that an enlargement of the subarachnoid space would not be pathological unless it reached the 95th percentile, which would be at a measurement of around 8 to 10mm. That is, 5 mm is not the 'cut-off' for BESS, the cut-off is at the 95th percentile. But he also described 5 mm as a 'convenient cut-off', which was why he used Kleinman in his original report, even though he would not use 5 mm in clinical practice.

557 As to the relevance of clinical observations, in addition to radiological measurements, Professor Ditchfield said this:

> There are conditions … which will mimic at BESS. And in those cases, ah, the head circumference will increase, um, at a greater, um — at a greater rate than you would expect for BESS. There will be signs of raised intracranial pressure, ah, and so, the clinical, um — the clinical setting of that is — is important. So, — so, yes. But, without the findings radiologically of this, the child, the child does not have BESS. So, the clinical — um, the clinical setting does not, um, make a diagnosis of BESS.

558 Professor Ditchfield accepted that he was not aware of the clinical signs that Kaleb had been experiencing but said that they were not relevant to the questions he had been asked to address in his report. That was because, without the radiological signs, Kaleb did not have BESS — and in his opinion, Kaleb did not have the radiological findings of BESS.

(3) Consideration of the BESS evidence

559 The apparently definitive exclusion of BESS as a pre-existing condition from which Kaleb suffered at the time of his collapse was, by the time of trial, based on Professor Ditchfield's report (Dr Tully having initially considered that it was possible that Kaleb had BESS). In my opinion, there are a number of deficiencies in Professor Ditchfield's initial report, in his rebuttal report and in his oral evidence that lead me to conclude that his exclusion of BESS ought not be accepted — that is, as I discuss later in these reasons, had the jury received the new evidence, it would not have been open for them to exclude the proposition that Kaleb suffered from BESS.

560 *First*, in Professor Ditchfield's initial report he stated that there is no agreed depth of the sub-arachnoid space for a diagnosis of BESS, but that most authors in the literature use a definition of greater than 5 mm to diagnose BESS, relying on Kleinman. That particular passage from Kleinman stated that children with BESS 'typically' have skull-to-cerebral cortex measurements of greater than 5 mm.[207] That suggests that there could be children with lower measurements who may nonetheless have BESS. However, Professor Ditchfield applied 5 mm as if it was an absolute cut-off, so that a child with measurements below 5 mm does not have BESS. In his oral evidence he described

[207] Kleinman, 422.

5 mm as a 'convenient cut-off', but when asked about Kleinman he said that he would not use the term 'cut-off'. He also said that in clinical practice he would not use 5 mm.

561 Later, in his oral evidence, he described the adoption of 5 mm as the radiological line for diagnosing BESS as 'arbitrary'. Yet in his initial report, and in his rebuttal report, he used 5 mm as if it were generally accepted and appropriate.

562 It is a matter of real concern that, in the context of a criminal proceeding for a very serious offence, Professor Ditchfield considered it appropriate to use, in his initial report, what he considers to be an arbitrary measurement as the basis for excluding a diagnosis of BESS, without acknowledging that it was arbitrary. Professor Ditchfield's explanation of his use of 5 mm in his initial report — 'well I had to come up with something, a number', otherwise 'it would have been a whole thesis on BESS' — was entirely inadequate. Similarly, it is a matter of real concern that, in his rebuttal report, prepared for filing in this Court, Professor Ditchfield chose to use data that he described as 'flawed', without acknowledging that in the report itself. Those reports were, in light of his later oral evidence, quite misleading.

563 *Secondly*, in his initial report one of the measurements was 5.01 mm and one of the measurements was 5.28 mm, suggesting that Kaleb was at least close to the diagnostic measurement that Professor Ditchfield had chosen to use. Further, Professor Wester had stated in his report that some of Professor Ditchfield's measurements did 'not cover the entire distance' claimed. In his oral evidence Professor Ditchfield accepted that, in his initial report, he had not measured all the way down to the brain surface, and said that the measurement of 5.28 mm could have been 5.35 mm.

564 *Thirdly*, Professor Ditchfield appeared to accept that it was appropriate to measure not only those distances he had initially measured, but also the sinocortical width and the interhemispheric distance. He thus produced those measurements in his rebuttal report, and plotted them on the Lam charts. However, he accepted in oral evidence that, in relation to his measurement of interhemispheric distance, and its plotting on the Lam chart, 'that was an error', because he had not measured at the same location as Lam had measured.[208] This error was significant because, instead of placing Kaleb's interhemispheric distance at 'minimally above the mean' for his age, it placed that distance as considerably higher.

565 Having realised his error, Professor Ditchfield then produced yet another set of measurements, which he again plotted on the Lam charts. On this occasion he measured the interhemispheric distance as 6.92 mm, and concluded that this placed Kaleb at around the 80th percentile on the Lam charts. That was significant because, in Professor Ditchfield's oral evidence he stated that a measurement at the 95th percentile would be considered abnormal (which would be 8 to 10 mm, he said). However, it remained unclear to me that his new measurement was located precisely where it ought to have been located, based on the Lam article and on Professor Wester's evidence. Thus I am

208 I note that Professor Ditchfield maintained that he had initially measured in an appropriate location. He relied on studies that used the same interhemispheric distance measurement point as he had used (see, eg, Yum et al and Regmi et al). However, those studies did not present their data by reference to Lam's charts. That is, those studies do not support what Professor Ditchfield did in his rebuttal report, which was to plot measurements on charts containing the Lam data when those measurements simply did not correspond to the Lam data.

not prepared to accept that the measurement of 6.92 mm, or the consequent plotting on the Lam chart, was accurate. It is reasonably possible that the correct measurement of the interhemispheric distance was greater than 6.92 mm and, as a consequence, that Kaleb was at or above the 95th percentile in relation to his interhemispheric distance.

566 I also note that Professor Ditchfield gave evidence that the interhemispheric distance measurement was artificially inflated by the subdural collection that extended down into the space between the hemispheres. However, the interhemispheric distance was (ultimately) not measured across the subdural collection, but below it. Even accepting that the extension of the subdural collection would inflate the measurement, Professor Ditchfield offered no opinion as to the extent of the effect — that is, by how much the measurement would be inflated. Ultimately, it is plain that the figure obtained would have been considerably higher than Professor Ditchfield's measurement in his rebuttal report.

567 *Fourthly*, notwithstanding his reliance, twice, on the Lam data and the plotting of Kaleb's measurements on the Lam charts, in his oral evidence Professor Ditchfield said that the Lam data was flawed, so that it was not appropriate to use it. It is difficult to square that evidence with the presentation to the Court, in a formal report, of a definite conclusion that Kaleb did not have BESS based, at least in part, on the Lam data. Again it is of real concern that a report would be prepared and presented in a criminal trial for a serious offence, based on what is later admitted to be flawed data.

568 *Fifthly*, although Professor Ditchfield excluded BESS in his initial report and in his rebuttal report, as if it was a condition that could be excluded, it appeared from his oral evidence that Professor Ditchfield did not accept that BESS was a valid or useful diagnosis. At one point he rhetorically asked 'so what is BESS?', and later 'when do we call BESS?' He said that BESS is not a term that he used in clinical practice because he considers it to be misleading, it is 'giving a diagnosis to something that's normal'. Again, however, there was no indication of that view in his written reports, which proceeded on the basis that BESS was a valid diagnosis that could be excluded.

569 Relatedly, Professor Ditchfield was of the view that if 5 mm was adopted as a 'cut-off' for BESS, then '50 per cent of children would be pathological'. He said that was not a 'useful medical approach'. However, Professor Wester gave evidence that a diagnosis of BESS was not based on radiological criteria alone. Professor Ditchfield agreed with that proposition. That is, both experts accepted that it is necessary to consider other clinical matters in addition to the radiological measurements when diagnosing BESS. According to Professor Wester, these additional matters include the size of the head circumference, the speed of head circumference growth and the presence of elevated intracranial pressure, including by reference to a raised fontanelle. If that is so, then it would not follow that, if 5 mm is used as a 'cut-off' for BESS, 50 per cent of children would have BESS. Rather, other clinical matters would also be necessary before such a diagnosis should be made.

570 Importantly, in the present case Kaleb in fact had those other clinical indicators. However, it appears that Professor Ditchfield was not aware of that when he prepared his reports. In his evidence on the appeal he said that additional clinical data of that kind would be 'helpful' and he accepted that, if he had known that Kaleb had increased intracranial pressure, or known of the bulging fontanelle (which is a symptom of raised

intracranial pressure), then, although his measurements would be unaltered, his interpretation of the images might be altered, because raised intracranial pressure is 'not normal'.

571 *Sixthly*, in two of the articles Professor Ditchfield relied upon in support of his approach to the location of the relevant measurements, the authors adopted figures other than 5 mm as the criteria for BESS.

(a) Yum et al referred to 'the traditional criteria' for BESS as 'IHW [interhemispheric width] > 6 mm, CCW [craniocortical width] > 4 mm, and SCW [sinocortical width] > 3 mm'.[209]

(b) Regmi et al referred to the 'normal upper limits of measurement' as 'CC: 4 mm, SC: 3 mm and IH: 6 mm',[210] although it also observed that another paper had described a 'more flexible range of measurement' as 'IH: 6 to 8.5 mm, SCW: 2 to 10 mm and CC: 4 to 10 mm.' Regmi et al stated that 'if the measurements are more than the given upper limits, subarachnoid spaces are said to be enlarged'.[211]

These figures were consistent with Professor Wester's evidence concerning the upper limits for which measurements are likely to fall into the abnormal range.

572 On those figures it seems highly likely from the measurements that Professor Ditchfield provided to the Court that Kaleb had BESS:

(a) Kaleb's sinocortical width was 3.3 mm;[212]

(b) Kaleb's craniocortical width was 4–5 mm; [213] and

(c) Kaleb's interhemispheric distance was at least 6.92 mm.

573 That is so even without resolving the disagreement between Professor Ditchfield and Professor Wester as to whether the measurements should include or exclude any subdural collection, because it is based on Professor Ditchfield's measurements, not Professor Wester's estimated measurements. If Professor Wester's measurements were accepted, then the conclusion that Kaleb had BESS would be irresistible.

574 In addition, and noting that a diagnosis of BESS does not turn on the radiological criteria alone, the uncontradicted evidence was that Kaleb had an abnormally high head circumference, which had increased dramatically over time (crossing from the 3rd percentile to the 95th percentile), and that he had a bulging fontanelle, which demonstrated increased intracranial pressure. He also had a subdural haematoma that predated the events of 23 January 2016.

209 Yum et al, 6.
210 Regmi et al, 1.
211 Regmi et al, 1 (citations omitted).
212 Professor Ditchfield confirmed in oral evidence that there was no need for him to remeasure this distance.
213 Professor Ditchfield confirmed in oral evidence that there was no need for him to remeasure this distance.

575 In light of all of the above evidence, I conclude that, had this evidence been adduced at trial, it would not have been open to the jury to accept that BESS had been excluded as a pre-existing condition from which Kaleb suffered at the time of the events on 23 January 2016.

(4) The significance of BESS

576 Of course, it is not sufficient to conclude that BESS could not be excluded. The further question is what impact that might have had on the jury's finding of guilt.

577 At trial, both Dr Tully and Dr Iles accepted that Kaleb's pre-existing conditions (which they did not describe as BESS) made him more susceptible to subdural haemorrhage. Each also accepted that it was unclear what degree of force would have been required to produce the subdural haemorrhages. However, Dr Tully, in particular, maintained that the presence of the retinal haemorrhages indicated significant force. In that regard, the following exchange occurred (emphasis added):

> [Would you] agree or disagree that there is further controversy as to whether the degree of existing pathology, natural or otherwise, can modify the extent and prognosis of these injuries?---As we've talked about, we don't know the magnitude or degree of force that's required. I think it is possible that, in a child who has a pre-existing enlarged extra-axial space, then we don't know the effect of that on the degree of force required to cause subdural haemorrhage; that's because the bridging veins are theoretically more stretched, because they're going through a bigger space. There's alternative opinion that says they are better buffered by all of the fluid in the space. We simply don't know. What we do know is that a small number of them can develop small subdural haemorrhages without any symptoms either spontaneously or with trivial forces. However, enlarged extra-axial spaces in your head don't affect your eyes, and **we know that to cause widespread multilayered retinal haemorrhages requires significant forces**.

578 Later, she said this (emphasis added):

> [Y]ou can only diagnose [the 'triad'] when you have very specific features or each of those elements, and **the retinal haemorrhages are crucial** to that, and in my opinion are very important findings, because they are highly associated with inflicted head trauma, and in particular with fatal inflicted head trauma, and because they're seen very often in cases of both inflicted head trauma, fatal inflicted head trauma in particular, then **we can make some assumptions, while we can't measure it, about level of force required to cause it**.

579 Thus the presence of retinal haemorrhages was a critical element in Dr Tully's evidence as to her diagnosis, as well as to her conclusion about the level of force required to produce the injuries to Kaleb, which was of course a key issue at the trial.

580 On the appeal, Dr Tully expressed the view that BESS could not explain the features with which Kaleb presented on 23 January 2016. She said that 'by definition' BESS is benign and so it does not cause clinically significant symptoms.

581 However, Dr Tully accepted that there is an increased rate of subdural haemorrhage in patients with BESS because of the stretching of the bridging veins, and that the tearing

of these vessels can occur in the setting of very minor trauma. Thus she accepted that a subdural haemorrhage or hygroma 'can develop with trivial or minor head injury in the setting of BESS'. She also accepted that 'idiopathic' — that is, unknown — causes of hygromas are 'frequent'.

582 Notwithstanding those matters, Dr Tully maintained that BESS would not cause retinal haemorrhages of the extent and pattern seen in Kaleb.

583 However, in relation to the level of impact that might be necessary to cause retinal haemorrhages in the context of BESS, the following exchange occurred (emphasis added):

> WALKER JA: Would it make any difference to the level of impact necessary to — in an accidental context to cause this, would it make any difference to take into account the pre-existing intracranial pressure, possible BESS, certainly hygromas?---**I think that the, um, the effect of a pre-existing brain injury in terms of brain vulnerability on later injury is not known. So, it's possible but I'm not sure that we have any evidence to suggest that that is — is the same thing for the eyes**. **So, for the brain, yes, it's possible but — but the eyes, no, so — so BESS or something doesn't** - - -
>
> FORREST JA: **Well, if you have no evidence, it can't be impossible. It's, you don't know?---Can't be impossible. I think the thing, if we relate it from — from the general to the specific, then in general terms I think, you know, if we — we don't have any evidence then absolutely it's not impossible**. I think we just don't know whether Kaleb had retinal haemorrhages on his original, um, admission on the 13th or 14th. Um, if he did the question of whether that could impact on the pattern of retinal haemorrhages that he was (indistinct) is simply I don't think [we] know. Um, you'd then have to, I suppose revert to a pathophysiological — is there a pathophysiological reason why it might and **I think in the brain with something like BESS, then yes, there is a pathophysiological reason why it might. In the eyes I'm not sure that's the case but I don't think we know**.

