CORONERS ACT, 2003

SOUTH *AUSTRALIA*

FINDING OF INQUEST

An Inquest taken on behalf of our Sovereign Lady the Queen at Adelaide in the State of South Australia, on the 10th, 16th and 19th days of February 2021 and the 30th day of June 2023, by the Coroner's Court of the said State, constituted of David Richard Latimer Whittle, State Coroner, into the death of Levi Shane Vanin.

The said Court finds that Levi Shane Vanin aged 29 days, late of 27 Laver Avenue, Gulfview Heights, South Australia died at the Women's and Children's Hospital, 72 King William Road, North Adelaide, South Australia on the 16th day of November 2016 as a result of severe neonatal encephalopathy post palliative care. The said Court finds that the circumstances of his death were as follows:

1. Introduction and cause of death

1.1. Levi Shane Vanin died at the Women's and Children's Hospital on 16 November 2016 at 29 days old. An initial pathology review carried out by Forensic Science South Australia provided the cause of death as '*cerebral hypoxia due to birth asphyxia*'.[1] Following a coronial direction, a post mortem examination was carried out by senior paediatric pathologist Associate Professor Lynette Moore of the South Australian Paediatric Autopsy Service on 24 November 2016.

1.2. The opinion as to cause of death then expressed by Professor Moore is '*severe neonatal encephalopathy, post palliative care*'.[2]

1.3. Neonatal encephalopathy is a complex disease of a newborn, characterised by an altered level of consciousness, seizures, poor tone, an inability to maintain respiration and may

[1] Exhibit C2a
[2] Exhibit C4a, Post Mortem Report

be associated with multi-organ dysfunction. Neonatal encephalopathy can result from a range of causes.

1.4. The post mortem report stated Levi was born in a poor condition, having been delivered post-term following a prolonged induction period with an unfavourable cervix, prolonged rupture of the membranes, chorioamnionitis, a rise in foetal heart rate and prolonged second stage of labour.

1.5. Professor Moore further concluded the pattern of damage to the brain fits with an acute hypoxic event, with no evidence of an underlying pre-existing structural brain abnormality. She stated that from the post mortem examination it was not possible to determine at what point during the induction, labour or the delivery process the brain injury occurred, however the imaging was considered to argue against any pre-existing brain damage occurring earlier in the pregnancy.

1.6. The post mortem report stated acute chorioamnionitis and foetal inflammatory response and foetal vessel thrombosis may have contributed to the brain injury. An examination of the umbilical cord and placenta revealed acute funisitis with neutrophils infiltrating the walls of the umbilical vein and one of the umbilical arteries. Fibrin thrombus was also observed.[3] The pathologist stated thrombosis of foetal vessels in the placenta may occur secondary to endothelial damage, secondary to chorioamnionitis and may be associated with thrombosis emboli with foetal organs including the brain.

1.7. Examination of the brain revealed evidence of gliosis and microglial activation with increased numbers of CD68 positive cells identified within the deep cerebral white matter and basal ganglia when compared to age-matched control sections.

1.8. Professor Moore recommended a review of the labour and delivery notes and CTG[4] by a consultant obstetrician to see if appropriate guidelines for management of labour were followed, and if examination of the CTG trace could give an indication of the timing of the brain damage.

1.9. This Court embarked on an investigation to establish, if possible, when the brain injury occurred. An agreed opinion by two experts in the field of obstetrics and gynaecology,

[3] Fibrin is a protein involved in the clotting of blood
[4] Cardiotocography, also known as electronic fetal monitoring (EFM)

Emeritus Professor Roger Pepperell and Emeritus Professor Alistair MacLennan, has assisted in identifying an underlying likely cause, although its timing remains uncertain.

1.10. Professor MacLennan and Professor Pepperell are highly qualified and experienced academic and clinical gynaecologists and obstetricians. The expertise of each to proffer opinions on the matters in question is undisputed.

1.11. A number of expert medical opinions upon a range of matters relevant to Levi's death, and the history of the obtaining of them, indicate the complexity of the determination of the cause and circumstances of Levi's death.

1.12. In the course of the coronial investigation of the cause and circumstances of Levi's death, Professor Pepperell was engaged by the Court to review the hospital case notes and the post mortem report, and he provided a report to this Court dated 1 August 2017 (the Pepperell Report). [5]

1.13. In the Pepperell Report, Professor Pepperell expressed the opinion that Levi's death could have been prevented. He stated:

> 'If delivery had occurred 2 – 3 hours earlier than it was, it is highly likely that Levi would have been in much better condition at delivery and survived. I believe the major neurologic problems in Levi were not due to severe hypoxia in labour but due to the inadequate resuscitation following delivery.' [6]

1.14. It is important at this stage to note, as I shall address later, that Professor Pepperell also recommended assessment by a consultant radiologist of Levi's post delivery ultrasound and MRI imaging to consider whether the findings were consistent with hypoxia occurring after delivery, and he also recommended review by a neonatal paediatrician as to the adequacy of care following transfer to the Women's and Children's Hospital. Reports were obtained from Dr Halliday, Dr Linke and Professor Liley, as a consequence of which Professor Pepperell revised his opinion, concluding that the intubation (inadequate resuscitation) was not a mechanism or cause for Levi's hypoxic event.

