IN THE CORONERS COURT
OF VICTORIA
AT MELBOURNE

Court Reference: COR 2019 5601

FINDING INTO DEATH WITHOUT INQUEST

Form 38 Rule 63(2)
*Section 67 of the **Coroners Act 2008***

Findings of:	Sarah Gebert, Coroner
Deceased:	David John MAIN
Date of birth:	13 May 1965
Date of death:	14 October 2019
Cause of death:	*Toxic leukoencephalopathy (of uncertain aetiology)*
Place of death:	Peninsula Private Hospital, 525 McClelland Dr, Frankston, Victoria
Relevant matters:	*Toxic encephalopathy (TLE), Ketamine infusion*

INTRODUCTION

1. David John Main,[1] born on 13 May 1965, was 54 years old at the time of his death. He lived at home with his mother, Beverley Simpson.

2. David was an *avid* Richmond supporter and a *choc-a-holic.* He was described by one of his friends as *a lovely guy that wouldn't hurt a fly*.

3. In 1986 David was involved in a car accident where he sustained a back injury which caused him pain throughout his life. His mother said it was a *miracle* he survived the accident and noted that he also suffered *psychologically* as a result of what he observed at the scene. David was involved in a further car accident in 2000 which aggravated his existing back pain.

4. Mrs Simpson said that her son hadn't been well in the last 10 years of his life but it had become *worse for him in the last few* [years] *due to debilitating pain.*

5. On 14 October 2019, David passed away at the Peninsula Private Hospital after being transferred from Beleura Private Hospital where he had undergone a ketamine[2] assisted oxycodone detoxification.

THE CORONIAL INVESTIGATION

6. David's death was reported to the coroner as it fell within the definition of a reportable death in the *Coroners Act 2008* (**the Act**). Reportable deaths include deaths that are unexpected, unnatural or violent or result from accident or injury.

7. The role of a coroner is to independently investigate reportable deaths to establish, if possible, identity, medical cause of death, and surrounding circumstances. Surrounding circumstances are limited to events which are sufficiently proximate and causally related to the death. The purpose of a coronial investigation is to establish the facts, not to cast blame or determine criminal or civil liability.

8. Under the Act, coroners also have the important functions of helping to prevent deaths and promoting public health and safety and the administration of justice through the making of

[1] Referred to in my finding as 'David' unless more formality is required.
[2] Ketamine is a scheduled drug, approved for use in hospitals as an anaesthetic and an analgesic. However, it is also a commonly abused "recreational" drug, due to its hallucinogenic, tranquilizing and dissociative effects.

comments or recommendations in appropriate cases about any matter connected to the death under investigation.

9. Victoria Police assigned Leading Senior Constable Emma Rainey (**SC Rainey**) to be the Coroner's Investigator. LSC Rainey conducted inquiries on my behalf, including taking statements from witnesses and submitting a coronial brief of evidence. The coronial brief comprises statements from David's mother, his two best friends, Dr Margaret Baker[3], Director of Clinical Services, Beleura Private Hospital, the pathologist who examined him and the Coroner's Investigator as well as other relevant documentation.

10. As part of the investigation, this case was referred to the Coroners Prevention Unit (**CPU**).[4] The CPU were asked to review David's admission and care at the Beleura Private Hospital and the Peninsula Private Hospital.

11. In the course of the coronial investigation copies of David's medical records were obtained from these hospitals and statements were also obtained from his general practitioner (**GP**), Dr Peter Drake and Dr Murray Taverner, pain specialist, Frankston Pain Management.

12. This finding draws on the totality of the coronial investigation into David's death, including evidence contained in the coronial brief and information provided by the CPU. Whilst I have reviewed all the material, I will only refer to that which is directly relevant to my findings or necessary for narrative clarity. In the coronial jurisdiction, facts must be established on the balance of probabilities.[5]

Background

13. David was referred to Dr Taverner by his GP Dr Peter Drake, for opinion and management of his multiple pains, opioid dependence and likely opioid induced hyperalgesia. Dr Taverner initially saw him on 21 June 2019.

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[3] Margaret Baker made three statements for the investigation dated 30 June 2020, 8 September 2020 and 7 May 2021.

[4] The Coroners Prevention Unit (CPU) was established in 2008 to strengthen the prevention role of the coroner. The unit assists the Coroner with research in matters related to public health and safety and in relation to the formulation of prevention recommendations. The CPU also reviews medical care and treatment in cases referred by the coroner. The CPU is comprised of health professionals with training in a range of areas including medicine, nursing, public health and mental health.