584 While the second part of Dr Tully's answer appears to be directed to the consequences if Kaleb had had retinal haemorrhages at the time of his original admission, in my opinion the first part of her answer is directed to the question she was asked by Forrest JA, concerning whether it was impossible that Kaleb's pre-existing conditions might have made him susceptible to the injuries he was observed to have. That is, she accepted that it was 'not impossible' that those injuries made a difference to the level of impact required. In my view, that aspect of her answer is further reflected in the later part of what she said, where she accepted that there is a known pathophysiological reason why BESS may affect the brain, but that for the eyes 'I don't think we know'. She is accepting a degree of uncertainty, even if, to some extent, that uncertainty is linked to the fact that it was not known whether Kaleb had retinal haemorrhages prior to the events of 23 January 2016.

585 In contrast, Professor Wester's evidence was that it was highly probable that the underlying cause of Kaleb's death was a rapidly developing external hydrocephalus, complicated by a spontaneously occurring subdural hygroma/haematoma, causing increased intracranial pressure that most probably elicited an ALTE with cardiopulmonary arrest. His evidence was that BESS, notwithstanding the term 'benign'

(which he said was used to distinguish it from 'more aggressive types of hydrocephalous'), can cause severe complications, including subdural haematomas, which can be a consequence of the stretching of the bridging veins. In that regard his evidence was consistent with Dr Tully's evidence, discussed above. He also gave evidence that BESS can cause increased intracranial pressure.

(a) The evidence on the appeal concerning retinal haemorrhages

586 Professor Eriksson gave evidence that retinal haemorrhages cannot be used to diagnose abusive head trauma. His opinion was that linking specific patterns of retinal haemorrhages to abusive head trauma is incorrect: 'the cause of the retinal haemorrhages cannot be concluded from the different patterns'.

(a) First, he pointed out that it is based on circular reasoning (addressed in greater detail below at [675] to [682], and discussed in Forrest and Emerton JJA's judgment).

(b) Second, he relied upon a study that showed that, where ophthalmologists are 'blinded' to the alleged circumstances in which the infant presented, they can diagnose the alleged cause of the retinal haemorrhages only slightly better than chance.[214]

(c) Third, he gave evidence that the theory that acceleration/deceleration causes retinal haemorrhages has never been proved. He pointed to a Swedish study, which he said demonstrated that there is no relation between shaking and retinal haemorrhages of any kind; rather, retinal haemorrhages are related to non-specific intra-cranial pathology, including raised intracranial pressure.

587 Professor Eriksson also gave evidence concerning Dr Binenbaum's change of position on retinal haemorrhages, as discussed in Forrest and Emerton JJA's judgment. Dr Binenbaum is a leading expert on retinal haemorrhages. He was not called as a witness on the appeal. Nonetheless this Court heard evidence concerning his views about the patterns of retinal haemorrhages associated with abusive head trauma, as published in scientific journals, and also evidence to the effect that he had recently changed his views, and that he now accepted that 'the overlap between intentional and accidental trauma regarding patterns of retinal haemorrhages is greater than previously appreciated' (as Professor Eriksson put it in his evidence). The Court was provided with a summary of what Professor Binenbaum said at a conference, which supported the general description given by Professor Eriksson.

588 Professor Wester also gave evidence that Dr Binenbaum had changed his views, but he understood the change to concern the overlap between retinal haemorrhages caused by shaking and those caused by medical conditions.

589 Professor Högberg also gave evidence about Dr Binenbaum's 'change of position'. He relied on an editorial published by Dr Waney Squier, who commented on Dr Binenbaum's contention, said to have been propounded at a conference, that 'there

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214 Alan Mulvihill et al, 'An Inter-observer and Intra-observer Study of a Classification of RetCam Images of Retinal Haemorrhages in Children' (2010) 95(1) *British Journal of Ophthalmology* 99 ('Mulvihill et al').

is generally more overlap than appreciated between abusive head trauma and accidental retinal haemorrhages.'[215]

590 It is appropriate at this stage to deal with the respondent's further submissions on this aspect of the evidence. First, the respondent pointed out that Dr Tully gave evidence on the appeal that Dr Binenbaum's pronouncements were not a significant change, because this had been known since 2018 (when a paper by Atkinson et al was published[216]) and that she had 'already incorporated' these changes into her practice. I pause to note that Dr Tully did not say whether her 'incorporation' of these changes had occurred before or after the applicant's second trial (which occurred in June 2019). But I infer that it would have occurred *after* Dr Tully prepared her initial report in which she diagnosed Kaleb as having been subjected to inflicted head trauma (prepared in 2016, well before the Atkinson et al paper was published). And, as I discuss further below, Dr Tully's evidence at trial did not acknowledge the findings by Atkinson et al to the effect that a short fall involving occipital impact could cause retinal haemorrhaging that 'mimicked' abusive head trauma. Rather, her evidence at trial was that retinal haemorrhages consistent with abusive head trauma would *only* occur following a fall if the fall was from a 'significant height'.

591 Dr Tully's evidence was that Dr Binenbaum was discussing the differences between retinal haemorrhages caused by *accidental* impact on the one hand and *abuse* on the other and not those caused by other medical conditions. The respondent also put before the Court an abstract of Dr Binenbaum's presentation[217] which, the respondent submitted, confirmed Dr Tully's description.[218] Further, the respondent put before the Court an article published by Dr Binenbaum in December 2021,[219] and submitted as follows:

> In that article, the Doctor considered the patterns of retinal haemorrhage associated with cardiac arrest and CPR. He found that CPR is rarely associated with retinal haemorrhages and that hypoxia is not a significant cause of isolated retinal haemorrhages. He suggests that when retinal haemorrhages are multi-layered, or more than a few in number, or extend outside the posterior pole, another aetiology for the retinal haemorrhage should be sought. In his article, he found that the retinal haemorrhage pattern in AHT cases was bilateral and 'of numerous, diffusely distributed, intraretinal and/or multi-layered retinal

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215 Waney Squier, 'Infant Retinal Haemorrhages Correlate with Chronic Subdural Haemorrhage, not Shaking' (2021) 111(4) *Acta Paediatrica* 714, 715.

216 Norrell Atkinson, Rick van Rijn and Suzanne Starling, 'Childhood Falls with Occipital Impacts' (2018) 34(12) *Paediatric Emergency Care* 1–5 ('Atkinson et al').

217 Abstract of Gil Binenbaum et al, 'Retinal Hemorrhage Patterns: a New Paradigm' (2021) 25(4) *Journal of the American Association for Pediatric Ophthalmology and Strabismus* e3.

218 I note, for completeness, that I do not entirely accept the respondent's characterisation of the abstract of Dr Binenbaum's conference presentation. It appears tolerably plain from the terms of the abstract that Dr Binenbaum was considering medical diagnoses, which he said were 'not addressed' by the 'current paradigm' (as well as the overlap between accidental and abusive injuries). This was one of the reasons that he sought to develop 'a new framework for interpreting retinal findings'. In that new paradigm, he said that interpretation should first seek 'to distinguish between a medical and traumatic cause, not to diagnose abuse'. However, given my views on the nature of the applicant's case and the relevance of the evidence about Dr Binenbaum's views to it, it is unnecessary to explore this further.

219 Gil Binenbaum et al, 'Patterns of Retinal Hemorrhage Associated with Cardiac Arrest and Cardiopulmonary Resuscitation' (2021) 25 *Journal of the American Association for Pediatric Ophthalmology and Strabismus* 324e1 ('Binenbaum 2021').

haemorrhages'.

592 I pause to note that to the extent that the additional material the respondent provided to the Court is new evidence, there is a real issue as to whether this Court should accept it at this stage of the proceeding, given that neither side has had an opportunity to put questions to any of the witnesses about this material. However, given that the applicant did not object to that material being received, I am prepared to admit the material as further evidence on the appeal.

593 In light of the above matters, the respondent submitted that:

> any mooted change of approach to the analysis of retinal haemorrhages that was conveyed by Dr Binenbaum (insofar as any such change could be understood) related to matters of no significance to the present trial, namely, the difference between the consequences of accidental as distinct from abusive trauma. Quite simply, the applicant's case in this Court is not to suggest a case for accident. His case is to suggest an organic cause: on the one hand BESS, or alternatively, birth-related hygroma.

594 It may be accepted that, on the appeal the applicant focused in particular on a medical, not an accidental, cause of Kaleb's collapse and death. But that is not to say that the applicant should be understood to have abandoned the case he ran at trial, namely that Kaleb's pre-existing condition made him susceptible to injury, such that a relatively minor degree of force — as described by the applicant in his record of interview — could have caused the injuries with which he was diagnosed, including the retinal haemorrhages. That is, as I understood it, the case was that Kaleb had BESS and that that either caused his injuries *or* meant that he could have sustained those injuries without having been forcefully shaken.

595 In that sense, I consider that the case the applicant ran at trial, and on appeal although to a lesser extent, was in fact one of accidental injury. That is, although the rough picking up and putting down of Kaleb to which the applicant admitted might be said to be deliberate, the injuries caused by that behaviour were nonetheless accidental in the sense that they were unintended and unforeseen. If that characterisation is correct, then Dr Binenbaum's change in position concerning the difference between accidental and medical causes could be highly relevant to whether retinal haemorrhages carry the diagnostic weight attributed to them by Dr Tully.

596 In relation to retinal haemorrhages, Professor Wester's evidence was that these can be caused by increased intracranial pressure (which is a potential consequence of BESS), resulting in a condition known as Terson syndrome. In addition, he stated that there are other causes of retinal haemorrhages, including cardiopulmonary resuscitation. He said that the correlation between increased intracranial pressure (due to non-traumatic medical causes) and retinal haemorrhages had been demonstrated by Binenbaum et al in a 2013 paper.[220]

597 It is necessary to observe that in the 2013 Binenbaum paper the conclusion was as follows:

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220 Gil Binenbaum et al, 'Patterns of Retinal Hemorrhage Associated with Increased Intracranial Pressure in Children' (2013) 132(2) *Pediatrics* e430 ('Binenbaum 2013').

> Only a small proportion of children with nontraumatic elevated ICP [intracranial pressure] have RHs [retinal haemorrhages]. When present, RHs are associated with markedly elevated OP [lumbar puncture opening pressure], intraretinal, and invariably located adjacent to a swollen optic disc. This peripapillary pattern is distinct from the multilayered, widespread pattern of RH in abusive head trauma. When RHs are numerous, multilayered, or not near a swollen optic disc (eg, elsewhere in the posterior pole or in the retinal periphery), increased ICP alone is unlikely to be the cause.[221]

598 The paper also stated that:

> the hemorrhage pattern observed in Terson syndrome, which is thought to arise from a sudden increase in ICP due to intracranial hemorrhage, is primarily that of preretinal and vitreous hemorrhage and rarely resembles the patterns of hemorrhage seen in AHT [abusive head trauma].[222]

599 However, it is plain that Binenbaum has more recently taken a different view about the use of retinal haemorrhages in the diagnosis of abusive head trauma. As Forrest and Emerton JJA point out, Binenbaum is reported to have said as follows at a conference in 2021:

> **Retinal hemorrhage severity is not perfectly correlated with abuse**. There are missing useful patterns. And it doesn't address medical causes of retinal hemorrhages. With regard to severity, there is generally more overlap than appreciated between abusive and accidental retinal hemorrhages.[223]

600 It is also necessary to observe that, both in the 2013 paper and his more recent conference presentation, Binenbaum's conclusions were not absolute: the particular pattern of retinal haemorrhages associated with abusive head trauma is 'unlikely' to be caused by intracranial pressure alone; the pattern observed in Terson syndrome is 'primarily' a particular pattern, that 'rarely' resembles the pattern seen in abusive head trauma; and the severity of retinal haemorrhages is not 'perfectly correlated with abuse'. Thus, even accepting that Professor Wester may have overstated the significance of the 2013 Binenbaum paper, I do not consider that it — or Binenbaum's work more generally — provides support for Dr Tully's definitive conclusion that intracranial pressure (resulting from BESS) does not cause retinal haemorrhages of the extent and pattern observed in Kaleb. That is particularly so in the context of a criminal trial where guilt must be proved to the criminal standard, not on the basis that it is 'unlikely' that Kaleb's retinal haemorrhages were caused by increased intracranial pressure.

601 Other papers in evidence before us adopted similar language. Thus, for example, a paper by Levin stated that abusive head trauma 'is a *primary* cause, and perhaps the most common cause, of retinal haemorrhages in young children beyond the neonatal period', and observed that 'a diagnosis of abuse should not be made solely based on retinal

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221 Binenbaum 2013, e430.

222 Binenbaum 2013, e433.

223 Patricia Nale, 'Recognising Retinal Haemorrhage Pattern Aids in Diagnosing Abuse', *Ocular Surgery News* (Web Page, 13 April 2021) <https://www.healio.com/news/ophthalmology/20210413/recognizing-retinal-hemorrhage-patterns-aids-in-diagnosing-abuse> (emphasis added).

haemorrhages', even though 'certain retinal findings make the likelihood dramatically high'.[224]

602 I also note that the particular nature and pattern of retinal haemorrhages to which the Levin paper referred was 'when they are multilayered, too numerous to count, extend to the retinal edge (ora serrata) *and occur with macular retinoschisis* with/without surrounding retinal folds'.[225] Levin described the presence of macular retinoschisis[226] as 'critical'.[227] None of the reports concerning Kaleb's retinal haemorrhages, nor any of the evidence at trial or in this Court, identified retinoschisis as present.[228]

603 A paper by Shiau and Levin concluded that '*in general*, elevated ICP [intracranial pressure] does not cause extensive hemorrhagic retinopathy', and reported that there are isolated case reports that 'severe hyperacute ICP elevation, unlike the subacute pressure increase in abusive head injury, in children *may rarely result in extensive retinal haemorrhage*', although the authors considered that these diagnoses are readily distinguished from child abuse.[229] Shiau and Levin also observed that Terson syndrome is '*rare* in nonabused children with intracranial bleeding, with *a maximal incidence estimated at 8 per cent.*'[230] In their conclusion they stated that:

> severe multilayered haemorrhagic retinopathy — in particular with retinoschisis, perimacular folds, and extension of haemorrhages to the ora without the readily diagnosed scenarios discussed here — is **highly suspicious** for AHT. **However, clinicians should maintain open minds to consider how multiple factors might aggravate retinopathy in atypical circumstances**.[231]

604 Another paper, by Piatt, noted the susceptibility of infants with BESS to subdural haematomas, and reported on a child with external hydrocephalus who developed retinal haemorrhages and subdural haemorrhages after a minor head injury.[232] The paper

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224 Alex Levin, 'The SBU Report: A Different View' (2017) 106(7) *Acta Paediatrica* 1037, 1038 (emphasis added) ('Levin').