1.15. Unbeknown to the Coroners Court, within months of Levi's death and before the Coroners Court requested an expert opinion from Professor Pepperell, Professor MacLennan also reviewed the case notes and the post mortem report and provided a

[5] Exhibit C18b
[6] Exhibit C18b, Page 11

report to the Crown Solicitor dated 10 May 2017 (the MacLennan Report).[7] The Crown Solicitor had requested this report on behalf of the South Australian Government Financing Authority (SAFA) which, among other functions, provides insurance and claims management for the State government[8]. It is an authority of the South Australian Government.

1.16. The Office of the State Coroner and the Coroners Court remained unaware of the existence of the MacLennan Report or its contents until it was disclosed to the Court on 3 February 2021, shortly before an inquest into the death of Levi Vanin was due to commence.

1.17. The MacLennan report proffered an opinion as to the cause of Levi's death:

> 'The cause of this neonatal death was on the balance of probabilities due to fetal inflammatory response syndrome … Although there was evidence of secondary neonatal hypoxia there was never evidence of fetal hypoxia requiring earlier delivery. The funisitis suggests that fetal infection was present for more than 24 hours and prior to established labour. The pregnancy, labour and delivery management were all within a normal spectrum of obstetric protocols and common management. Currently there are no protocols that are proven to reduce the fatal neurological risks of fetal inflammatory response syndrome. Thus, the sad death of Levi was not preventable.'

1.18. Professor MacLennan also recommended, '*that this independent report is read by the coroner, the parents, the reporting pathologists, the neonatologist involved and the Lyell McEwin obstetricians involved*'.[9] Clearly, Professor MacLennan understood very well that an event as tragic as the death of a newborn baby causes extreme trauma to the parents, who understandably want to know why the baby died and whether anyone or anything contributed to the death. (In this case, I am aware that Levi's parents were concerned that conduct of clinicians at the Lyell McEwin Hospital had contributed to Levi's death). Professor MacLennan also understood that the death of a baby is traumatic for clinicians involved in the mother's care and the baby's delivery, and that they would anxiously await assessments of their conduct and any possible contribution. Professor MacLennan also understood that his clinical opinion, based on his extensive and undoubted expertise, would be of great assistance to the State Coroner in the investigation of the cause and circumstances of Levi's death.

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[7] Exhibit C27
[8] About SAFA | SA Government Financing Authority
[9] Exhibit C27, page 25

1.19. The disclosure of the report, which was urged by Professor MacLennan in May 2017, did not occur until the eve of an inquest into the death of Levi, which was due to commence almost three years after Professor MacLennan prepared his report.

1.20. Once I became aware of the MacLennan Report, on 10 February 2021 a directions hearing was convened and I ordered that the experts, Professors Pepperell and MacLennan, confer and provide a joint report in the spirit of the Uniform Civil Rules 2020 rule 74.9.

1.21. I asked at the directions hearing why the report had not been disclosed earlier. Ms Gavranich, instructed by the Crown Solicitor, told me that the report was obtained not for the purposes of a coronial inquest but for another purpose, such that the report was privileged. I was then told by counsel that she could provide no further information as any reasons for claiming or exercising the privilege are also subject to privilege. A claim of legal professional privilege is an absolute protection from any obligation to disclose and a court cannot look behind the claim of legal professional privilege.

1.22. Levi's parents and the clinicians at the Lyell McEwin Hospital remain unaware of the reason or reasons the report was not released earlier, with privilege waived, which would have informed them at an early stage of Professor MacLennan's opinion that Levi's death was not preventable and was not due to any inappropriate obstetric or paediatric care. In an email to the Coroners Court from Levi's parents dated 17 February 2021,[10] various concerns were expressed including, '*the fact that the Crown had this report in 2017 and did not disclose it until one week before the inquest when it is completely in their favour also screams alarm bells to us*'. In the absence of an explanation, such a sentiment from Levi's parents is unsurprising.

1.23. As I have observed, Professors MacLennan and Pepperell are two eminent academic and clinical experts in obstetrics and gynaecology. They conferred, considering all of the then available material and each other's opinions. As an independent expert is entitled to do – and is required to do if so persuaded – Professor Pepperell revised his initial opinion that an earlier delivery could have led to Levi's survival. As I have mentioned, on the basis of other reports, he had previously revised his opinion that

[10] Administratively tendered as exhibit C17a

Levi's major neurologic problems were not due to severe hypoxia in labour, but due to the inadequate resuscitation following delivery.

1.24. The joint report of Professors Pepperell and MacLennan was provided on Monday, 15 February 2021 and is now referred to as 'the Joint Report'. It states:

> 'The cause of death was very likely a severe neurological fetal inflammatory response during the neonatal period extending from an intrauterine/placental/fetal infection which would probably have been present for at least several days before birth but not detectable at that time. There was secondary neonatal hypoxia, but this was not the primary cause of death. The source of the infection cannot be ascertained but a long maternal history of antenatal and postnatal urinary tract infections and hydronephrosis is noted.
>
> This death was not preventable and was not due to any inappropriate obstetric or paediatric care.' [11]

1.25. Once the Coroners Court received the Joint Report, it became clear that, in the light of agreement between Professors MacLennan and Pepperell, there was no longer any arguable controversy as to whether Levi's death was caused or contributed to by inappropriate obstetric or paediatric care.