[5] Subject to the principles enunciated in *Briginshaw v Briginshaw* (1938) 60 CLR 336. The effect of this and similar authorities is that coroners should not make adverse findings against, or comments about, individuals unless the evidence provides a comfortable level of satisfaction as to those matters taking into account the consequences of such findings or comments.

14. Dr Taverner made a thorough assessment of David's medical, pain, functional, emotional and developmental history. He was noted to have had a significant pain history from multiple injuries sustained over a 30-year period and was an ex-intravenous (**IV**) drug user.[6]

15. Dr Taverner reported that David had a complex mix of cervical, thoracic and lumbar spine pain with significant amplifying psychosocial factors, high opioid use and high pain interference. David's oral morphine equivalent daily dose (oMEDD) was 330mg morphine daily at the time of his first assessment.

16. Dr Taverner explained to David that daily doses above 50mg are associated with a four-fold increased cardiorespiratory risk and doses above 100mg morphine equivalents are not recommended or supported for pain management. Doses above 100mg morphine equivalents are treating dependence and affective (mood) elements rather than pain. Severe pain despite adequate doses of several opioids, indicates the pain is opioid insensitive and ideally analgesics should be weaned so that they do not add to the symptom burden.

17. Dr Taverner considered that David needed to reduce his opioids, such as the Oxycontin he had been taking, as it was likely contributing to or maintaining pain by a hyperalgesic effect[7]. He recommended a seven-day ketamine infusion to help reduce David's sensitisation, unhelpful medications, improve activity tolerance, mood and coping and to guide future treatment approaches and then reassess the need for diagnostic injections, radio frequency treatment and other interventions.

18. Dr Taverner noted that ketamine is a widely used medicine in pain and palliative care and is used for difficult-to-treat pain.

19. Dr Taverner advised David of the following possible side effects which included: nausea and hallucinations were very common (>10%), vomiting, confusion, anxiety, impaired memory, headache, blurred vision, tingling sensations, insomnia, somnolence, chest tightness, cramps, dizziness, fatigue, hypertension, elevated mood, shortness of breath, transient hepatic dysfunction were common (1-10%), and cystitis that may present as a sterile urinary tract infection (UTI) was a rare problem.

20. Dr Taverner recommended a multidisciplinary assessment and management via inpatient opioid reduction at Beleura Private Hospital (Ramsay Healthcare) followed by a rehabilitation admission to St John of God (**SJOG**).

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[6] He had had hepatitis C and possibly had hepatitis B.
[7] Where a person develops an increased sensitivity to pain.

21. David was reviewed to complete his examination and assessment on 28 June 2019. He was again reviewed on 12 September 2019 to organise the inpatient ketamine infusion and opioid detoxification.

MATTERS IN RELATION TO WHICH A FINDING MUST, IF POSSIBLE, BE MADE

Circumstances in which the death occurred

22. On Monday, 30 September 2019, David was taken the Beleura Private Hospital by his mother where he was admitted for a ketamine assisted oxycodone detoxification under the care of Dr Taverner.

23. Dr Taverner's standard ketamine protocol started with a 5mg loading bolus and 10mg/hour infusion that is adjusted by 2-5mg/hour to a maximum 60mg/hour as needed and tolerated to control pain and withdrawal symptoms.

24. Dr Taverner also prescribed some 'ketamine associated' medications to help manage the withdrawals. They included:
    - Nightly zopiclone[8] to assist with sleep (ketamine activates);
    - Ondansetron[9] for nausea which was used once;
    - Quetiapine[10] 25-50mg q2h[11] for distress that was used once or twice daily from 30 September to 4 October 2019 but five times on 5 October 2019;
    - Regular oral clonidine[12] 50-100mcg bd[13] and rescue IV 50mcg clonidine for withdrawal symptoms that was used on three occasions; and
    - Diazepam[14] 5mg qid[15] prn[16] that was used twice daily for the six days during the infusion period. David also had daily doses of 10mg OxyContin for withdrawal and pain on 4, 5 and 6 October 2019.

25. Dr Taverner planned a three-stage opioid reduction over 7-14 days, with the first goal being to halve the OxyContin, the second stage after three or four days was to reduce OxyContin to 10-20mg bd and then finally to cease it altogether.

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[8] Sedative.
[9] Anti nausea medication.
[10] Major tranquiliser.
[11] Every 2 hours.
[12] An anti-hypertensive medication used for management of substance withdrawal to alleviate symptoms.
[13] Twice daily.
[14] Benzodiazepine sedative.
[15] Four times a day.
[16] When required.