225 Levin, 1037 (emphasis added).

226 Retinoschisis means the splitting of the retina into two layers.

227 Levin, 1037.

228 I note that Forrest and Emerton JJA appear to conclude that Dr Rodriguez, a neuropathologist who provided a report but was not called at trial or on the appeal, had found retinoschisis in his examination of Kaleb's eyes (see [426], above). Dr Rodriquez's report did not say that, but Forrest and Emerton JJA rely upon the statement that there was 'widespread separation of the neural retina from the retinal pigment epithelium and retinal fragmentation'. It is not clear to me, from the evidence before this Court or at trial, that that statement identified retinoschisis. As I understand it, retinoschisis is the splitting of the retina into two layers. That does not appear to me to be the same as the separation of the retina from the retinal pigment epithelium, nor the same as retinal fragmentation, although without expert evidence to explain Dr Rodriguez's report I am not prepared to reach a definitive conclusion in that regard. However, I note that Dr Leikin, an ophthalmologist who examined Kaleb's eyes on 25 and 26 January 2016, made no mention of retinoschisis or anything capable of being interpreted as retinoschisis. I would expect that, had retinoschisis been observed, one of the experts would have said as much in clear and express terms.

229 Tiffany Shiau and Alex Levin, 'Retinal Hemorrhages in Children: The Role of Intracranial Pressure' (2012) 166(7) *Archives of Pediatrics & Adolescent Medicine* 623, 623 (emphasis added) ('Shiau and Levin').

230 Shiau and Levin, 625 (emphasis added).

231 Shiau and Levin, 627 (emphasis added).

232 Joseph Piatt, 'A Pitfall in the Diagnosis of Child Abuse: External Hydrocephalus, Subdural Hematoma, and Retinal Hemorrhages' (1999) 7(4) *Neurosurgical Focus* 1–8 ('Piatt').

concluded that 'whether or not external hydrocephalus imparts a special susceptibility to ocular haemorrhage is unknown'.[233]

605 A paper by Minns et al found that 38 per cent of children studied who had raised intracranial pressure also had retinal haemorrhages.[234] Further, of the children who had raised intracranial pressure, 100 per cent of those diagnosed with inflicted traumatic brain injury had retinal haemorrhages, whereas only 20 per cent of children with accidental or non-traumatic encephalopathies had retinal haemorrhages. In addition, there were significantly more intraretinal haemorrhages in those with raised intracranial pressure.[235] Minns et al observed that an earlier, comprehensive review of the literature had concluded that, '*in general*, elevated ICP did not cause extensive haemorrhagic retinopathy, but *isolated cases were recognized.*'[236] They observed a relationship between raised intracranial pressure and retinal haemorrhages, but could not reach a conclusion on causality.[237]

606 In contrast, in a paper by Shi et al the authors concluded as follows:

> In our study of 56 children with increased ICP due to primary and secondary hydrocephalus, shunt obstruction, brain tumors, and cerebral edema, we found no cases of increased ICP causing RH [retinal haemorrhage] regardless of the etiology of increased ICP, age of the child, or duration of ICP elevation and despite a clinical severity of increased ICP in all children that warranted intervention. Although acute increased ICP can present in children with a pattern of peripapillary superficial RH in the presence of papilledema, with our results, we provide clear evidence to support the **conclusion that RHs rarely occur in the absence of optic disc swelling and do not present beyond the peripapillary area in the entities we have studied**.[238]

607 Significantly, the paper by Atkinson et al, discussed above, assessed eight children who had struck the back of their heads during a short fall.[239] The authors observed varying degrees of retinal haemorrhages in all eight of the children, with four of those involving bilateral haemorrhages.[240] The authors stated that their findings suggested that children who strike their occiput (ie the back of their head) become symptomatic immediately after they are injured. They said as follows:

> **The children's injuries all mimicked findings seen with AHT.** If witnesses to the events had not been present, a high suspicion of abuse in these cases would have been justified. This high suspicion is justified because of the fact that short-distance falls generally do not cause serious intracranial injury.

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233 Piatt, 5.

234 Robert Minns et al, 'Raised Intracranial Pressure and Retinal Haemorrhages in Childhood Encephalopathies' (2017) 59(6) *Developmental Medicine & Child Neurology* 597, 599 ('Minns et al').

235 Minns et al, 599.

236 Minns et al, 601 (emphasis added).

237 Minns et al, 603.

238 Angell Shi et al, 'Retinal Findings in Young Children with Increased Intracranial Pressure from Nontraumatic Causes' (2019) 143(2) *Pediatrics* 1–8, 6 (emphasis added) (citations omitted).

239 The authors did not have data which allowed them to calculate the exact fall height in each case, but noted that in all eight cases, the fall height was to be regarded as a 'short fall': Atkinson et al, 4. For example, the presenting history of the children included falls while standing, and those from a high chair and a couch.

240 Atkinson et al, 3.

…

> In conclusion, **if a pediatrician or a medical examiner is confronted with a child with a SDH [subdural haematoma] and RH [retinal haemorrhage] and a history of a fall with an occipital impact, in the absence of other signs of maltreatment, accidental injury should be considered**. Further research should be conducted regarding this mechanism of head injury in young children.[241]

This paper is contrary to Dr Tully's evidence at trial that retinal haemorrhages consistent with abusive head trauma would *only* occur following a fall if the fall was from a 'significant height'.

608 Recently, a paper by Thiblin et al concluded as follows:

> The results of the present study indicate that non-birth-related retinal haemorrhage in infants are secondary to intracranial pathology associated with raised intracranial pressure, which may be of traumatic or non-traumatic origin and not the result of traction forces between the retina and vitreous body acting directly on the retinal vessels. Consequently, **the presence or non-presence of RH [retinal haemorrhage] cannot be regarded as a reliable basis for determining the underlying aetiology of the intracranial pathology**. Furthermore, retinal haemorrhage is likely to have low sensitivity for detecting head trauma and thus also for ruling out infant maltreatment, making them of limited value in the investigation of suspected infant abuse.[242]

609 In relation to the possibility that cardiopulmonary resuscitation with chest compression may cause retinal haemorrhages, a paper by Pham et al concluded that:

> **retinal hemorrhage is uncommon after CPR-CC** [cardiopulmonary resuscitation with chest compression], and when it occurs, it does so in the setting of coexisting risk factors for hemorrhage with a very mild hemorrhagic retinopathy confined to the posterior pole.[243]

610 Again, it may be noted that the paper concludes that retinal haemorrhages are 'uncommon' after cardiopulmonary resuscitation, not that they never occur.

611 Professor Wester also relied on an article by Scheller, which was a study of ten infants (all less than ten months old) admitted to hospital for one or more of the following symptoms: large head circumference; alteration of consciousness; emesis; or irritability.[244] None had any evidence of neglect, external injury, fractures or neck injury. No caregiver confessed to child abuse. All had a subdural collection of some

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241 Atkinson et al, 4 (emphasis added) (citations omitted).

242 Ingemar Thiblin et al, 'Retinal Haemorrhage in Infants Investigated for Suspected Maltreatment is Strongly Correlated with Intracranial Pathology' (2021) 111(4) *Acta Paediatrica* 800, 807 (emphasis added).

243 Hang Pham et al, 'Retinal Hemorrhage After Cardiopulmonary Resuscitation With Chest Compressions' 2013 34(2) *American Journal of Forensic Medicine and Pathology* 122, 123 (emphasis added).

244 Joseph Scheller, 'Infantile Retinal Haemorrhages in the Absence of Brain and Bodily Injury' (2017) 106(12) *Acta Paediatrica* 1902 ('Scheller').

kind, but no brain injury. Several of the infants had bilateral multilayer retinal haemorrhages. The Scheller article stated that:

> The retinal haemorrhages found in these cases suggest that **macrocephaly and chronic subdural hygroma[245] may make a child prone to develop retinal haemorrhages.** It is also possible when subdural hygroma is present, an event that triggers a small acute subdural or subarachnoid haemorrhage can also cause unilateral or bilateral retinal haemorrhages. **These cases also suggest that the triggering event need not be potent enough to cause any brain injury**.[246]

612 The Scheller article concluded that:

> **[E]xtensive multilayer retinal haemorrhages occur in infants who have not suffered any apparent brain injury**. Clinicians should reassess the importance of retinal haemorrhages in the setting of suspected abusive head trauma.[247]

613 Dr Tully was not asked about the Scheller article in the course of her evidence on the appeal.

614 Further, as noted earlier, Professor Eriksson's evidence, based on a paper by Mulvihill et al, was that, where ophthalmologists were 'blinded' to the suspected cause of the retinal haemorrhages, they could not reliably identify whether an image of retinal haemorrhages was from a case of abusive head trauma, a medical disease or from accidental trauma. Mulvihill et al said as follows:

> We have demonstrated that a clinical classification of RetCam images of retinal haemorrhages in children, based on the generally held defining features of haemorrhages in different retinal layers, lacks consistency between examiners and even on re-examination by the same examiner.[248]

615 The article also observed that:

> Retinal haemorrhages after some days may assume a different appearance and not be like a typical textbook description. This may account for the limited inter-observer agreement.[249]

(b) Conclusions on the evidence concerning retinal haemorrhages

616 Ultimately, in my opinion, the evidence before us on the appeal reveals that there is a real disagreement between experts about the significance of retinal haemorrhages and their use in the diagnosis of abusive head trauma, even where they are widespread, multilayered and bilateral. In particular, there is disagreement about:

(a) the possible causes of extensive retinal haemorrhages of the nature and pattern associated with abusive head trauma;

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245 Professor Wester's evidence, in cross-examination, was that macrocephaly with subdural haematomas is a synonym for BESS.

246 Scheller, 1903 (emphasis added).

247 Scheller, 1904 (emphasis added).

248 Mulvihill et al, 101.

249 Mulvihill et al, 101.

(b) whether retinal haemorrhages of the nature and pattern associated with abusive head trauma can be caused by a short fall (from less than one metre) — the relevance in the present case being that if a short fall could cause such retinal haemorrhages, then it is possible that, in a child with BESS, placing him down in his cot 'a bit rough' could cause such haemorrhages;

(c) whether retinal haemorrhages of the nature and pattern associated with abusive head trauma are also seen in children with non-traumatic brain injuries, such as spontaneously occurring subdural collections and increased intracranial pressure; and

(d) whether ophthalmologists have the ability to reliably interpret images of the retina so as to conclude that abusive head trauma is the definitive cause of the retinal haemorrhages.

617 Perhaps more importantly, even those studies this Court received in evidence that support the proposition that retinal haemorrhages that are widespread, multilayered and bilateral are associated with abusive head trauma, rather than a medical or accidental cause, use language of qualification, such as 'unlikely' or 'rarely' or 'generally' or 'highly suspicious'. And even those studies accept that there may be atypical cases.

618 In my opinion the evidence before this Court concerning the connection of retinal haemorrhages with abusive head trauma is not sufficient to support Dr Tully's definitive position at trial that the retinal haemorrhages must have been caused by the application of significant force. In particular, the evidence before this Court indicates that the following evidence given by Dr Tully at the trial was not appropriately qualified to reflect the lack of scientific and medical certainty about this issue (emphasis added):

> What do you say about the proposition that he was rendered more vulnerable because of the pre-existing condition?---**I think the key here is the presence of those retinal haemorrhages**. So, I think there is no doubt in my mind that Kaleb had a pre-existing abnormality inside his head; we know he did. He had enlarged extra-axial spaces and subdural fluid. And I think as we said yesterday, I think that may — it may confer slightly increased risk of him being vulnerable to subdural bleeding. As I said, there is some evidence that the converse may apply, in that, the fact that there's more fluid around the brain actually buffers it more, against impact against the skull. So, I think we have to acknowledge that there may be some vulnerability in relation to that. However, **that vulnerability does not extend to his eyes and the presence of this pattern of such severe retinal haemorrhaging is indicative of significant, high level forces being applied to Kaleb just prior to his collapse.**

619 Similarly, Dr Tully made the following statements concerning the significance of the retinal haemorrhages seen in Kaleb:

(a) Dr Tully said that 'we *only* see this specific pattern of retinal haemorrhaging in association with trauma in situations of significant force, so high velocity motor vehicle accidents, crush injuries to the head, falls from a height' (emphasis added).

(b) Dr Tully said that 'if the triad is of subdural haemorrhages, extensive multilayered, all over the retina retinal haemorrhages and severe global hypoxic

brain injury sufficient to cause death, then that is significant force that has required that, yes'.

(c) Dr Tully said that 'the evidence base would suggest that, with this combination of findings even in the presence of an enlarged extra-axial space, that the retinal haemorrhages and the severe damage to the brain would indicate significant force. The subdural haemorrhage, I accept, maybe it's less — we don't know, it's unclear'.

620 As noted above, I consider that Dr Tully's evidence in this regard appears to be inconsistent with the article by Atkinson et al, upon which Dr Tully relied in the course of her evidence. That article was quite clear that a short fall with occipital impact could mimic abusive head trauma.

621 Not only does my view that Dr Tully failed to appropriately qualify her evidence emerge from the literature concerning retinal haemorrhages discussed above, it is consistent with part of Dr Tully's own evidence in this Court, extracted above. Her evidence was that it was 'not impossible' that Kaleb's pre-existing conditions had caused the retinal haemorrhages and that, while there is a pathophysiological reason why BESS may predispose a child to brain injury (such as, I infer, a subdural collection), '*in the eyes I'm not sure that's the case but I don't think we know*'.

(5) Conclusion on effect of new evidence

622 In light of these matters, I consider that, had the new evidence that this Court has heard been presented to the jury, it would not have been open to the jury to conclude, beyond a reasonable doubt, that the applicant had committed child homicide either by an unlawful and dangerous act, or by criminal negligence.

623 That is, the new evidence provided such a doubt as to (a) the cause of Kaleb's death and (b) the level of force required to produce the injuries to Kaleb that, in my opinion, had the jury heard it, they must have had a reasonable doubt. That is because a reasonable hypothesis consistent with innocence would not have been excluded.

624 As to the first proposition — that the new evidence raised a sufficient doubt about the *cause* of Kaleb's death, I accept that at trial causation was not seriously put in issue by the defence (although of course the prosecution had to prove causation as an element of the offence, and causation was not formally conceded). However, had the new evidence been available then it might well be that the trial would have been run differently, and the defence case might have been that causation was not proved beyond a reasonable doubt. I do not consider that, when this Court considers the effect of the new evidence, it is bound to consider that effect only in relation to the case that was actually run at trial (in contrast to consideration of ground 5, which is limited to the manner in which the case was run at trial).

625 As to the second proposition, once it is accepted, on the basis of the new evidence, that BESS could not properly be excluded, and that that condition could cause a variety of pathological consequences — including a susceptibility to subdural haemorrhages, increased intracranial pressure and, potentially, retinal haemorrhages — then, in my view, had the new evidence been led at trial the jury could not have concluded beyond

a reasonable doubt that the applicant's actions on 23 January 2016 necessarily involved significant force, or were anything other than those that he described (which, in my opinion, did not constitute any admission of handling or shaking Kaleb with significant force).

626 That is, I consider that, even if the jury accepted that the applicant had caused Kaleb's death by his handling of Kaleb that day, it would not have been open to the jury to have concluded, beyond a reasonable doubt, either that:

(a) a reasonable person in the position of the applicant, performing that act, would have realised that he was exposing Kaleb to an appreciable risk of serious injury; or

(b) that his conduct involved a great falling short of the standard of care which a reasonable person would have exercised in all of the circumstances, and that there was a high risk that death or serious injury would result from that conduct.