1.26. Prior to explaining how Professors Pepperell and MacLennan arrived at their joint opinion, and referring to subsequent slight revision of the opinion, it is pertinent to set out some of the relevant background, including important features of the care provided at the Lyell McEwin Hospital and also at the Women's and Children's Hospital, to which Levi was transferred on 18 October 2016 and where Levi remained until his death on 16 November 2016. As I intend to accept, as proposed in the Joint Report, that the severe brain damage to Levi had occurred prior to delivery, his care at the Women's and Children's Hospital is not contentious.

2. Levi's mother's pregnancy

2.1. Levi's mother, Ms Tosha Noakes, was 27 years old at the time she gave birth to Levi. She is referred in the medical records as Ms Noakes and I shall refer to her as such. Levi's father is Mr Daniel Vanin, and Levi was their first child.

2.2. All of Ms Noakes' relevant medical records were tendered at the inquest. On 17 February 2016, an ultrasound examination found the gestational age to be five weeks and four days, plus or minus five days, with an expected date of confinement to be

[11] Exhibit C20

15 October 2016. On 18 March 2016, the first trimester screening revealed no abnormalities.

2.3. On 4 April 2016, the next screening, an ultrasound radiology report placed the gestational age at 12 weeks and six days, plus or minus five days and the expected date of delivery as 10 October 2016.

2.4. On 19 May 2016 upon ultrasound examination, the expected date of delivery was again given as 10 October 2016, with all measurements within normal limits and foetal morphology assessed with no abnormalities detected and the amniotic fluid volume normal. Maternal cervical length and nutrient artery were all normal.

2.5. On 15 June 2016 at 23 weeks gestation, Ms Noakes was treated for a urinary tract infection (UTI). This UTI is noted in the medical records as being recurrent. Recurrent UTI and thrombocytopenia[12] are noted as pregnancy issues in Ms Noakes' pregnancy record.

2.6. On 16 June 2016, an ultrasound examination report revealed foetus maturity at 23 weeks and one day. The report also stated there were signs consistent with left maternal hydronephrosis.[13]

2.7. On 23 August 2016 Ms Noakes woke up with abdominal cramps, back pain, nausea, and vomiting. Ms Noakes was admitted to the Lyell McEwin Hospital with a UTI and remained in the hospital until 26 August 2016. Ms Noakes was appropriately treated with antibiotics.

2.8. At the last antenatal appointment on 13 October 2016 examination revealed no abnormalities. It was noted that the cervix was very posterior and unable to be reached. Ms Noakes was advised to attend the Lyell McEwin Hospital on 16 October 2016 at 5pm for induction of labour.

2.9. In summary, both mother and baby appeared healthy with no apparent abnormalities detected in the nine months prior to admission.

[12] Deficiency in platelets
[13] Swelling of the kidney

3. The Lyell McEwin Hospital admission for Levi's birth

3.1. As spontaneous labour had not commenced by then, Ms Noakes presented with Mr Vanin at the Lyell McEwin Hospital for induction of labour on Sunday 16 October 2016.

3.2. On 17 October 2016 at 3:35am Cervidil was inserted to commence induction of labour. At the time of the insertion, dilation was noted as zero, just as it was at the time of Ms Noakes' admission 10½ hours prior. Expert opinions of Professors Pepperell and MacLennan do not suggest that this delay should be considered as a clinical issue or a factor contributing to Levi's death.

3.3. At 8:10pm, still on 17 October 2016, it is noted that Ms Noakes did not tolerate an examination. She was teary and wanted a caesarean section. It appears from the records that options were discussed with Ms Noakes.

3.4. Professors Pepperell and MacLennan agree that during labour and prior to delivery, there were no signs of foetal distress and no basis to conduct a caesarean section.

3.5. The rupture of membranes occurred at 8:40pm on 17 October 2016.

3.6. During her admission Ms Noakes received epidural anaesthetic for pain relief and anti-nausea medication. The evidence is that she was appropriately managed for pain relief and nausea.

3.7. The following staff were on duty on the date of delivery, 18 October 2016:

- Dr Casie Staehr, who had had some dealings with Ms Noakes' induction, was the emergency obstetrics and gynaecology registrar. Dr Staehr's shift commenced at 8am and finished at 8pm.

- Dr Elizabeth Beare was the second obstetric registrar available on that shift and was the labour ward registrar.

3.8. At 8pm Dr Staehr and Dr Beare handed over to Dr Lodewyk du Plessis, who was on duty as the night shift doctor.

3.9. Overseeing the medical staff was the on-call consultant, Dr Anna Limgenco, whose shift commenced at 8am and ran for 24 hours.

3.10. Midwife Karen Richards' sole patient was Ms Noakes and midwife Wendy McNally was her supervisor. Ms Richards commenced her shift at 1pm and was expected to finish at 9:30pm.

3.11. At 11:50am Ms Noakes was noted to be 7cm dilated, cephalic with station at head -2cm. At 1:15pm maternal temperature was 37.7°C. Dr Beare described this as a mild temperature and prescribed paracetamol. At 4pm, the cervix was 9.5cm dilated with full effacement. There was some confusion leading to the inquest about the vaginal examination conducted at 4pm, due to Professor Pepperell's comments in the Pepperell Report[14] in which he stated the cervix was fully dilated. A fully dilated cervix marks the point at which stage 2 of labour commences, which was a matter relevant to Professor Pepperell's initial opinion.