26. The ketamine infusion was started with the 5mg bolus, 10mg per hour infusion and titrated to 30mg per hour overnight. David's baseline urine drug screen taken at 8.30pm on 30 September 2019 was positive for opioids (codeine 808 mcg/L- not known to be prescribed) and cannabinoids[17]. Baseline pain was 8/10[18] dropping to 5-6/10 during the day. David told Dr Sam Lieblich, liaison psychiatrist, *the opiates just haven't worked*.

27. On 1 October 2019, the infusion was stopped for about six hours due to unexpected sedation from 7.00am. OxyContin was reduced to 20mg bd. The ketamine infusion was resumed at 20mg per hour at 6.00pm.

28. On 2 October 2019, David was noted to be sleepy but rousable, curled up, not mobile. The OxyContin 20mg bd was stopped after the 8.00pm dose as he seemed to be coping better than expected and was more sleepy than expected. The ketamine infusion was slowly increased to 34 mg per hour. Reported pain was 5-8/10.

29. On 3 October 2019, David was more awake and alert. A second drug screen taken at 5.30pm on 3 October 2019 was positive for cannabinoids and benzodiazepines (prescribed), no opioids were detected. The ketamine infusion was titrated to 40mg per hour. Reported pain was 5-6/10.

30. On 4 October 2019 David slept overnight, reported pain varied between 3-7/10, the ketamine was increased to 42 mg per hour. His GCS was 15[19].

31. On 5 October 2019, David complained of pain 9/10, on examination was myofascial[20] and widespread rather than focal. The ketamine infusion was running at 48 mg per hour. SJOG rehabilitation assessment for the following Monday was requested.

32. David was also reviewed by Dr Jeremy Stone, liaison psychiatrist on 5 October 2019. Dr Stone recommended continuing with the current plan as David was doing okay.

33. On 6 October 2019 at around 3.00pm the High Dependency Unit nurse advised Dr Taverner that David had a lowered GCS score of 12/15. The ketamine infusion was running at 28mg per hour. David's blood glucose was 7.6 mmol/L[21], blood pressure (**BP**) 163/97, oxygen

[17] A group of substances found in the cannabis plant.
[18] Numerical rating scales (NRS) use numbers to rate pain, with 0 classed as no pain and 10 being the worst pain possible.
[19] The Glasgow Coma Scale (GCS) is the most commonly used scoring system to describe someone's consciousness. Clinicians use this scale to rate the best eye opening response, the best verbal response, and the best motor response an individual makes. Best response is 15/15 and worst is 3/15.
[20] Muscular.
[21] Normal range 3.5 – 7.5 mml/L.

saturation was 92 per cent[22]. His pulse was 86 beats per minute and regular. He was noted to be 'pink and warm' and his respiration was 24 per minute[23]. The ketamine infusion was stopped at this time.

34. The physician on-call Dr Vikram Bhalla reviewed David at 3.35pm noting a lowered conscious state and low GCS score of 5/15. David's pupils were normal and reactive. His respiratory rate was 22 per minute. Oxygen saturation was 100 per cent on 2 litres of oxygen. Dr Bhalla commented on possible causes of the decreased conscious state being polypharmacy[24], intracranial bleed, sepsis and liver problems.

35. David was transferred to Peninsula Private Hospital intensive care unit (**ICU**) where it was documented that David presented with an altered level of consciousness without focal neurological signs. David was afebrile and described as cachexic and emaciated. Antibiotics and antiviral agents were commenced.

36. On 7 October 2019, the working diagnosis was 'encephalopathy[25] FI'. The CT scan findings were of '*diffuse white matter disease*'. The treating team considered that the medications at Beleura Hospital did not adequately explain David's clinical condition. A CT angiogram and venogram to assess for vascular issues was normal and David was intubated.

37. On 8 October 2019, neurologist Dr Swee Tan reviewed David and his MRI. He concluded that David had widespread leukoencephalopathy[26] with possible cerebral oedema[27] that had developed during a ketamine infusion. There was no evidence of a stroke or haemorrhage. He wrote that ketamine had <u>not</u> been reported to cause such pathology and that he could not identify a potential drug/toxic cause. His differential diagnosis was viral/bacterial infection or an unidentified toxin or an autoimmune encephalitis. Further tests were ordered to check syphilis serology and vitamin B12 deficiency which were all negative.