627 Those conclusions are based on the new evidence concerning BESS, discussed above, together with the following matters.

628 First, Kaleb had had an increasing head circumference over the course of the three months prior to his death, crossing from the 3rd percentile to, ultimately, the 95th percentile. Professors Wester, Högberg and Ditchfield, and Drs Tully and Iles, all acknowledged that that was a concern. Dr Iles' evidence on the appeal was that there are a number of causes of increasing head circumference, including hygromas, and that they can be traumatic or atraumatic.

629 Secondly, Kaleb had been sufficiently unwell in the weeks prior to his death for his mother to seek medical treatment for him on several occasions.

(a) On 10 November 2015, Kaleb's medical records contained a notation indicating that at five weeks of age, Kaleb had an increasing head circumference of greater than 90 per cent that had crossed two centiles and a bulging, pulsatile fontanelle.

(b) On 28 December 2015, Kaleb presented to the Emergency Department of Casey Hospital because he was vomiting.

(c) On 6 January 2016, the child maternal health nurse referred Kaleb to Dr Zhou, a general practitioner, due to his head circumference increasing from the 15th centile to the 85th centile.

(d) On or about 11 January 2016, Dr Zhou referred Kaleb to the Monash Medical Centre Outpatient Clinic.

630 On 14 January 2016, Kaleb was admitted to Casey Hospital because he had been vomiting, was not feeding well, was lethargic, had a raised fontanelle and his head circumference was increasing. These were all symptoms of raised intracranial pressure. An ultrasound found that two of Kaleb's ventricles were dilated and that his extra-axial spaces also appeared to be larger than normal, as well as probable fluid in the subdural space, but no evidence of bleeding. A doctor at Casey Hospital observed that Kaleb had

'sunsetting eyes', which are also a symptom of increased intracranial pressure. In addition, vomiting is a symptom of raised intracranial pressure.

631 On 15 January 2016, Kaleb was transferred from Casey Hospital to the Monash Medical Centre, where an MRI scan was performed of Kaleb's head. It confirmed the results of the ultrasound: mild ventricular dilation, together with small bilateral frontal subdural hygromas, but no sign of recent haemorrhage. A discharge summary from Monash Medical Centre stated that the diagnosis on discharge included 'macrocephaly'.

632 A tap of the fluid on Kaleb's anterior fontanelle was considered, for diagnostic and therapeutic purposes (the diagnostic purpose being to ascertain whether there was blood present in the extra-axial spaces, and the therapeutic purpose being to relieve the increased intracranial pressure). However, it was not performed. Dr Tully's evidence was that she 'would have liked [a tap] to have been done'.

633 Kaleb's eyes were not examined at Casey or Monash, thus there is no evidence one way or the other as to whether Kaleb had retinal haemorrhages in the period from 14 to 17 January 2016. If he had had retinal haemorrhages at that stage, it is possible that they could have persisted for up to a week.

634 No bruising, fractures or other injuries were observed in the course of Kaleb's admission from 14 to 17 January 2016.

635 It is clear from the results of the tests at Casey and Monash that Kaleb had a subdural collection as at 14 January 2016. That was later identified as a subdural haematoma, that is a collection containing blood, on the basis of the existence of membranes around the collection identified by Dr Iles on the autopsy and on the basis of Professor Ditchfield's initial report on the MR images from 15 January 2016. A subdural collection is pathological. That subdural collection could have been up to three weeks old.

636 It is also clear that Kaleb had increased intracranial pressure at the time of his admission to hospital on 14 January 2016, and that a therapeutic tap was not performed to relieve that pressure.

637 As a consequence of these pre-existing conditions, Kaleb was more vulnerable to subdural haemorrhage. That is, a subdural haemorrhage could occur spontaneously, or alternatively with 'trivial forces'. Thus it was not possible to state with certainty what degree of force (if any) might have been required to cause a further subdural haemorrhage on 23 January 2016.

638 In addition, at the time of his admission on 23 January 2016:

(a) Kaleb had no bruising that could be linked to an allegation of shaking;[250]

(b) Kaleb had no fractures of any kind;

(c) no MRI scan of Kaleb's spine was performed, thus it was not known whether Kaleb had any trauma to the third or fifth cervical spine as a result of

[250] He had some bruising as a consequence of medical intervention.

hyperflexion to the neck (being an injury commonly associated with abusive head trauma);

(d) CPR had been performed on Kaleb shortly prior to his admission to hospital, which could have caused or contributed to his retinal haemorrhages (which were not observed until 25 and 26 January 2016).

639 Notwithstanding these matters, Dr Tully's evidence at trial and before this Court was that Kaleb's pre-existing conditions did not explain the injuries with which he presented on 23 January 2016 — in particular, the retinal haemorrhages. It is for that reason that I have agreed with Forrest and Emerton JJA as to the resolution of ground 5: on Dr Tully's evidence at trial, which was not contradicted or undermined by any countervailing expert evidence, I consider that it was open for the jury to conclude, beyond a reasonable doubt, that the applicant had handled Kaleb with sufficient force to satisfy the relevant tests for child homicide.

640 However, in my opinion when the evidence given by Dr Tully and Dr Iles at trial is considered in light of the evidence this Court heard on the appeal, I consider that it would not have been open for the jury to accept Dr Tully's definitive evidence that Kaleb must have been subjected to significant force in order to cause his injuries. Or, to put it differently, I consider that the totality of the evidence at trial and admitted in this Court was such that a reasonable hypothesis consistent with innocence had not been excluded.

(a) The first such hypothesis was that the applicant's description of his handling of Kaleb was correct, and had been sufficient to cause Kaleb's death, but not so forceful as to rise to the level required to prove child homicide.

(b) The second such hypothesis was that Kaleb's injuries were not caused by the manner in which the applicant handled Kaleb, but were caused by his pre-existing condition, namely BESS.

641 I note that I make no positive finding either that Kaleb's death was caused by BESS or as to the nature of the manner in which the applicant handled Kaleb and the level of force involved. Nor do I make any positive finding that the applicant did not shake Kaleb with significant force. Ultimately, this is a case that invokes the passage from *Henderson* extracted at the commencement of these reasons;[251] namely one where, when the new evidence is considered, the applicant's guilt has not been proved beyond a reasonable doubt. That is because the cause of Kaleb's injuries is uncertain and because, even if caused by the applicant, the level of force required to produce Kaleb's injuries was also uncertain. Thus I do not consider that the necessary elements of child homicide were proved beyond a reasonable doubt. It may be *likely* that the applicant shook Kaleb with such force that he caused his injuries. It may also be *likely* that he did so either by an unlawful and dangerous act or by criminal negligence. But that is not sufficient.

642 It is important to emphasise that, as should be clear from the analysis above, I have not concluded that a diagnosis of abusive head trauma, based on the presence of subdural haemorrhages, retinal haemorrhages of the relevant nature and pattern, and hypoxic

251 [2010] EWCA Crim 1269, [1] (Moses LJ for the Court).

ischemic encephalopathy, cannot be made or is inherently unreliable or otherwise inadmissible. That is, I have not concluded that the SBU report and/or the evidence of Professors Eriksson, Högberg and Wester concerning the reliability of a diagnosis of abusive head trauma based on the 'triad' is to be preferred over the evidence of Dr Tully and Dr Iles on this issue. Rather, my conclusion is based on the new evidence of Professor Eriksson, Professor Högberg, Professor Wester, Professor Ditchfield and Dr Tully concerning BESS and retinal haemorrhages, together with my review of the studies and articles tendered in evidence that are relevant to those issues. That is, my conclusion is squarely based on Kaleb's particular medical history and pre-existing conditions.

643 For these reasons, I would uphold ground 3, allow the appeal and order that an acquittal be entered.

(6) Alternative conclusion: a reasonable possibility of acquittal

644 In addition, it is appropriate to record for completeness that, if I am wrong in my conclusion that the new evidence is such as to mean that a verdict of guilty was not open to the jury, I would in the alternative conclude that the new evidence demonstrated that a substantial miscarriage of justice had occurred, on the basis that had the evidence been before the jury, and accepted by them, there is a reasonable possibility that they would have acquitted the applicant. As I explain below, I do not consider that the authorities that distinguish between fresh and new evidence preclude me from reaching that conclusion.

645 It is necessary to bear in mind that, notwithstanding what is sometimes presented as an absolute rule concerning the admissibility of new evidence, the distinction between 'fresh' and 'new' evidence is not one drawn in the legislation governing appeals to this Court. It is one that has been developed by the courts. But the source of the Court of Appeal's power to allow an appeal on the ground of fresh or new evidence is found in Division 1 of Part 6.3 of the *Criminal Procedure Act 2009* ('*CPA*'), which governs appeals against conviction. Section 274 of the *CPA* provides that a person convicted of an offence may appeal to the Court of Appeal against the conviction on any ground of appeal if the Court of Appeal gives the person leave to appeal. Section 276 provides that the Court of Appeal must allow the appeal if the appellant satisfies the court that:

> (a) the verdict of the jury is unreasonable or cannot be supported having regard to the evidence; or
>
> (b) as the result of an error or an irregularity in, or in relation to, the trial there has been a substantial miscarriage of justice; or
>
> (c) for any other reason there has been a substantial miscarriage of justice.

646 The power of this Court on an appeal to receive evidence not adduced at trial is to be found in s 276(1)(c) of the *CPA*: that is, where the court is satisfied that the evidence demonstrates that there has been a 'substantial miscarriage of justice'. While the tests associated with the distinction between 'new' and 'fresh' evidence are of assistance in determining whether there has, in a particular case, been a substantial miscarriage of

justice, they cannot replace the statutory text.[252] Nor should those tests be regarded as having crystallised into absolute or inflexible rules.[253] That is, the question of whether there has been a substantial miscarriage of justice remains the ultimate question that an appellate court must assess when determining whether to allow an appeal based on evidence that was not adduced at trial.[254] As Winneke P observed in *AHK*:

> [A]t the end of the day, it should not be forgotten that the expressions of judicial opinion to which I have referred are **practical guidelines which do not detract from the force of the fundamental principle that an appellate court must allow an appeal if a miscarriage of justice is shown to have occurred**. An appellate court will always receive 'fresh evidence' if it can be clearly shown that the failure to receive it might have the result that an unjust conviction is permitted to stand.[255]

647 To similar effect, in *Gallagher* Gibbs CJ said as follows:

> Although many cases have provided a gloss on the words of s 6 [of the *Criminal Appeal Act 1912* (NSW)] and similar provisions, it is important to remember that the fundamental question is whether a miscarriage of justice has occurred, and that the principles that may be extracted from the authorities 'should not ... be regarded as absolute or hard and fast rules'. The circumstances of cases may vary widely, and it is undesirable to fetter the power of Courts of Criminal Appeal to remedy a miscarriage of justice. …
>
> The authorities disclose three main considerations which will guide a Court of Criminal Appeal in deciding whether a miscarriage of justice has occurred because evidence now available was not led at the trial. The first of these, that the conviction will not usually be set aside if the evidence relied on could with reasonable diligence have been produced by the accused at the trial … is not a universal and inflexible requirement: the strength of the fresh evidence may in some cases be such as to justify interference with the verdict, even though that evidence might have been discovered before the trial.[256]

648 More recently, Priest JA (which whom Maxwell P and Kidd AJA agreed) observed in *Bowden* that the rules concerning fresh and new evidence 'should be applied so as to serve, rather than frustrate, the interests of justice'.[257] His Honour went on to cite with approval the remarks of Winneke P quoted above. While Maxwell P and Kidd AJA, in separate concurring reasons, emphasised 'the stringency of the "new evidence" test',[258] it does not appear to me that their Honours were departing from the remarks made by Winneke P in *AHK* or Gibbs CJ in *Gallagher*.

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252 *Baini v The Queen* (2012) 246 CLR 469, 476 [14], 479 [25] (French CJ, Hayne, Crennan, Kiefel and Bell JJ); [2012] HCA 59.

253 *R v AHK* [2001] VSCA 220, [8] (Winneke P) ('*AHK*'); *Gallagher v The Queen* (1986) 160 CLR 392, 395 (Gibbs CJ); [1986] HCA 26 ('*Gallagher*').

254 *AHK* [2001] VSCA 220, [8] (Winneke P). See also *Weng v The Queen* (2013) 236 A Crim R 299, 311 [45] (Osborn JA); [2013] VSCA 221; *Bowden* (2017) 54 VR 135, 142–3 [33] (Priest JA); [2017] VSCA 46; *Ramlagun v The Queen* [2015] VSCA 337, [50] (Whelan JA, Santamaria JA agreeing at [70], Kaye JA agreeing at [71]).

255 *AHK* [2001] VSCA 220, [8] (emphasis added) (citations omitted).

256 *Gallagher* (1986) 160 CLR 392, 395; [1986] HCA 26 (citations omitted).

257 *Bowden* (2017) 54 VR 135, 142 [32]; [2017] VSCA 46.

258 *Bowden* (2017) 54 VR 135, 138 [7]; [2017] VSCA 46.

649 That said, it would be an exceptional case where a court would conclude that evidence that could, in the exercise of reasonable diligence, have been discovered and adduced at trial, leads to a substantial miscarriage of justice on the basis that, had it been led, it might have led the jury to reach a different verdict. That is, as Priest JA put it in *Bowden*, 'this Court will be slow to conclude that justice has miscarried in circumstances where the applicant has been convicted following a trial free from irregularity, but where evidence that could have been adduced was not'.[259] Importantly, though, the authorities to which I have referred leave open the possibility that, in an exceptional case, a court might reach that conclusion.

(a) How the applicant put his case on the evidence of Professors Eriksson, Högberg and Wester

650 By and large, the applicant's case on the appeal was that the evidence of Professors Eriksson, Högberg and Wester was 'new' rather than 'fresh'. In particular, ground 3 was framed in the language adopted by the courts in relation to cases of new evidence. The applicant's written and oral submissions were also framed on that basis. And that is certainly how the applicant's case was understood by the Crown. I say 'by and large' because, in his written submissions dated 9 August 2021, the applicant observed that 'confines to [the] reasonable diligence of an accused (or his or her counsel) must exist — and particularly so where expert opinion is subject to such a controversy'. He submitted that, 'despite reasonable diligence, some matters of medicine and science extend beyond ordinary and reasonable expectations of counsel's knowledge and ability to navigate such matters'. To that limited extent, it might be said that the applicant sought to suggest that the evidence of Professors Eriksson, Högberg and Wester was not available in the exercise of reasonable diligence of counsel.

651 After the evidence had been heard on the application for leave to appeal, the Court sought further submissions from the parties on whether the evidence was properly characterised as fresh, rather than new, and submissions on whether it may be necessary for the applicant to amend ground 3 if he wished to contend that the evidence was fresh.