3.12. Professor Pepperell in the Joint Report accepts that the contemporaneous record makes clear that the small lip of the cervix was apparently still present at the time, meaning that the cervix was not fully dilated. Ms Richards, the experienced midwife who conducted the 4pm examination, recorded 9.5 cm dilation '*and lip*'.[15] It is therefore clear that at 4pm Ms Noakes was not fully dilated and had not commenced stage 2 of her labour.

3.13. Ms Richards planned to conduct another examination at 6pm, to allow an hour to reach full dilation and a further hour to allow for the baby to descend further down the birthing canal, before commencing active pushing.

3.14. At 4:50pm Ms Richards noted that Ms Noakes had developed a temperature of 38.5°C. Ms Richards sought advice from Dr Limgenco, whose advice was passed on to Dr Beare, who subsequently prescribed antibiotics. An entry on the drug chart reveals benzylpenicillin was prescribed by Dr Beare at 5pm.[16] Ms Noakes' temperature remained high from this point until Levi's delivery.

3.15. In the MacLennan Report, Professor MacLennan mentions maternal pyrexia and treatment with intravenous benzylpenicillin and gentamicin at 5pm and 8:15pm,[17] although case notes rather suggest the administration of the gentamicin was earlier at

[14] Exhibit C18b, page 6, paragraph 3
[15] Exhibit C8, pages 138-139
[16] Exhibit C8, page 98
[17] Exhibit C27, pages 4-5

7:45pm.[18] Nothing turns on that, however; Professor MacLennan returns to the topic of maternal pyrexia[19] and antibiotics, setting out the differing approaches for antibiotic treatments in different maternal situations before concluding that the treatment was appropriate.[20]

3.16. In the Pepperell Report, Professor Pepperell also describes the antibiotic treatment as appropriate.[21]

3.17. Ms Richards recorded, as the result of the 6pm vaginal examination, that the cervix was fully dilated at 10cm and the baby's head was positioned at +1, which suggests the baby had descended during the previous two hours as hoped and expected. The plan was for the mother to commence pushing. It is clear that by 6pm Ms Noakes was fully dilated, and stage 2 had commenced. Neither of the experts levelled any criticism at the timing of the 6pm examination.

3.18. In an email to the Coroners Court five days after Levi died, Mr Daniel Vanin[22] expressed concerns that things did not go smoothly at the Lyell McEwin Hospital and that mistakes may have caused Levi's death. Mr Vanin stated in that email that he was told by a midwife at approximately 5:15pm that Ms Noakes was fully dilated.

3.19. Delivery of Levi occurred at 9:21pm, three hours and 21 minutes after the recorded commencement of stage 2. Stage 2 may have been longer, but its exact duration is not known. Ms Richards in her affidavit makes no mention of Mr Vanin being informed at 5:15pm, although she stated it was her expectation that after an hour from 4pm full dilation should have been reached. If, for argument's sake, 5:15pm is accepted as the time of full dilation, then stage 2 would have extended to four hours and six minutes. In the end though, the discrepancy does not require resolving, as there is not evidence that an extended stage 2 contributed to the cause of death.

3.20. The next examination, at 7:20pm, was undertaken by Dr Beare, who regarded Ms Noakes as having been in stage 2 for 80 minutes. Dr Beare stated at that time there was no clear indication to perform an instrumental delivery.

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[18] Exhibit C8, page 98
[19] Fever
[20] Exhibit C27, pages 23-24
[21] Exhibit C18b, page 6
[22] Exhibit C17

3.21. Ms Richards stated she recalled feeling frustrated after that examination, something she later told her supervisor Ms McNally.[23] Ms Richards felt that more proactive steps were needed or warranted to progress the labour. There had been prolonged pushing and Ms Noakes was tired and progressing very slowly.

3.22. Ms Richards nevertheless continued to monitor the mother and the foetal heart rate. There was nothing to suggest to her that she needed to contact the consultant, Dr Limgenco or otherwise call for help or call a code blue in relation to the progress.

3.23. There was, however, between 7:55pm and 8pm, a code blue medical emergency called in relation to another patient[24] which Dr Staehr, Dr Beare and Dr du Plessis attended, as they were obliged to do. It became apparent to Dr Beare that her assistance was not required.

3.24. Unfortunately, at 8pm it was also the time for handover, which was conducted by Dr Beare. Ms McNally raised the concerns Ms Richards had shared with her, particularly regarding whether Ms Noakes' labour should be permitted to continue. Dr Beare stated that she considered the concerns raised by the midwives and saw no clinical justification for an expedited delivery.

3.25. Dr du Plessis missed the handover due to the code blue. He received his handover from Dr Beare and Dr Staehr at about 8:20pm, in preparation for his night shift. It was not said to Dr du Plessis that Ms Noakes required any urgent review.

3.26. Whilst the code blue and handover were taking place, Ms Richards continued to monitor which remained reassuring. At 8:15pm Ms Richards instructed Ms Noakes to stop pushing due to fatigue. Despite Ms Richards' concerns, the clinical picture did not warrant calling a code blue.

3.27. At about 8:40pm Dr Beare and Dr du Plessis examined Ms Noakes and commenced preparation for an instrumental delivery. Dr Beare contacted Dr Limgenco, the on-call consultant, and said she would remain beyond her shift to assist with supervising Dr du Plessis in the delivery. Dr du Plessis was in his first year of specialist training and

[23] Exhibit C29, paragraphs 67-75
[24] Exhibit C12

required direct supervision of forceps birth by an accredited senior registrar such as Dr Beare, or a consultant.