(i) The applicant's submissions on new vs fresh evidence

652 Following that invitation, the applicant filed further submissions seeking leave to amend ground 3 and submitting that the evidence of Professors Eriksson, Högberg and Wester was not available to the applicant at trial in the exercise of reasonable diligence. He also relied upon the fact that the three experts, in particular in their oral evidence, had addressed matters that were plainly fresh. He gave as an example the evidence that an expert ophthalmologist, whose work underpins much of the current thinking on retinal haemorrhages and their link to abusive head trauma, Dr Binenbaum, had changed his view on these issues in 2021. Ultimately, although he maintained that the evidence of Professors Eriksson, Högberg and Wester was such as to require a conclusion that a jury must have entertained a doubt had the evidence been led at trial, he also contended that the evidence was not available to the applicant in the exercise of reasonable diligence. In that sense he contended that it should be characterised as fresh.

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259 *Bowden* (2017) 54 VR 135, 146 [38]; [2017] VSCA 46.

(ii) The respondent's submissions on new vs fresh evidence

653 In contrast, the respondent contended that the evidence was new, and submitted that leave to amend ought not be granted at that late stage in the proceeding. The respondent emphasised that until the close of oral evidence, the applicant had maintained that the evidence in question was new, not fresh. The respondent contended that, had the applicant raised at the hearing the proposition that the evidence was fresh, not new, the respondent would have conducted its case differently. For example, it would have sought to challenge the applicant's assertion that 'the level and extent of forensic experience and expertise could not realistically, if at all, be found in Australia'. I note immediately that I have not relied upon this passage from the applicant's submissions in forming my conclusion, thus it is not necessary to consider this aspect of the respondent's submissions further.

654 The respondent also submitted that it was plain that the applicant could, at trial, have mounted a case that BESS was an alternative cause of Kaleb's death, but deliberately did not do so. Similarly, it is known that trial counsel was aware of the SBU Report, but chose not to deploy it. Further, in relation to the proposition that the evidence of Professors Eriksson, Högberg and Wester might be fresh because some of the material to which they referred, or which was otherwise put before this Court, post-dates the trial, the respondent submitted as follows:

> [T]he simple fact that publications may have been referred to which post-date the trial does not qualify the content of those publications as 'fresh' unless it can be said that in some manner, perhaps, the science upon which expert opinion is based has developed — relevantly — in the intervening period. Then, of course, the task is to explain how that development might have made a difference at trial.

655 The respondent further submitted that the applicant had not particularised any such material, save for the evidence concerning Dr Binenbaum. The respondent submitted that it 'cannot be expected to sift through the voluminous material before the court to address any other *potential* arguments of fresh evidence' (emphasis in original). If there was some other particularised case to be made, the respondent submitted that it would need to be made aware of it so that 'a meaningful response might be made'. In so far as the evidence about Dr Binenbaum was concerned, the respondent made detailed submissions concerning that evidence, and provided the Court with a copy of an article published by Dr Binenbaum in 2021 directed to that issue.[260] (I have dealt with the significance of Dr Binenbaum's evidence to the substantive issues in the trial earlier in my reasons).

(iii) Consideration of the new vs fresh evidence issue

656 The policy considerations that underpin the distinction drawn between new and fresh evidence are important. It would subvert the trial process if an accused person was free to mount on appeal a case that could have been mounted at trial. Trial by jury does not mean trial by jury in the first instance and trial by the judges of the Court of Appeal in

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260 Binenbaum 2021.

the second instance.[261] Those observations apply as much to expert evidence as to lay evidence.

657 However, it must also be accepted that expert evidence has some relevant differences from lay evidence. Where an accused seeks to rely on expert evidence not given at trial, some years after the trial has occurred, there may be cases where it is difficult to characterise the evidence as either completely 'new' or completely 'fresh'. That is because, where an expert is giving an opinion about scientific or medical issues, the basis for those opinions is not necessarily static. Scientific and medical research and knowledge may develop over time, as the respondent appeared to accept. Likewise, scientific and medical opinions — and the strength and validity of those opinions — may change over time.[262] An opinion that was orthodox and accepted at some point in time may later come not to be so accepted. Thus, even in a case where a particular expert could have been called at trial, but was not, it may be that the research in the particular field has developed in such a way as to render the expert's later opinion fresh, rather than new. It is thus not always sufficient simply to observe that the expert could have been called at trial. Much will depend on the particular evidence to be given and the state of the discipline or field to which it relates.

658 I accept that the evidence of Professors Eriksson, Högberg and Wester was available to the applicant at the time of trial in 2018 (in the sense that it appears that each of those witnesses could have been approached to give evidence). Thus it might be said that the witnesses were fresh, but their evidence was not.[263] I also accept that the applicant had obtained an expert report from Professor Duflou, on similar issues to those addressed by Professors Eriksson, Högberg and Wester, and that he resolved not to call Professor Duflou.

659 However, in my opinion the evidence of Professors Eriksson, Högberg and Wester does not neatly fit into the categories of 'fresh' or 'new' evidence. That is because it involved opinions that, while they might have been held at the time of the trial, were also based on research conducted and articles published since the trial. The scientific validity of a diagnosis of abusive head trauma is currently a matter of controversy, and work directed to the resolution of that controversy is ongoing. In that sense, this area of medical or scientific knowledge is developing. Further, the issues raised by the evidence of Professors Eriksson, Högberg and Wester are complex and are of such importance that their evidence warrants admission in this case.

660 In addition, this Court heard evidence from Dr Tully, Dr Iles and Professor Ditchfield, adduced by the respondent, which could have been given at trial, but was not. Some of that evidence raised a real question as to whether, in relation to the evidence she gave at trial, Dr Tully had properly excluded BESS as a pre-existing condition that Kaleb had at the time of the events of 23 January 2016.

661 Ultimately, I do not consider that it is necessary to resolve the character of the evidence in the present case. That is because, even if the evidence is properly characterised as new, rather than fresh, that is not determinative of whether the appeal should be allowed. Rather, the statutory test for determining an appeal against conviction is whether the

261 *R v Pendleton* [2002] 1 All ER 524, 534 [17] (Bingham LJ); [2001] UKHL 66.

262 See, eg, *R v Cannings* [2004] 1 All ER 725, 768 [178] (Judge LJ); [2004] EWCA Crim 1.

263 *Henderson* [2010] EWCA Crim 1269, [81] (Moses LJ for the Court).

Court considers that there has been a substantial miscarriage of justice. Thus, this Court may consider and act upon the evidence of Professors Eriksson, Högberg and Wester, and the evidence of Dr Tully, Dr Iles and Professor Ditchfield, if it considers that that evidence makes good the proposition that a 'substantial miscarriage of justice has occurred', consistently with the remarks of Winneke P in *AHK* and Gibbs CJ in *Gallagher*.

662 Thus, in this particular and exceptional case, I consider that the evidence adduced in this Court reveals a significant miscarriage of justice because, as I explain below, I consider that it raises a significant possibility that, if believed, it would have led the jury to acquit.

(b) Relevance, admissibility and credibility

663 In *R v Nguyen* this Court stated that, ordinarily, a court will not be satisfied that a 'fresh evidence' ground is made out unless:

(a) the evidence was not available, or could not with reasonable diligence have become available at the trial;

(b) the evidence is relevant and otherwise admissible;

(c) the evidence is apparently credible (or at least capable of belief); and

(d) there is a significant possibility (or maybe a likelihood) that the evidence, if believed, would have led the jury, acting reasonably, to acquit the applicant if the evidence had been before it at the trial.[264]

664 Although I have resolved that it is not necessary to characterise the evidence of Professors Eriksson, Högberg and Wester for the purposes of the present proceeding, so that issue (a) in a sense does not arise, I consider that it is appropriate to apply this framework to resolution of the present application. In particular, I consider that propositions (b) and (c) are relevant to the consideration of the evidence of Professors Eriksson, Högberg and Wester.

665 As to whether the evidence is 'relevant and otherwise admissible', the evidence of Professors Eriksson, Högberg and Wester is plainly relevant. In my view it is also 'otherwise admissible', by reason of s 79 of the *Evidence Act 2008*. Although there was some attempt by the Crown to challenge the expertise of these three witnesses, by reference to the time at which they ceased to practice and by reference to their lack of clinical experience in some respects, in my view none of those matters meant that the three experts lacked the expertise necessary for them to express the opinions that they did. To put it another way, had the applicant sought to adduce their evidence at trial, it would have been admissible.

666 In that regard I note that, when a court is considering the admissibility of expert evidence under s 79(1), it does not engage in an assessment of the reliability of the evidence. This Court held in *Tuite v The Queen*[265] that the reliability of evidence is not

264 *R v Nguyen* (1998) 4 VR 394, 400–1 (Kenny JA, Winneke P and Callaway JA agreeing at 395).

265 (2015) 49 VR 196; [2015] VSCA 148 ('*Tuite*').

relevant to its admissibility under s 79(1). That was because the language of s 79(1) leaves no room for reading in a test of evidentiary reliability as a condition of admissibility.[266] Thus, following *Honeysett v The Queen*,[267] this Court observed that s 79 only imposed two conditions on admissibility. The first was that the person giving the opinion evidence 'has specialised knowledge'.[268] The second was that the opinion be 'wholly or substantially based' on that specialised knowledge.[269] The Court observed as follows:

> [T]he knowledge must be 'based on the person's training, study or experience'. To take an example discussed in argument, a medical specialist with expertise in occupational lung disease may have come up with a new theory about the link between a particular form of lung disease and a particular industrial emission. Notwithstanding its novelty, the theory could properly be viewed as part of the expert's 'specialised knowledge' provided that the theory was demonstrably based on 'the person's training, study or experience'. **Once that was established, it would be no objection to admissibility that there was dispute in the relevant field about whether the theory was 'correct'**.[270]

667 The Court thus concluded as follows, in relation to s 79:[271]

> It follows … that a person's knowledge may qualify as 'specialised knowledge' for the purposes of s 79(1) even if the area of knowledge is novel or the inferences drawn from the facts have not been tested, or accepted, by others. The position would have been different if, instead, s 79(1) had provided that an opinion was only admissible if shown to be based on a 'reliable' or 'established' body of knowledge. No such language was used, however, and the legislative history makes clear that this was a deliberate legislative choice.[272]

On this issue *Tuite* has since been followed in other cases.[273]

668 There can be no doubt, in my opinion, that the evidence of Professors Eriksson, Högberg and Wester satisfied the requirements that they have specialised knowledge and that their opinions were based on that specialised knowledge. I note that Professors Eriksson and Wester accepted in cross-examination that their evidence was based on a minority view about how abusive head trauma might be proved. However, that does not render

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266 *Tuite* (2015) 49 VR 196, 217 [70] (Maxwell ACJ, Redlich and Weinberg JJA); [2015] VSCA 148.

267 (2014) 253 CLR 122, 131–2 [23] (French CJ, Kiefel, Bell, Gageler and Keane JJ); [2014] HCA 29.

268 *Tuite* (2015) 49 VR 196, 217 [72] (Maxwell ACJ, Redlich and Weinberg JJA); [2015] VSCA 148.

269 *Tuite* (2015) 49 VR 196, 217 [73] (Maxwell ACJ, Redlich and Weinberg JJA); [2015] VSCA 148.

270 *Tuite* (2015) 49 VR 196, 218 [76] (Maxwell ACJ, Redlich and Weinberg JJA); [2015] VSCA 148 (emphasis added) (citations omitted).

271 In *Tuite* the Court went on to consider the role of reliability in relation to admissibility of evidence under s 137 of the *Evidence Act*. It concluded that reliability of expert evidence can be considered under that section: (2015) 49 VR 196, 221–2 [85]–[87] (Maxwell ACJ, Redlich and Weinberg JJA). There is now some doubt as to whether that conclusion can stand, in light of the High Court's decision in *IMM v The Queen* (2016) 257 CLR 300; [2016] HCA 14. See, eg, *Xie v The Queen* [2021] NSWCCA 1, [300] (Bathurst CJ, R A Hulme and Beech-Jones JJ). See also Gary Edmond, 'Icarus and the Evidence Act: Section 137, Probative Value and Taking Forensic Science Evidence "At Its Highest"' (2017) 41(1) *Melbourne University Law Review* 106, 152. However, even if that be so, there is no reason to doubt the correctness of this Court's approach to s 79 in *Tuite*.

272 *Tuite* (2015) 49 VR 196, 218 [77] (Maxwell ACJ, Redlich and Weinberg JJA); [2015] VSCA 148.

273 See, eg, *Chen v The Queen* (2018) 97 NSWLR 915, 926 [62] (Hoeben CJ at CL, Schmidt and Campbell JJ); [2021] NSWCCA 106.

their evidence inadmissible under s 79 (although it may go to the weight to be given to it).

669 As to whether the evidence is 'apparently credible (or at least capable of belief)', I consider that the evidence of Professors Eriksson, Högberg and Wester was of such a nature. They are respected senior practitioners of their disciplines in Sweden, who have published numerous articles in peer reviewed journals on matters relevant to the opinions they expressed in their reports. There was no challenge to their bona fides. None of the matters put to Professors Eriksson, Högberg and Wester in cross-examination caused me to consider that their evidence was not 'apparently credible'. Thus their evidence satisfies the threshold requirement of 'apparent credibility'.

(c) The role of this Court in evaluating reliability and credibility of fresh evidence

670 I note for completeness that neither the admissibility analysis nor the credibility analysis require this Court to form a view on whether we prefer the evidence of Professors Eriksson, Högberg and Wester or the evidence of Dr Tully, Dr Iles and Professor Ditchfield. That is in large measure because of the nature of the inquiries this Court is required to make into admissibility (which does not require or permit any evaluation of the reliability of the expert evidence, let alone an evaluation of whether it is to be preferred to the competing expert evidence adduced) and credibility (which is focused on whether the evidence is 'apparently credible', or 'capable of belief', not on whether the appellate court accepts the evidence or prefers it over the evidence of other experts). But it is also because, under our adversarial system, as a general proposition it is the role of the jury to resolve conflicts between experts, not the role of this Court.[274] As this Court observed in *R v Anderson*, it is for the jury to decide whether opinion evidence given by an expert is credible and what weight it should be given.[275]

(d) Significant possibility of acquittal?

671 It thus remains to consider whether the evidence heard on this appeal reveals that, even if I was wrong to conclude that the jury must have had a reasonable doubt had it heard the evidence, a substantial miscarriage of justice has occurred. In my view the answer to that question is 'yes'. That is because, had the evidence of Professors Eriksson, Högberg and Wester, and the rebuttal evidence of Drs Tully and Iles and Professor Ditchfield, been adduced at trial, there is a significant possibility that a jury would have acquitted the applicant.

672 I address below various discrete issues raised by the evidence adduced on the appeal, and explain why, in my opinion, that aspect of the evidence would have led to a significant possibility of acquittal. However, it is also appropriate to observe that my conclusion in that regard is even stronger when all of the issues are considered cumulatively, rather than independently.

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274 *R v Duke* (1979) 22 SASR 46, 48 (King CJ).

275 (2000) 1 VR 1, 25 [59] (Winneke P); [2000] VSCA 16. See also *Velevski v The Queen* (2002) 76 ALJR 402, 432 [180], 433 [182] (Gummow and Callinan JJ); [2002] HCA 4.