3.28. Dr Limgenco does not recall the conversation with Dr Beare. The anaesthetist and paediatric team attended for the instrumental delivery. Dr du Plessis recalls that there was no strong objection by Ms Noakes to an assisted vaginal delivery and she had no desire for a caesarean section at that point. He discussed with her the risks and the potential for a caesarean, should assisted vaginal delivery be unsuccessful.

3.29. At 8:50pm Dr du Plessis, with the supervision of Dr Beare, commenced assisted vaginal delivery. At 8:52pm Dr du Plessis failed to lock the forceps, due to the position of the head. Dr Beare took over and the forceps were locked.

3.30. At 9:02pm Dr du Plessis attempted the first pull, which was unsuccessful, following which Dr Beare took over and achieved significant descent before handing back the forceps to Dr du Plessis. After a third pull at 9:14pm an episiotomy was performed.

3.31. With the fourth pull, Levi was delivered at 9:21pm. Dr du Plessis explains that the delivery was complicated by the fact that contractions had spaced out significantly.

3.32. One of the issues which was to be considered at inquest was whether an earlier delivery would have changed the outcome, although the answer was clear once the Joint Report was received. The joint opinion of Professors Pepperell and MacLennan is that the second stage of labour was longer than normal, but foetal heart rate was mostly normal and satisfactory, although at 8:50pm, when forceps delivery was being attempted, there were abnormal deep variable decelerations and virtually absent baseline variability after 9:10pm.

3.33. The Joint Report notes that delivery was delayed because the medical staff were attending to a medical emergency elsewhere in the hospital and:

> 'It is not known whether on-call consultant staff could have been called to help deliver Ms Noakes a little earlier. It is most unlikely that such earlier (sic) would have changed the outcome given the later finding of causation.' [25]

3.34. To answer the question of the joint experts, the on-call consultant Dr Limgenco performed a delivery of twins at 3:51am on 19 October 2016, and I infer that

[25] Exhibit C18a, page 1

Dr Limgenco was available. It is apparent she was contacted by Dr Beare prior to Levi's delivery. A likely opportunity for an earlier delivery prior to the code blue would have been after Dr Beare's examination at 7:20pm.

3.35. Dr Limgenco has reviewed the case notes and addressed the issue:

> 'If I was provided with that clinical picture at that time, or following Dr Beare's review at 19.20, I would have recommended that Dr Beare proceed to deliver the baby as a priority, and ideally within the hour.' [26]

3.36. Dr Limgenco goes on to explain that she would have taken Ms Noakes to the theatre and used Kielland forceps to turn the baby with a view to making the labour progress faster and the baby to descend faster to effect immediate delivery. If this did not work, Dr Limgenco would then proceed to caesarean delivery.

3.37. However, Dr Limgenco did not say that delivering the baby earlier as she has suggested would have made any difference at all to Levi's clinical condition upon delivery.

3.38. There is no dispute that Ms Noakes' labour and stage 2 were prolonged. The delivery itself was protracted. The joint experts' report opines that the delay was unlikely to have changed the outcome.

4. <u>Resuscitation</u>

4.1. Dr Hauser, a paediatric registrar, was present when Levi was delivered. Levi was described as being '*flat*' with no respiratory effort, his limbs were floppy, he was pale, almost colourless. Levi had bruises to his face and swelling to his head.

4.2. Within ten seconds Levi was commenced on intermittent positive pressure ventilation using a face mask. His oxygen was commenced at 21% and steadily increased to 100%. At one minute, Levi's APGAR score was 2 and his oxygen-saturation was 72%, with a heart rate of 168 beats per minute.

4.3. At three minutes the other paediatric doctors on evening shift were contacted and asked to attend to assist with the resuscitation. At 15 minutes Levi was transferred to the Special Care Nursery and intravenous access was established. Venous blood gases

[26] Exhibit C30, paragraph 55

taken at the time showed PH of 6.8, PCO2 of 79.4, base excess of minus 17.4, and lactate of 12.6.

4.4. The on-call paediatric consultant, Dr Thomas, was consulted and suggested intubation and passive cooling. At 10:05pm Levi was orally intubated using an endotracheal tube. The position of the tube was confirmed by X-ray and subsequently readjusted.

4.5. Antibiotics were administered at 10:15pm and 10:18pm. MedSTAR were contacted and reassessed the position of the endotracheal tube. Levi was then transported to the Women's and Children's Hospital at 10:45pm.

4.6. In the Pepperell Report, Professor Pepperell advised obtaining a report from a neonatal paediatrician about the inadequacy of Levi's resuscitation which, at that time, Professor Pepperell regarded as the likely cause of the neurologic problems suffered by Levi. Professor Pepperell also advised that an expert consultant radiologist should be asked to review the neonatal ultrasound and MRI examinations to determine whether cerebral oedema was present consistent with intrapartum hypoxia and whether such findings could be consistent with hypoxia occurring after delivery rather than in labour.[27]

4.7. As suggested by Professor Pepperell, this Court sought independent expert opinion as to whether the resuscitation, including the positioning and the manner of intubation was the cause of Levi's brain damage. Opinions were obtained from Dr Robert Halliday and subsequently from Professor Helen Liley, both of whom are senior specialist neonatologists. Neither considered that there was any departure from usual or best practice in the resuscitation and intubation of Levi, and the management of his carbon dioxide levels both at the Lyell McEwin Hospital and in the care of MedSTAR. In the opinion of both specialists, Levi's resuscitation and intubation did not contribute to the cause of his death.[28]