(i) The SBU Report

673 Professor Eriksson's evidence was principally directed to explaining and defending the conclusions in the SBU Report, as well as explaining the relevance of those conclusions to the evidence that had been given by Dr Tully and Dr Iles at trial. As explained in Forrest and Emerton JJA's reasons, that report involved a systematic literature review directed to assessing the reliability of attributing the presence of the 'triad' — that is, subdural haemorrhage, retinal haemorrhages and encephalopathy — to traumatic shaking. Professor Eriksson explained that the key conclusions of that report were that:

(a) there is insufficient (or very low) scientific evidence on which to assess the diagnostic accuracy of the 'triad' in identifying traumatic shaking; and

(b) there is limited (or low) scientific evidence that the 'triad' and therefore its components can be associated with traumatic shaking.

674 That evidence was generally relevant to the reliability of Dr Tully's evidence and, to a lesser extent, Dr Iles' evidence at trial. Had the jury accepted that there is limited scientific evidence that the 'triad' is associated with traumatic shaking, there is a real possibility that they would not have accepted Dr Tully's and Dr Iles' diagnosis of abusive head trauma. That is, they might have rejected the conclusion that, because the triad of subdural haemorrhage, retinal haemorrhages and encephalopathy was present, Kaleb must have been shaken.

(ii) Circular reasoning

675 Each of Professors Eriksson, Högberg and Wester gave evidence that the scientific basis for utilising the presence of the 'triad' to conclude that a baby has been shaken is based on circular reasoning.

676 Professor Eriksson explained that the studies and evidence that underpin a conclusion that the 'triad' is evidence of traumatic shaking are based on circular reasoning. That is, they use cases where a child protection team had concluded that a child had been shaken, and where the 'triad' was present, to demonstrate that the 'triad' was associated with traumatic shaking. In his report he explained this as follows (emphasis in original):

> [P]roblems of circularity exist with classifications made [by] Child Protection Teams who have developed the criteria for classification of shaken baby cases *versus* controls, if the criteria are based on the assumption that a parent or guardian is untruthful when denying having shaken the baby; in these cases, if the triad is present, the baby has by default been violently shaken. However, over the past three to four decades, researchers have routinely adopted the classification and criteria of the Child Protection Team, when attempting to conduct observational studies to explore whether the triad actually implies that the baby has been violently shaken. Such an adopted method of classification that presupposes that an infant with the triad has been violently shaken results in circular reasoning. Or, in other words, what is investigated is already assumed to be true: if the triad is present, the baby must have been violently shaken.

677 Dr Iles accepted in her evidence on the appeal that the SBU report had in fact exposed the circular reasoning present in existing studies. In cross-examination the following exchange occurred:

> Dr Iles, you are fair in your rebuttal report to recognise the positive contribution made by the SBU report, in terms of exposing the circularity in existing studies?---Yes.
>
> What is your understanding of how studies so far have been circular?---So I think in terms of the sort of literature in this area, probably the biggest, in terms of circular reasoning, if, for example, so retinal haemorrhages are part of the diagnostic triad, and then so a child is diagnosed with having inflicted head injury because they have the triad, and then you look at the proportion of retinal haemorrhages in children that have inflicted injury, then that's quite clearly circular reasoning, and that is what the SBU report is pointing out.
>
> That was the most significant criticism of existing studies?---Yeah, and it's a legitimate criticism, I agree.
>
> FORREST JA: Is that because you are importing, in effect, the conclusion into the reasoning process, towards that conclusion?---Yes.

678 Similarly, Dr Tully accepted before us that there is a legitimate criticism about circularity in at least some of the existing literature. She said this in cross-examination:

> Circular reasoning is existent in the existing literature in relation to the abusive head trauma. There are some studies that are affected by circular reasoning. There is a degree of inevitability that circular reasoning will exist in the literature base because we can never do direct studies in relation to shaken infants.

679 She summed up her position on circular reasoning as follows (emphasis added):

> So I think, **yes, circular reasoning exists**. It needs to be acknowledged. It … does not affect the entire evidence base around abusive head trauma and I think **there has to be a pragmatic approach taken that the principles of evidence base[d] medicine clearly state that you must use the best available evidence** and I think we use the best available evidence.

680 Dr Tully also explained that there are more recent studies that are designed to minimise the effect of circular reasoning. She said that 'it has been suggested that the entire evidence base is subject to circular reasoning and that's simply not the case'. Her opinion was that there is a substantial body of literature that is not subject to circular reasoning. She did not, however, give particular references to relevant literature that she said was not subject to circular reasoning, either in her written report or in oral evidence. When asked about that, she pointed to an article in a law journal that was not a medical or scientific study, but which 'addresses circular reasoning'.

681 In my opinion the evidence of Professors Eriksson, Högberg and Wester concerning circular reasoning, together with the acknowledgment by Dr Tully and Dr Iles on the appeal that at least some portion of the scientific and medical literature in this field *is* subject to circular reasoning, could have undermined the certainty with which Dr Tully gave her evidence at trial.

682 Further, Dr Tully's acknowledgement that, notwithstanding the circularity present in some of the literature, she considers that a 'pragmatic approach' must be taken, so that one uses the 'best *available* evidence' could have been used as the foundation for a

submission to the jury that a pragmatic approach of that kind does not rise to the necessary level of certainty required for conviction on the criminal standard of proof. Again, there is a significant possibility that, had the jury accepted an argument of that kind, they would not have convicted the applicant.

(iii) Whether there is a medical controversy concerning the 'triad'

683 Professor Eriksson and Professor Wester both gave evidence that there is a scientific controversy concerning whether the 'triad' can be used as a diagnostic tool to conclude that a baby has been shaken. They were also both critical of the Consensus Statement: Professor Wester observed that it was not a scientific paper, and Professor Eriksson stated that it was not a 'methodologically sound' document. Indeed, in his written report, Professor Eriksson set out a series of flaws in the methodology used in the Consensus Statement. It is not necessary to set those out in detail here. He also observed that, in a hierarchy of forms of scientific studies, a systematic literature review is the highest and most credible form, and a consensus statement is the second least credible form.

684 Before us Dr Tully appeared to accept that a consensus statement is a relatively low ranked form of scientific evidence. She observed that the National Health and Medical Research Council table of ranking does not include consensus statements.

685 The evidence before us on the appeal concerning the existence of a controversy as to the diagnostic relevance of the 'triad', if accepted, could have been used to undermine Dr Tully's credibility and reliability, given she had denied that there was any medical controversy concerning the 'triad' and the diagnosis of abusive head trauma. Similarly, the evidence about the Consensus Statement, if accepted, could have been used to undermine Dr Tully's reliance on that statement at trial, and hence undermine the perception with which the jury were likely left: that there is an undisputed medical consensus that the 'triad' can be properly used to diagnose abusive head trauma, and that no relevant controversy or uncertainty exists.

(iv) The use of a 'differential diagnosis'

686 At trial Dr Tully gave evidence that the findings she had made in relation to Kaleb — in particular the bleeding in the subdural and subarachnoid space, widespread, multilayered retinal haemorrhages and a severe hypoxic ischaemic brain injury — were diagnostic of inflicted head trauma. She said this:

> [T]he combination of these findings, when an infant has been fully investigated for any other medical reason and there's no history of significant trauma, then we don't have another diagnosis other than inflicted head trauma.

687 Later the following exchange occurred:

> We may well have covered this, but in terms of any metabolic cause, that is, disease, infection and so forth, has that been considered and ruled out by you in coming to your opinion as to the cause of Kaleb's injuries?---Yes. I have ruled out any alternative medical cause.

And in cross-examination she agreed that she would rigorously try to exclude other causes for the individual findings.

688 In their rebuttal reports on the appeal, both Dr Iles and Dr Tully elaborated on the differential diagnosis of inflicted head trauma. Dr Tully said this:

> I fully concur with the opinions of the experts that the presence of the triad alone should not be used to conclude that an infant has been subject to forceful shaking.
>
> …
>
> Rigorous consideration of alternative causes, such as birth-related injury, accidental trauma and rare medical conditions that may mimic AHT, must be undertaken and any realistic possibility of an alternative cause must be acknowledged. A complex inferential and deductive reasoning process is undertaken that allows a diagnosis to be made that best explains the entire medical picture. Following this process, the medical diagnosis of AHT can be established with certainty.

689 Similarly, Dr Iles said as follows:

> The assessment of head injury in infants in some cases is complex in order to exclude presentations that may be due to natural disease processes. Consideration of alternative diseases requires additional evaluation well beyond the presence of some or all elements of the triad. The differential diagnosis of [subdural haematomas] in infants and children includes birth and accidental trauma; metabolic and genetic diseases; haematological and clotting disorders, oncological, and autoimmune diseases; vascular anomalies, congenital malformations; and others, all of which are readily assessed. Excluding alternative causes that may be realistically applicable in a particular infant, particularly when there are no cutaneous or bony stigmata of trauma, is a critical component of the diagnostic process, and if this has not or cannot be done, this lessens diagnostic certainty.

690 Both Dr Tully and Dr Iles expressed the view that the 'triad' is never the sole basis of a diagnosis of infant head injury.

691 Professors Eriksson and Wester observed that the proponents of using the 'triad' as a diagnostic tool to conclude that a baby has been shaken, say that they use the 'whole picture' in their diagnosis, not just the 'triad', and point to what they describe as a 'differential diagnosis', namely a process by which all other potential causes of the 'triad' are ruled out. However, Professor Eriksson gave evidence that, even utilising such a process, those proponents will still conclude that shaking must have occurred if no other explanation is found. That is, as he explained in his oral evidence, if 'the triad findings … are there and they do not find an alternative explanation, medical or otherwise, they conclude without additional support that the child must have been shaken. So it boils down, as I said before, to the presence of the triad'. Similarly, Professor Wester, when asked if the process described by Dr Tully was a 'careful process of exclusion', said 'No, it's not careful', pointing out that it is an exclusion diagnosis.

692 That is, their evidence was to the effect that, although those who use the 'triad' claim it is not the sole basis for the diagnosis of inflicted head injury, in fact it will often be the

only *positive* basis, the other aspects being exclusionary in nature. Further, a diagnosis of this kind excludes known conditions, but not unknown conditions.

693 Had this evidence been led and accepted at trial, it could have undermined Dr Tully's evidence that she used a 'rigorous and accurate medical diagnostic pathway' to exclude other known conditions that could have caused the injuries observed in Kaleb.

(v) Did Kaleb have BESS? And, if so, was this relevant to the diagnosis?

694 For the reasons already given in relation to this issue, I consider that had Professor Wester's evidence and Professor Ditchfield's evidence concerning BESS been led at trial, that would have undermined Dr Tully's evidence that she had excluded all other possible causes of Kaleb's injuries, and that his injuries must therefore have been caused by forceful shaking. It thus follows that, had that evidence been led, and had it been accepted, there is a significant possibility that the jury would have acquitted the applicant.

(vi) The role of retinal haemorrhages in diagnosing abusive head trauma

695 For the reasons already given in relation to this issue, I consider that the evidence of Professors Eriksson, Högberg and Wester, and of Dr Tully on the appeal, concerning retinal haemorrhages, if accepted, could have undermined Dr Tully's evidence at trial that the presence of retinal haemorrhages meant that Kaleb must have been subjected to significant force. It thus follows that, had that evidence been adduced at trial, and had it been accepted, there is a significant possibility that the jury would have acquitted the applicant.

(vii) Other matters

696 I note that on most, if not all, of the matters outlined above, the respondent put to each of Professors Eriksson, Högberg and Wester various journal articles and reports that either supported the use of the 'triad' as a tool for the diagnosis of abusive head trauma or rebutted the points made by those witnesses. Other articles and reports were put to Drs Tully and Iles and Professor Ditchfield. I have not found it necessary to consider the content of all of those journal articles and reports in detail in my reasons. It is plain that there is a disagreement amongst experts about the legitimacy of the 'triad' as a diagnostic tool, and about the specific issues discussed above. On my alternative conclusion, that disagreement is one that ought properly be ventilated before a jury; it is not one to be resolved by this Court.

(7) Conclusion on ground 3

697 In conclusion, the totality of the evidence heard on the appeal — that is, the evidence of Professors Eriksson, Högberg and Wester, the evidence of Dr Tully, Dr Iles and Professor Ditchfield, and the various journal articles and reports which I have discussed — leads me to conclude that there has been a substantial miscarriage of justice. That is primarily because I consider that, had the evidence concerning BESS and retinal

haemorrhages been led at trial, the jury must have had a reasonable doubt as to the applicant's guilt. Thus the appeal must be allowed and an acquittal entered.

698 In the alternative, I consider that the totality of the new evidence was such that there is a significant possibility (or even likelihood) that, had that evidence been adduced at trial, the jury would have acquitted the applicant, such that the appeal should be allowed. However, this would lead to an order for a new trial, not to an acquittal.

GROUND 2

699 Proposed ground 2 of the appeal, as amended, is as follows:

> A substantial miscarriage of justice occurred because the expert witness, Dr Joanne Tully, gave evidence that was incorrect and contrary to her obligations as an expert witness and new evidence should be admitted to demonstrate how the evidence of Dr Tully has caused a substantial miscarriage of justice in the applicant's trial.

700 The particulars to this ground, as set out in the written case, identify the evidence said to be incorrect as follows:

> (i) That there is no scientific controversy, or dispute, in the scientific community as to the diagnostic utility of the 'triad' to confirm that an infant has died as a result of non-accidental physical abuse;
>
> (ii) That there is a 'consensus' in the scientific community that the 'triad' can be used to determine whether the death of an infant is the result of non-accidental physical abuse.

701 This grounds turns on the admission of evidence not adduced at trial. That evidence can be divided into three categories:

(a) first, two PowerPoint slide presentations given by Dr Tully as part of the education and training provided by the Victorian Forensic Paediatric Medical Service ('VFPMS') to paediatric trainees and paediatricians throughout Victoria in relation to child abuse. Both were entitled 'Abusive head trauma — mechanisms, myths and mysteries'. One was presented in 2017 and one in 2019;[276]

(b) secondly, the expert evidence given by Professors Högberg, Wester and Eriksson concerning whether the 'triad' can or should be used to diagnose abusive head trauma (or inflicted head trauma); and

(c) thirdly, Dr Tully's evidence given on the appeal, by the filing of a rebuttal report and the giving of oral evidence.

702 In summary, I have concluded that ground 2 should succeed, based on Dr Tully's PowerPoint slides. That evidence was fresh evidence, thus the question is whether, had it been before the jury, there is a 'significant possibility' the jury, acting reasonably,

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276 It appears that the 2019 slides were created and presented in March 2019, before the applicant's second trial in June 2019.

would have acquitted the applicant. In my opinion, there is such a possibility. Dr Tully's evidence was the critical evidence that provided a pathway for the jury to convict. I consider that, had Dr Tully's PowerPoint slides been in evidence, the jury could have accepted that there is a real (medical) controversy concerning the diagnostic utility of the 'triad'. Dr Tully's certainty as to her diagnosis, and her credibility, could have been undermined. Therefore, whether as a matter of substance or as a matter of credibility, the prosecution may not have successfully excluded a reasonable hypothesis consistent with innocence. In that sense, I am satisfied that there has been a substantial miscarriage of justice, and ground 2 is made out.