4.8. Also as suggested by Professor Pepperell, the Court obtained an expert report from Dr Rebecca Linke,[29] a paediatric consultant radiologist, who examined the available imaging of Levi. The first of those was a cranial ultrasound approximately 11 hours after delivery. Dr Linke stated the ultrasound demonstrated slit-like ventricles and abnormal increase in echotexture within the basal ganglia bilaterally. The latter, she

[27] Exhibit C18b, page 12
[28] Exhibits C5a and C7
[29] Exhibit C6a

said, is consistent with ischaemic change and together those appearances are indicative of brain swelling and an early perinatal event. Having regard to the usual timeframe of development of cerebral oedema, findings of which typically become apparent on ultrasound after 24 to 48 hours following hypoxic ischaemic injury, Dr Linke stated that the presence of brain oedema (slit-like ventricles) and areas of abnormality in the thalami and basal ganglia on the initial ultrasound imaging at 11 hours of age is strongly suggestive that the hypoxic insult occurred prior to delivery in the prenatal period. Dr Linke also considered an MRI undertaken at 40.5 hours following delivery, a subsequent MRI at eight days of age and a further ultrasound at nine days of age.

4.9. As I have already alluded to, the care and treatment provided by the Women's and Children's Hospital is not in contention. MRI imaging on 20 and 21 October 2016 reveal the extent of the brain damage, and Levi passed away on 16 November 2016.

5. The Joint Report

5.1. Following the conference between Professors MacLennan and Pepperell, the experts were in complete agreement, as stated in the Joint Report.

5.2. It was agreed that the clinical management during the antenatal period of the pregnancy could not be criticised.

5.3. It was agreed that the management of the first stage of labour was normal.

5.4. The management of labour and delivery was not criticised.

5.5. It was agreed there was not severe hypoxia in labour or evidence for it at birth. Having noted the disagreement between their initial reports about the length of the second stage of labour, it was explained in the Joint Report that Professor Pepperell had reviewed the notes and agreed that the second stage of labour was 3¼ hours. It was agreed that this was longer than normal, and it was considered that the foetal heart rate was mostly normal and satisfactory, noting that a variation occurred during attempting of the forceps delivery, which could have been a factor in the mild acidosis event seen in the cord blood samples obtained immediately after delivery. Reference was made to whether the on-call consultant could have been called to deliver Levi earlier, but it was considered most unlikely that this would have changed the outcome, given the joint experts' opinion of the cause of Levi's death.

5.6. The Joint Report stated that Professor Pepperell had rescinded his opinion that resuscitation and inadequate tracheal tube placement were the likely cause of neonatal asphyxia. Expert neonatal opinion, referred to earlier as having been sought by the Coroners Court at the suggestion of Professor Pepperell, was cited as the reason for rescinding his earlier opinion.

5.7. Professors Pepperell and MacLennan's conclusion is stated thus:

> 'The cause of death was very likely a severe neurological fetal inflammatory response during the neonatal period extending from an intrauterine/placental/fetal infection which would probably have been present for at least several days before birth but not detectable at the time. There was secondary neonatal hypoxia, but this was not the primary cause of death. The source of infection cannot be ascertained but a long maternal history of antenatal and postnatal urinary tract infections and hydronephrosis is noted.
>
> This death was not preventable and was not due to inappropriate obstetric or paediatric care.' [30]

5.8. The basis for the conclusion, stated in short form, appeared in the Joint Report under the heading 'Causation':

> 'The experts now agree that the most likely cause of death was intrauterine infection extending prior to and during labour and throughout the neonatal period. There was severe chorioamnionitis, funisitis and the evidence of infection and thrombosis on the foetal side of the placenta. Later at autopsy increased CD68 activated glial cells were evident suggestive of a severe fetal inflammatory response syndrome. As discussed in Professor MacLennan's report this pathology is currently unpreventable. Clinical signs in pregnancy are late. Antibiotics, steroids and neonatal head cooling have not been shown to prevent this often-fatal disease. Survivors can have severe degrees of cerebral palsy and some of these have been shown to have a genetic variant predisposing the child to neurological infection and brain damage.'[31]

5.9. In his initial report (the MacLennan Report), Professor MacLennan explained in detail the basis for his conclusions, which were adopted by Professor Pepperell in the Joint Report.

5.10. In his initial report, Professor MacLennan referred to and applied the criteria published on behalf of the International Cerebral Palsy Taskforce in 1999 and 2003,[32] in concluding that the evidence did not show that acute hypoxia as a cause of brain damage in Levi began in labour or around birth.

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[30] Exhibit C18, page 3
[31] Exhibit C18, page 3
[32] Exhibit C27, Appendices 1 and 2

5.11. Of these, the four essential criteria to show the presence of hypoxia at birth are:

5.11.1. **Umbilical arterial cord blood gases – a metabolic acidosis around birth (pH less than 7.0 and BASE < -12**. Professor MacLennan explained from the gases measured at birth and thereafter that a moderate metabolic acidosis at birth was likely but without arterial samples it could not be shown to have been severe. Therefore, the first criterion was not met.

5.11.2. Early moderate to severe neonatal encephalopathy. This criterion was met.

5.11.3. **Cerebral palsy of the spastic quadriplegic or dyskinetic type**. Professor MacLennan explained that because Levi did not live long enough to define the type and extent of long-term clinical neurological disability, the criterion was neither met, nor not met. However, because major disability would have been likely, the criterion was met.

5.11.4. Exclusion of other identifiable causes of cerebral palsy e.g., coagulation or genetic disorders, infectious conditions, intrapartum pyrexia, antepartum haemorrhage, prematurity, intrauterine growth restriction, tight nuchal cord, complications of multiple pregnancy. In Levi's case, such an identifiable likely cause other than severe intrapartum hypoxia was considered by Professor MacLennan to be present in the form of acute chorioamnionitis and funisitis along with an intrapartum pyrexia. The placental histology also showed foetal vessel thrombosis and neutrophil invasion of the umbilical foetal vessels. The microscopy of the brain showed '*microglial activation with increased CD68-positive cells*' and '*No white matter cystic changes were observed*'. On this basis, Professor MacLennan concluded that Fetal Inflammatory Response Syndrome was a possible major diagnosis and cause of the neurological outcome. There were other minor risk factors for subsequent neurological damage. Thus, this criterion was not met.

5.12. Professor MacLennan then considered the five non-specific criteria which as a group help to define the timing of a putative acute hypoxic event in labour, as follows:

5.12.1. A sentinel (signal) hypoxic event sufficient to cause sudden severe hypoxia in a healthy foetus e.g., a cord prolapse, antepartum haemorrhage, ruptured uterus, et cetera. The opinion expressed by Professor MacLennan is that no

sentinel acute hypoxic event occurred during labour which could have changed a healthy well-oxygenated foetus suddenly into a severely hypoxic baby. Rather, he said, the established chorioamnionitis and funisitis are likely to have contributed to the poor condition and blood gases of Levi at birth. Therefore this criterion was not met.

5.12.2. **Sudden sustained foetal heart rate bradycardia from that event**. Professor MacLennan stated that there is no electronic trace record of recurrent late decelerations or prolonged bradycardia. Such foetal heart rate alterations were also not recorded on the partogram.[33] There was movement artefact on the trace during forceps application and delivery which could not be assumed to be decelerations. Therefore, this criterion was not met.

5.12.3. **An Apgar score under 4 after 5 minutes**. Professor MacLennan considered that the Apgar score of 3 at five minutes and 4 at 10 minutes essentially met the criterion of under 4 after 5 minutes and, therefore, this criterion was met.

5.12.4. **Signs of multisystem failure in the neonate**. Professor MacLennan said there was early mild renal dysfunction according to slightly raised urea and creatinine levels during the first neonatal week and therefore, on balance, this criterion was just met.

5.12.5. **Early (within 5 days) neuroimaging signs of oedema and intracranial haemorrhage**. No brain oedema or intracranial haemorrhage was reported on brain imaging during the first neonatal week although potential ischaemic changes were noted. Therefore this criterion was not met.

5.13. In summary, a major essential criterion to show that a severe acute hypoxic event initiated the neuropathology was not met because another possible cause of neurological disability was present, and in terms of a putative intrapartum timing of the brain damage there was a lack of a hypoxic sentinel event during labour and no subsequent bradycardia. Only two of the five non-specific timing criteria were met.

[33] A composite graphical record of key data (maternal and foetal) during labour

5.14. On the basis of the application of those criteria, which Professor MacLennan said exemplified the consensus of modern senior scientific foetal and maternal opinion around the world, a clear intrapartum severe acute hypoxic event was not established.

5.15. On the other hand, opined Professor MacLennan there was supporting evidence of major intrauterine pathology and, on the balance of probabilities, foetal inflammatory response syndrome as evidenced by:

- acute chorioamnionitis and funisitis (which is a longer standing inflammation usually of 24 hours or more).
- maternal intrapartum pyrexia which is a relatively late sign of intrauterine infection.
- thrombus in a foetal stem vessel with two groups of avascular villi (a subacute sign of placental pathology on the foetal side).

6. Further expert opinion and reports

6.1. Associate Professor Moore

6.2. Since receiving all the evidence so far referred to in this Finding, the Court, through counsel assisting, directed a query to Professor Moore and received a response, being a statement annexed to an email dated 22 June 2022, marked as Exhibit C4b.

6.3. Professor Moore was directed to the conclusion of Professors MacLennan and Pepperell referred to in paragraph 1.24 of this Finding:

> 'The cause of death was very likely a severe neurological foetal inflammatory response during the neonatal period extending from an intrauterine/placental/fetal infection which would probably have been present for at least several days before birth but not detectable at that time. There was secondary neonatal hypoxia, but this was not the primary cause of death. The source of the infection cannot be ascertained but a long maternal history of antenatal and postnatal urinary tract infections and hydronephrosis is noted.'

6.4. Professor Moore was asked for her opinion as to how the cause of death would be most appropriately expressed in the event that the Court accepted this statement of Professors Pepperell and MacLennan, given Professor Moore's pathological findings and her conclusion as to cause of death of '*severe neonatal encephalopathy, post palliative care*'.

6.5. In responding, Professor Moore pointed out, in relation to the placental examination, that the severity of the chorioamnionitis was not indicated in the original report and that she did not comment on it in her review. She stated it would be best categorised as mild. In other words, Professor Moore did not accept the description by Professors MacLennan and Pepperell of the chorioamnionitis as severe.