(1) Dr Tully's evidence at trial, as relevant to ground 2

703 The evidence of Dr Tully at trial that is relevant to ground 2 is relatively discrete. It concerned whether the use of the 'triad' to diagnose abusive head trauma is the subject of controversy. In cross-examination, that evidence was as follows (emphasis added):

> It's right to say that the collection of subdural haemorrhage, retinal haemorrhage and encephalopathy, if I've got that right, is called in your world 'the triad'; is that right?---Actually, no, not entirely right, if I can explain. The triad is a term that's rightly been associated with the three features: subdural haemorrhage, retinal haemorrhage and damage or disruption to the brain, encephalopathy, yes. However, it's not a term really that we use in relation to this, it's a term that has been used more in the legal setting. And I can explain further why that is if necessary?
>
> Before you do that, let me ask you this. You were asked this question by me at the previous trial of this matter, 27 March 2019, so a few months ago, p.346, lines 6–10: … 'You've referred in this case or it's been referred to in this case the tried [sic] of injuries which, unless I'm terribly mistaken, are the subdural haemorrhage, the retinal haemorrhages and the encephalopathy; is that right?' That's the end of the question and you answered: 'That's correct, yes'? ---Yes.
>
> So I asked you that question and you gave that answer?---Yes.
>
> And you were of course being truthful and accurate and honest and all of those things?---Yes.
>
> Is that right? Okay, you'd agree that those findings collectively don't immediately and conclusively say non-accidental injury, but they raise concerns; that's right, isn't it?---And that's — yes.
>
> You would accept this, wouldn't you, that there is **controversy as to the required level of force required to produce this triad of injuries because no biofidelic model can be created?---Yes.**
>
> And biofidelic model, that's biofidelic meaning — and I suspect you'd probably agree with this definition: something constructed to mimic a biological entity?---Yes.
>
> Because you can't create that biofidelic model, you can't measure or quantify the level of forces involved, quite simply; correct?---Yes.
>
> …

I know you'll be able to tell me whether you agree or disagree with any of these propositions quite competently. The pre-existing conditions that the deceased had in this case made him more susceptible to the head injury that he received than a normal healthy infant; correct?---It made him more — possibly made him more susceptible to subdural haemorrhage.

And agree or disagree that as to the mechanism required to produce the triad there is controversy? The triad of injuries, the three things we've been talking about?---This is the problem when you use the term 'triad'. **When you have the pattern of subdural haemorrhage, retinal haemorrhage and encephalopathy seen in Kaleb, then I do not believe there is a medical controversy about that diagnosis, no**.

Agree or disagree that there is further controversy as to whether impact alone can cause these changes?---Impact alone?

M'mm?---**Yes, I don't know that we're clear that impact alone can cause this**.

And agree or disagree that there is further controversy as to whether the degree of existing pathology, natural or otherwise, can modify the extent and prognosis of these injuries?---As we've talked about, **we don't know the magnitude or degree of force that's required. I think it is possible that, in a child who has a pre-existing enlarged extra-axial space, then we don't know the effect of that on the degree of force required to cause subdural haemorrhage;** that's because the bridging veins are theoretically more stretched, because they're going through a bigger space. There's alternative opinion that says they are better buffered by all of the fluid in the space. We simply don't know. What we do know is that a small number of them can develop small subdural haemorrhages without any symptoms either spontaneously or with trivial forces. However, enlarged extra-axial spaces in your head don't affect your eyes, and we know that to cause widespread multilayered retinal haemorrhages requires significant forces.

…

If I said to you that there was unsettled science and uncertainty in relation to the mechanisms and the existence of shaken baby syndrome as a diagnosis, you wouldn't accept that?---No, I wouldn't.

704 In re-examination, Dr Tully's evidence was relevantly as follows (emphasis added):

There was a question also put as to **whether the science in relation to what's described as 'shaken baby syndrome' is unsettled**. What opinion do you offer in relation to that proposition?---**I don't think there is a medical controversy about — in relation to an infant who presents with very specific features of what has been described in court as the triad when rigorous and accurate medical diagnostic pathway is followed**. And, last year there was a very important, we believe, **consensus statement that was published by a number of colleges and Royal Colleges throughout America, and Europe and Japan, that provided support in relation to the diagnosis of abusive head trauma**. Now, we don't call it shaken baby syndrome, that was something that was — a term that was used years ago, still is used colloquially, but the term that is used is 'abusive head trauma', and that's because of the idea that this

constellation or combination of findings is thought to occur as a result of forceful shaking, but also maybe associated impact. So, we don't use the term shaken baby syndrome because it's so specific; **we use an umbrella term, abusive head trauma, and there is very well recognised medical consensus opinion that, when done properly, that is a valid diagnosis**.

705 Relatedly, Dr Tully's evidence was that 'current understanding' was that the combination of findings made in relation to Kaleb was most likely to be caused by forceful shaking, with or without associated impact. That reference to 'current understanding' suggested a definitive medical position in relation to the conclusions to be drawn from the presence of those findings. It did not admit of any controversy or difference of opinion.

(2) The PowerPoint slides

706 As noted above, there were two sets of PowerPoint slides put in evidence: the 2017 slides and the 2019 slides.

(a) The 2017 slides

707 Slide 3 of the 2017 slides contained the following statement:

> Triad of;
>
> - SDH
> - RH
> - Encephalopathy

708 Slide 20 of the 2017 slides was as follows:

709 The final slide of the 2017 slides contained the following by way of summary:

> **Summary**
>
> - AHT poses many challenges to the clinician

- Careful consideration of other possible aetiologies is required
- High quality evidence exists but the area is also plagued by "non believers!"
- Strategies to aid prevention are essential

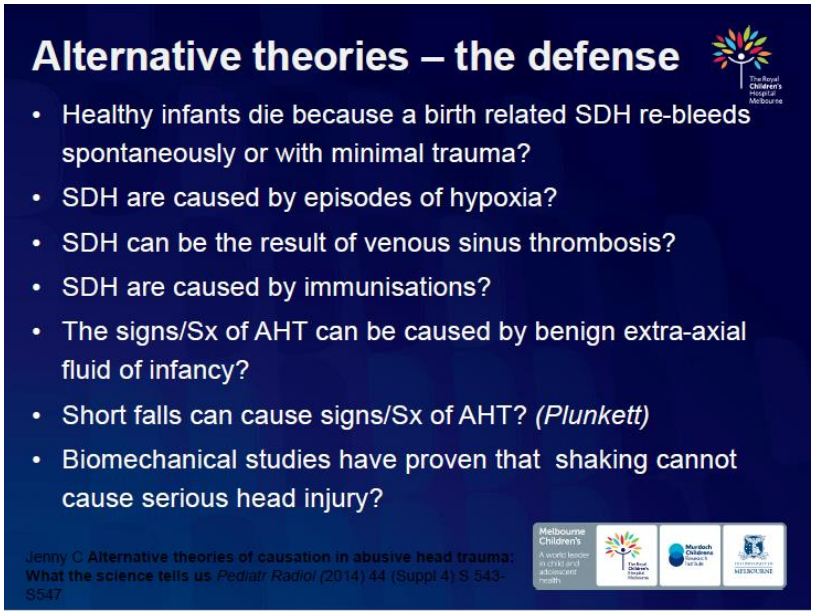
(b) The 2019 slides

710 Slide 34 of the 2019 slides was headed 'The controversies —what the defence make of AHT'. It was as follows:

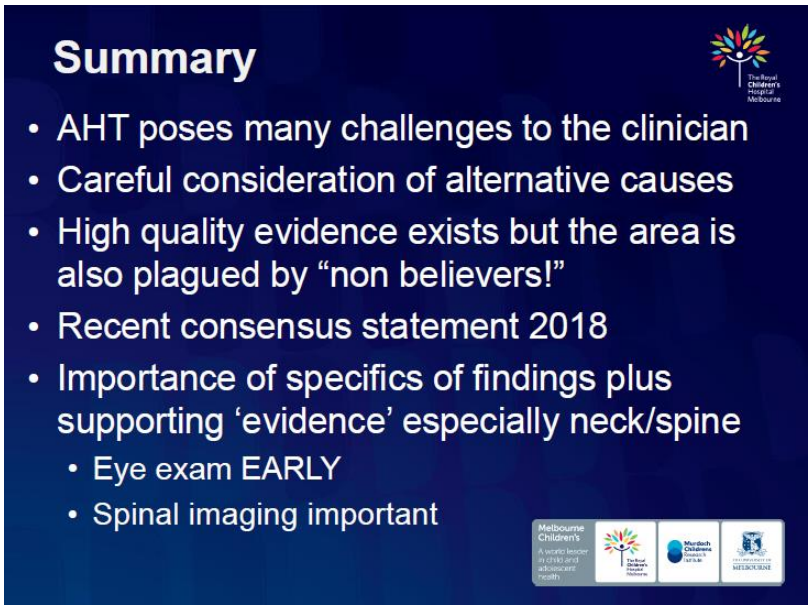
711 The next slide was a continuation, and was as follows:

712 The next slide showed a picture of Dr Waney Squier,[277] alongside a picture of the cover sheet of the UK High Court decision *Squier v General Medical Council*.[278]

713 The next slide was as follows:



714 The penultimate slide was the summary slide:



277 Dr Squier is a neuropathologist who gave evidence for the defence in various cases concerning non-accidental head injury in the United Kingdom.

278 [2016] EWHC 2739 (Admin). That case was a challenge to a decision by the UK General Medical Council to erase Dr Squier's name from the medical register. The Court found that the decision to erase her name from the register was 'in many significant respects flawed', and that the decision to sanction her would need to be 'retaken': [137] (Mitting J).

715 The final slide was as follows:

(3) Dr Tully's evidence on the appeal concerning the slides

716 Dr Tully gave oral evidence concerning the PowerPoint slides. She acknowledged that the PowerPoint slides were not listed on her CV, but said that she had included in her CV that she had 'taught at seven or eight VFPMS' seminars. She explained that the VFPMS offers a three-day seminar and said that she often delivered 10 or 11 of the lectures, as well as some of the other interactive teaching sessions, and that she did not think it was necessary to detail all the lectures she gave on her CV. In that regard, she said as follows:

> I have hundreds of presentations on my computer. I teach, very often. I have included in my CV that I teach and that that is part of my role within VFPMS. I don't think it's reasonable to include my power point presentations in a CV, they're just — they're just — there's just too many.
>
> …
>
> [I]t wouldn't cross my mind that I was under an obligation to provide teaching sessions and if I was asked for them, I would provide them.

In that regard, Dr Tully agreed that she simply provided her standard CV, not a CV tailored to the particular case.

717 Dr Tully also indicated in her evidence that there is a 'difference between what's written on a PowerPoint slide and what [she] might speak to' in the lecture.

718 As to the controversies identified in the slides, Dr Tully said this:

> [T]hey include the fact that there are perceived controversies within the legal forum and sometimes reflected in the media in relation to, um, alternative theories around the causation of the findings that are seen in infants with head trauma including inflicted head trauma.

719 Dr Tully stated that the reason that the controversies were included in her lecture was as follows:

> [P]aediatricians may need to appear in — in court as expert witnesses. And I think it would be remiss of us within the VFPMS to fail to acknowledge alternative theories that are proffered in — in the courts in order that those paediatricians can understand and therefore fulfil their obligations as an expert witness.

720 In relation to the term 'non-believers' in the 2019 slides, the following exchange occurred:

> Well you refer in the 2019 seminar at least to the areas plagued by, 'Non-believers'?---Yes, and if you look at that, the non-believers is in quotation mark. It's not what I said. It's what other people have said.
>
> …
>
> You adopt it, that you would say that?---In quotation marks.
>
> There are, 'Non-believers' here?---To illustrate, I think it's probably — it's probably fair to say and I — that when I teach I do adopt a hearts and minds approach. I try very hard to engage an audience. Yes, the seminar is long. It's three days of lectures and I think that that is my style of teaching. Now, I think it must be made clear that the, 'Non-believers', was in quotation marks. It's just something that has been said in relation to the perceived controversy that is driven in the media.
>
> Can I say this, Dr Tully? That appearing there — I want to suggest — just that you think the field is divided between believers like yourself, and non-believers who don't accept what you're saying. Do you accept that?---I can accept that that — that phrase implies that — but what it doesn't — what it doesn't do is give any kind of idea of — of the relative numbers, I suppose, if you like to say. So it is well acknowledged that there are a small number of researchers or clinicians who do not support the consensus view that shaking or shaking of impact can injure an infant's brain. Yes, there are people who don't — who don't share that view.
>
> Not sharing a view shouldn't come with a label of being a non-believer though, I suggest?---Yes, I think that's fair.

721 The key passage of Dr Tully's evidence in relation to the existence of a controversy concerning abusive head trauma was as follows:

> I don't say there's no controversy. It is my view that there is not a valid legitimate debate in relation to the medical diagnosis of abusive head trauma when the process is done as it should be done. So I — I think that's a little different. I acknowledge that there are alternative theories of it but I think what — what we're talking about here is there unsettled science, is there legitimate debate about an issue and so if you use the example of biomechanical data, then there is unsettled science because you have a body of biomechanical research that says — that suggests that forcible shaking can generate forces that do exceed the tolerance of the bridging veins and neck. You have an alternative um evidence base that suggests maybe that isn't the case. So that in my view is —

> is unsettled. In relation to the — in relation to, can forcibly shaking an infant with or without impact result in injury or result in findings that have been described as the triad, then I don't believe there's unsettled science about that mechanics [sic] because you have a very large body of evidence that supports that association through multiple disciples across the globe in thousands of cases. I am not aware of, and I'm happy to stand corrected, but I'm not aware of any studies, well-designed large studies that are able to refute that association, so the way I interpret that is that there isn't unsettled science.

722 Dr Tully rejected the proposition that she was an advocate for one side of the debate:

> … I have no vested interest in diagnosing inflicted head trauma. I am not an advocate for inflicted head trauma. I simply believe that unfortunately child abuse is common and probably commoner than, than, than a lot of people believe and that we have a need unfortunately, to do this work with a high level of integrity and with a high attention to detail, such that we can provide opinions that best support children and families that are in these difficult situations. So I refute the suggestion that I am an advocate for this condition.

723 Finally, Dr Tully was asked some questions about her obligation, under *Practice Note SC CR 3: Expert Evidence in Criminal Trials*,[279] to disclose the existence of any significant and recognised disagreement or controversy within the relevant field of a specialised knowledge (emphasis added):

> I want to suggest in this case that it was an obligation upon you to recognise and inform in your report the nature of what I want to suggest is a significant dispute between scientists and clinical practitioners about this issue. What do you say?---**I agree that if I believed there was a — a valid controversy about the medical diagnosis or the diagnosis I had made that I would need to include that.** I think I've — I've referred in my report to um — to the fact that the degree of force to require to cause certain injuries is — is not known but given that I don't think there is a — a — there's unsettled science in relation to the medical diagnosis of inflicted head injury, then I don't think I — I'm under an obligation to do so and that isn't something that is um — is — is acknowledged routinely in VFPMS medical legal reports. Where there — where I don't know, I must say and I believe that that's what I do. If I don't know the cause mechanism, timing of an injury, I'm — say that and that is what I would do.
>
> I suppose my point is those seminars you present indicate that there is a controversy. You might say the other side is providing, you know, inappropriate, insufficient but there is I want to suggest a proper controversy which is reflected in your own papers or seminar papers?---I — **the PowerPoint presentation has a title — ah slide entitled, 'Controversies', and I think we visited that before in terms of the reason that is there and I do think it is important to understand when you do this work that there are, as I've said, alternative — a small minority of individuals who offer alternative views but that these are not at current times substantiated by any evidence.** So we have a large body of evidence that consistently, reliably and repeatedly demonstrates this association including information that has been gained from people who said what they've done. We don't as yet have any well-designed large studies that refute that association. So I don't think I'm under an obligation where there isn't a study that I can refer to, to — to — to include that

[279] *Practice Note SC CR 3: Expert Evidence in Criminal Trials*, 30 January 2017 ('Expert Practice Note').

information in my medical legal report. I've made a medical diagnosis and that is what is in my report.