6.6. Professor Moore considered the other available history, including that provided by the expert witnesses in relation to the history, and concluded:

> 'I agree with the assessment of the other experts that the brain injury occurred prior to birth.
>
> I agree that there is reasonable possibility that intrauterine infection may have been the cause.
>
> I do not agree the chorioamnionitis was severe.
>
> I have no reason to believe the chorioamnionitis was present for several days.'

6.7. Associate Professor Moore updated her opinion as to cause of death as '*severe neonatal encephalopathy, post palliative care. Possible underlying cause: intrauterine infection*'.

6.8. <u>Professors MacLennan and Pepperell</u>

6.9. Associate Professor Moore's further report was forwarded to Professors MacLennan and Pepperell for consideration and comment, resulting in a further report dated 21 July 2022.[34]

6.10. Professors MacLennan and Pepperell stated that this did not change their opinion on the cause of death or the clinical management. They accepted that the chorioamnionitis was not '*severe*' commenting that it was *'histologically confirmed along with funisitis which appears after a longer standing chorioamnionitis*'.

6.11. They stated:

> 'Our assessment of a fetal inflammatory syndrome severe enough to cause the death of Levi was based on the whole clinical evidence and our perinatal expertise. This evidence included
>
> - A long medical history of urinary tract infections
> - urinary tract infections during the pregnancy
> - kidney damage as shown by bilateral hydronephrosis

[34] Administratively tendered and marked Exhibit C34

- thrombocytopenia in late pregnancy
- raised white blood counts before and after delivery
- intrapartum maternal pyrexia treated with antibiotics
- raised neonatal nucleated red blood cell counts
- postnatal urinary tract infection
- acute funisitis
- chorioamnionitis
- CD68 cells seen on histology.'

6.12. Professors MacLennan and Pepperell agreed to remove the word '*severe*' as it applied to the histological evidence but maintained that '*on the balance of probabilities the fetal inflammatory response was sufficient to cause the neonatal death*'.

6.13. Professors MacLennan and Pepperell emphasised the absence of any obstetric report claiming objective evidence of another cause and in particular a preventable cause.

6.14. Discussion

6.15. There is a tension between the opinions of Professors MacLennan and Pepperell on the one hand and Associate Professor Moore on the other. Professor Moore appears to place the likelihood of intrauterine infection being the root cause of Levi's brain injury as a reasonable possibility, whereas Professors MacLennan and Pepperell regard it as very likely. Further, Professor Moore disagrees that an infection may have been present for several days.

6.16. In considering this issue I bear in mind the different disciplines in which these experts practise. I bear in mind Professor Moore's recommendation at the conclusion of her initial post mortem report that there be a review of the labour and delivery notes and CTG by a consultant obstetrician to see if appropriate guidelines for management of labour were followed and if examination of the CTG trace can give an indication of the timing of the brain damage.

6.17. Professors MacLennan and Pepperell provided the suggested expert clinical opinions and agreed that the clinical management during the antenatal period of the pregnancy was normal and could not be criticised. They raised a query about whether Levi could have been delivered a little earlier but expressed the view that it was most unlikely that an earlier delivery would have changed the outcome. They did not otherwise criticise

the management of labour and delivery and stated that there was not severe hypoxia in labour or evidence for this at birth.

6.18. Findings by the Coroners Court are to be made on the balance of probabilities. I find that Levi's cause of death is properly expressed as '*severe neonatal encephalopathy, post palliative care*'.

6.19. The strong opinion of Professors MacLennan and Pepperell, applying the same standard of proof, is that the severe neonatal encephalopathy was due to a severe neurological foetal inflammatory response due to an intrauterine/placental/foetal infection.

6.20. Their opinion is also that Levi's death was not preventable and was not due to inappropriate obstetric or paediatric care.

6.21. Professor Moore's conclusions, taken together with the other various experts reports to which I have referred, do not suggest inappropriate obstetric or paediatric care, or that Levi's death was preventable, even though she does not conclude, on the basis of the pathological findings, that the neonatal encephalopathy was more likely than not due to intrauterine infection. Rather, she regards that as a reasonable possibility.

6.22. Having regard to this point of disagreement I have come to the conclusion that it is not appropriate to make a positive finding as to the cause of Levi's neonatal encephalopathy, but rather to find that intrauterine infection is a possible cause, as suggested by Professor Moore. I nevertheless find that there is no evidence of inappropriate obstetric or paediatric care provided to Ms Noakes or to Levi, which may have caused or contributed to his death.

7. Conclusions

7.1. Baby Levi Shane Vanin was born on 18 October 2016 at the Lyell McEwin Hospital at Elizabeth Vale, South Australia.

7.2. Levi died on 16 November 2016 at the Women's and Children's Hospital, North Adelaide, South Australia.

7.3. The cause of death was severe neonatal encephalopathy, post palliative care.

7.4. Levi's death was a tragic loss for both Ms Noakes, Mr Vanin and their family and friends. The evidence received during this inquest has demonstrated that although the induction, labour, stage 2, and delivery were complicated and protracted, Levi's death was not preventable.

7.5. I make no recommendations.

Key Words: Infant; Hypoxia; Forcep Delivery

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 30th day of June, 2023.

State Coroner