(4) The parties' submissions concerning the PowerPoint slides

724 The applicant advanced his case based on the PowerPoint slides in several distinct, albeit related, ways.

(a) First, he submitted that Dr Tully ought to have disclosed the PowerPoint slides prior to trial and that, had she done so, a useful line of cross-examination would have been opened for the defence.

(b) Second, he submitted that the PowerPoint slides and the evidence of Professors Högberg, Wester and Eriksson demonstrate that Dr Tully gave evidence at trial that was incorrect; namely, her evidence that there is no 'controversy' about the use of the 'triad' to diagnose abusive head trauma.

(c) Third, he submitted that the PowerPoint slides and the answers given by Dr Tully at trial concerning whether there is a controversy, viewed in light of the evidence of Professors Högberg, Wester and Eriksson, reveal that Dr Tully is an advocate for a cause, not a truly independent expert. As a consequence, he submitted, she breached her obligations as an expert witness as set out in the Expert Practice Note.

725 In contrast, the respondent submitted that:

(a) Dr Tully was not asked in terms about, and did not comment on, any 'dispute' concerning the 'triad';

(b) in any event, Dr Tully conceded that there are several 'controversies' concerning the 'triad', namely:

(i) a controversy as to the mechanism that might produce the relevant injuries and whether they could be caused by 'impact alone';

(ii) a controversy concerning the level of force required to produce the 'triad'; and

(iii) a controversy concerning whether '"the degree of existing pathology, natural or otherwise", might modify the extent and prognosis of the relevant injuries'.

726 The respondent also submitted that Dr Tully did not find it helpful to speak of the 'so-called "triad"' in some abstract sense, divorced from the particular circumstances of a given case. Thus, the respondent submitted, the existence of the 'triad' collection of injuries did not 'immediately and conclusively' mean non-accidental brain injury, but their presence 'raised concerns'. The respondent emphasised that Dr Tully's evidence was specific to 'the nature and extent of the particular injuries sustained' by Kaleb — it was in that context that she did not think that the diagnosis of inflicted head trauma was controversial.

727 In oral argument, the respondent contended that Dr Tully had not been required to disclose the PowerPoint slides, either in her CV or otherwise.

(5) Consideration of the PowerPoint slides

728 It is convenient to consider first, and separately, the aspect of ground 2 based on Dr Tully's PowerPoint slides, because it is independent of the evidence of Professors Högberg, Eriksson and Wester.

(a) Were the PowerPoint slides 'fresh' or 'new' evidence?

729 The first question to address in relation to the PowerPoint slides is whether they constitute 'new' or 'fresh' evidence, because this will impact on the manner in which this Court approaches the significance of the evidence.

730 I agree with Forrest and Emerton JJA that Dr Tully's PowerPoint slides are properly characterised as fresh, rather than new, evidence. That is because I do not consider that they could, with reasonable diligence, have been produced by the accused at his trial. The PowerPoint slides were not referred to in Dr Tully's curriculum vitae in express terms.[280] They were not published papers, nor were the presentations to which they relate given at a public event. There was no basis for the defence to know of the existence of the PowerPoint slides, in the absence of them having been disclosed by Dr Tully. Noting that 'great latitude' is to be given to an accused in this regard,[281] I do not think that the applicant could, with reasonable diligence, have produced the PowerPoint presentations. That is, I do not think that reasonable diligence required that the defence call for all documents relating to all presentations Dr Tully had given in relation to a particular topic, in the absence of any indication that such presentations existed. The mere statement that Dr Tully had given VFPMS seminars, without more, did not provide such an indication.

731 Some of Dr Tully's oral evidence on the appeal also related to the PowerPoint slides. Plainly that evidence was not in existence at the trial. That evidence was given in response to the tender of the PowerPoint slides, which, as explained above, are properly characterised as fresh evidence. In my opinion Dr Tully's evidence on the appeal takes its character from the evidence to which it is responsive. Thus I consider that, in so far as Dr Tully's evidence concerned the PowerPoint slides, it is properly characterised as fresh.

732 As already noted, if fresh evidence adduced on appeal reveals that there is a 'significant possibility' that that evidence, if believed, would have led the jury, acting reasonably, to acquit the applicant if the evidence had been before it at trial, then an appeal should be allowed. It is thus necessary to consider the effect of the PowerPoint slides, in light of the way in which the applicant sought to rely on them to impugn his conviction.

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280 Dr Tully's evidence was that in her CV she listed the fact that she had given VFPMS seminars, but did not provide titles or other details of those seminars.

281 *Ratten v The Queen* (1974) 131 CLR 510, 517 (Barwick CJ, McTiernan J agreeing at 524, Stephen and Jacobs JJ agreeing at 533); [1974] HCA 35. See also *Bowden* (2017) 54 VR 135, 145 [38] (Priest JA, Maxwell P and Kidd AJA agreeing at 137 [1]); [2017] VSCA 46.

(b) Effect of the fresh evidence concerning the PowerPoint slides

733 It may be immediately observed that in her evidence at trial Dr Tully acknowledged the existence of two specific 'controversies' or uncertainties as to the diagnosis of abusive head trauma based on the 'triad':

(a) she accepted that there is a controversy as to the amount of force required to produce the symptoms known as the 'triad'; and

(b) she accepted that it is unclear as to whether impact alone can cause the symptoms known as the 'triad'.

734 However, at trial Dr Tully did not accept that, in any more general sense, the science surrounding the 'triad' is 'unsettled' or that there is any 'medical controversy' about the diagnosis of abusive head trauma when the relevant diagnostic pathway is followed. Rather, her evidence was that there is a medical consensus that such a diagnosis is a valid diagnosis when properly done; and she relied upon the Consensus Statement in support of that proposition. She did not refer to the SBU Report, although she acknowledged in her oral evidence on the appeal that she was aware of that report at the time she gave evidence at trial. Further, the SBU Report was referred to in the 2019 slides.

735 It is plain from Dr Tully's evidence both at trial and on the appeal that, while she accepts some degree of controversy concerning the amount of force required to produce the 'triad', she does not consider that there is any *legitimate* controversy about the diagnosis of abusive head trauma when the 'triad' — including the specific pattern of extensive retinal haemorrhages — is present. She considers that there is a 'perceived' controversy, which she describes as being 'within the legal forum' and 'driven in the media'. There is no reason to doubt that that is her genuinely held opinion as to the state of the medical evidence, based on her reading and experience.

736 The question that arises, however, is whether, when asked about whether there is 'unsettled science and uncertainty' in relation to the 'mechanisms and existence of shaken baby syndrome as a diagnosis', it was appropriate for her to answer 'no'; and whether it was appropriate for her to state that there is no 'medical controversy' about such a diagnosis. Her evidence in that regard now falls to be assessed in light of the PowerPoint slides, in which Dr Tully had used the term 'controversies' to identify 'alternative theories' about the cause of the 'triad'. More specifically, had the PowerPoint slides been available to the defence at trial, could they have made any difference to the cross-examination of Dr Tully and the jury's reliance on her evidence?

737 Of course, in making that assessment, this Court cannot know precisely what questions defence counsel might have asked Dr Tully had he had access to the slides. But it is not difficult to postulate that, Dr Tully having stated in her oral evidence at trial that she did not believe there is a 'medical controversy' about the diagnosis of abusive head trauma, and her disagreement with the proposition that the science is unsettled, defence counsel would have been able to put to Dr Tully that she herself had acknowledged the existence of controversies in relation to abusive head trauma and the 'triad'. That could have potentially had two impacts.

(a) First, it could have undermined Dr Tully's credibility, in that:

(i) her definitive answer was contradicted by her own PowerPoint slides, which she had presented only months prior to the trial; and

(ii) it may have suggested that she had adopted a partisan approach to the diagnosis of abusive head trauma, and her role as an expert, as evidenced by her reference to the area being 'plagued' by 'non-believers'.

(b) Secondly, it could have been used by the jury in a more substantive sense, to reason that, contrary to Dr Tully's evidence, the science *is* 'unsettled', and that there *is* a 'medical controversy' concerning the utility of the 'triad' as a diagnostic tool to demonstrate abusive head trauma.

738 In addition, the PowerPoint slides could have been used by the defence to reinforce the proposition raised at the trial that both an early eye exam and spinal imaging were important, neither of which occurred in relation to Kaleb.

(a) The absence of an 'early' eye exam was arguably important in two different ways, namely because no eye exam was done during Kaleb's admission between 14 January 2016 and 17 January 2016, and because the eye exam that was eventually done was not done 'early' — that is, it was done outside the recommended time frame for such examination. Dr Tully was asked about this during her evidence at trial. Had the 2019 slides been available, they could have been deployed to interrogate her answers to those questions.

(b) The absence of evidence of spinal injury was potentially important because when Dr Tully described the 'triad' to the jury, she described the third element as being injury to 'the spinal cord … and … brainstem'. Yet no such injury was found in Kaleb, either upon admission to hospital on either occasion (because no spinal imaging was taken) or on post-mortem (on Dr Iles' evidence). Thus, based on the 2019 slides, an 'important' step was not undertaken, and arguably there was no evidence of an important aspect of the 'triad'.

739 Of course Dr Tully would have had an opportunity to give evidence about the content of the slides, including what she meant by 'controversies' and why she did not consider the controversies she had identified in the slides to be 'legitimate' controversies. Her slides could have been tendered in their entirety, and that could have posed some risks for the defence. It might be that her evidence concerning the slides would have satisfied the jury both of her credibility and of the reliability of her diagnosis. But that is speculation.

740 To some extent, this Court can infer what Dr Tully might have said in answer to such questions by reference to the answers she gave to questions concerning the slides on the appeal. But, assuming that she would have given the same kind of evidence at trial as she gave on appeal concerning the slides, I do not think that that evidence would have foreclosed the jury from reasoning that there was (and is) a medical controversy about the use of the 'triad' to diagnose abusive head trauma. In particular, Dr Tully acknowledged in evidence before us that there is a 'small number' of clinicians and researchers who do not support the consensus view that she supports. Thus, to that extent, she acknowledged a difference of opinion, but sought to defuse that difference

by pointing to it as a minority view. She did not suggest that the persons expressing that different view lack relevant qualifications and expertise. Dr Tully also said in evidence before us that she did not 'say that there's no controversy'. Literally, that is true, because what she said at trial was that there is no 'medical controversy'. But that is a somewhat disingenuous distinction.

741 Ultimately, there is a significant possibility that, had Dr Tully's slides been in evidence, the jury would have accepted that there is a real (medical) controversy concerning the diagnostic utility of the 'triad'. Dr Tully's certainty as to her diagnosis could have been undermined. Her credibility could have been undermined. This possibility is reinforced by Dr Iles' evidence, set out in the judgment of Forrest and Emerton JJA, which was considerably more equivocal than Dr Tully in relation to the possibility that Kaleb's pre-existing condition had pre-disposed him to injury from less force than would ordinarily be required to produce the 'triad'. Dr Tully's evidence was the critical evidence that provided a pathway for the jury to convict. Without her evidence, a conviction would not have been possible. If the jury accepted that there is a medical controversy concerning the diagnosis of abusive head trauma where the 'triad' is present, they might not have concluded that the applicant had handled Kaleb in a manner that reached the relevant standard of gross negligence or unlawful and dangerous act. Thus, had the defence had available to it material that potentially undermined Dr Tully's evidence, whether as a matter of substance or as a matter of credibility, the prosecution may not have successfully excluded a reasonable hypothesis consistent with innocence.

742 Thus, in my opinion, had the PowerPoint slides been available to the defence at trial, there is a significant possibility that the jury would have acquitted the applicant, noting that, where fresh evidence is concerned, it is not for this Court to resolve what we would have made of the evidence, but to consider what a reasonable jury might have made of it. I consider that Dr Tully's evidence as to the absence of any medical controversy was, objectively understood by reference to her own PowerPoint slides, incorrect. Had the PowerPoint slides been in evidence before the jury, I consider there is a reasonable possibility that they might not have accepted Dr Tully's unequivocal assertion that there was no medical controversy concerning the 'triad'. Had they taken that path of reasoning, there is a reasonable possibility that they would not have convicted the applicant. Thus, I am satisfied that there has been a substantial miscarriage of justice, and ground 2 is made out. It is thus unnecessary to consider the effect of the evidence of Professors Högberg, Eriksson and Wester in relation to ground 2.

743 The consequence of my conclusion on ground 2 is that the conviction should be set aside and a new trial ordered.

744 It remains to make some remarks about Dr Tully's failure to disclose her PowerPoint slides. To the extent that the applicant suggested in the course of the appeal that, by that failure, Dr Tully behaved in a manner contrary to her obligations as an expert witness, I wish to emphasise that I have not made any such finding. Dr Tully provided a CV that was her 'ordinary' CV, not one that was tailored to the particular case in which she was appearing. That was not contrary to her obligations. I accept that a person in Dr Tully's position would not include a list of PowerPoint presentations given at seminars of the kind she delivered on behalf of the VFPMS in their 'ordinary' CV. There was, in my opinion, no reason for Dr Tully to have, of her own accord, produced any different CV or produced the particular PowerPoint slides in issue on this appeal.

745 However, it is apparent from my reasons that I consider that the PowerPoint slides were potentially significant to the manner in which the applicant could have conducted his case at trial, had the slides been available to him. That raises the question of whether those slides should have been disclosed to the defence prior to trial. In my opinion, they ought to have been so disclosed. However, the responsibility for ensuring such disclosure rested with the respondent, not with Dr Tully. In my view there is an obligation on the prosecution to ensure that an expert witness is briefed in such a way as to make clear that that expert should disclose — either in their report, in their CV or, at least, to the prosecution — *particular* materials that are relevant to the *particular* case in issue, even if those materials would not ordinarily be included by that person in their CV. That is, the prosecution is in a position to understand the potential significance of materials of this kind; and an obligation falls on the prosecution to ensure that an expert witness called by the Crown provides complete and full disclosure of materials produced by that witness that relate to the issues before the court.

CONCLUSION

746 For the preceding reasons I would grant the extension of time, grant leave to appeal and allow the appeal on grounds 2 and 3. By reason of allowing the appeal on ground 3, I would set aside the applicant's conviction and order that an acquittal be entered.

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