38. On 9 October 2019, the ICU doctors considered the most likely cause of David's condition was toxic encephalopathy. Levels of lead, tin and aluminium were ordered which were <u>negative</u>. Dr Taverner reviewed David and wrote the MRI showed leukoencephalopathy and that the literature showed relationships to several drugs: heroin, cocaine, 'chasing the

[22] Oxygen saturations without supplemental oxygen and normal lung anatomy, normal range >96 per cent.
[23] Normal respiratory rate 8-16 per minute.
[24] Multiple medications producing interactions.
[25] A general term for a disease that affects the structure or function of the brain.
[26] A general term for a disease that affects the white matter of the brain.
[27] Swelling of the brain.

dragon[28]' snorting, but that he had found no reports of ketamine causing the condition. He suggested urine drug samples from the week prior be sent for further analysis. He also considered that rapid rehydration following a period of dehydration may have caused cerebral oedema.

39. On 11 October 2019, Dr Tan again reviewed David and noted the urine drug screen results showed opioids, benzodiazepines and cannabinoids. He wrote "supportive care, recovery expected".

40. Dr Taverner spoke with David's mother on 12 October 2019 by phone. She believed that he had not been using any recreational drugs for some years other than 1g of marijuana daily.

41. Also on 12 October 2019, the ICU team consulted an Austin Hospital toxicologist, Dr Liang, regarding potential causes and management. Synthetic cannabis or methamphetamine were suggested as possible causes but the toxicologist was unsure if ketamine could cause the problem. Dr Liang recommended ceasing ketamine. Low dose propofol was commenced for sedation. David was extubated. He developed a right sided paralysis and complex partial seizures which were treated with anticonvulsants. It was decided he would not be re-intubated and in consultation with family, was transitioned to comfort care. David unfortunately died at 4.00am on 14 October 2019.

42. There was a suggestion that David had been supplied with drugs while an in-patient at the Beleura Hospital prior to his deterioration. David's mother said that David only had two friends, being a loner, and that both had confirmed to her they had not supplied any substances to David (also confirmed by their statements). The Director of Clinical Services, Beleura Private Hospital advised that she had spoken to the nursing staff who cared for David and they were unable to recall him being visited *by anyone at all.*[29] I note that his mother visited him on 3, 4, 5 and 6 October 2019. She noted that he was in *good spirits* on 5 October but had deteriorated the following day. I have been unable to find any evidence to suggest that David was provided with drugs by an outside source prior to his deterioration.

Identity of the deceased

43. On 17 October 2019, Patricia Baker visually identified her nephew, David John Main, born 13 May 1965.

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[28] Inhaling the vapor from a heated solution of morphine, heroin, oxycodone, opium, or ya ba (a pill containing caffeine and methamphetamine).
[29] Statement of Margaret Baker dated 8 September 2020.

44. Identity is not in dispute and requires no further investigation.

Medical cause of death

45. Specialist Forensic Pathologist Dr Malcolm Dodd from the Victorian Institute of Forensic Medicine (**VIFM**) conducted a post mortem examination on 17 October 2019 and provided a written report of his findings dated 10 March 2020.

46. Specialist Forensic Pathologist Dr Linda Iles also from VIFM prepared a Neuropathology Report following an examination of the brain on 22 October 2019 and provided a written report of her findings dated 20 December 2020.

47. Dr Iles noted in her report,

> *The pattern of pathology in this case is unusual. The white matter changes observed raise a possibility of a toxic leukoencephalopathy; this type of pathology can be seen following opioid toxicity. Elsewhere, there are some features that are seen in acute haemorrhagic leukoencephalopathy (Hurst's disease), but the full constellation of features is unusual. A systemic microvasculopathic process should be excluded. The features seen are most in keeping with a toxic leukoencephalopathic process.*

48. Dr Dodd noted that toxicological analysis of body fluids showed medications consistent with therapeutic usage. In addition, there were no apparent suspicious circumstances.

49. Dr Dodd provided an opinion that the medical cause of death was *Toxic leukoencephalopathy (of uncertain aetiology*[30]*).*

50. I accept Dr Dodd's opinion.

CPU REVIEW

51. The CPU conducted a review of the available evidence including David's medical records and statements from clinicians who had cared for him.

<u>Toxic encephalopathy</u>

51. The term toxic encephalopathy (**TLE**) is used to indicate brain dysfunction caused by toxic exposure.[31] There are several forms described. David's case would most likely fit with *acute diffuse* toxic encephalopathy.

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[30] The cause, set of causes, or manner of causation of a disease or condition.

Acute diffuse toxic encephalopathy

52. Acute diffuse TLE reflects a global cerebral dysfunction of rapid onset (typically days or weeks) and may be associated with alterations in the level of consciousness. The neurotoxins that produce acute encephalopathy interfere with basic cell functions in the brain. The causative agents include organic solvents[32], and some gases (e.g., gas anaesthetics, carbon monoxide, hydrogen sulphide, and cyanide), heavy metals (e.g., mercury, lead and tin). In general, the greater the exposure, the more severe the impairment of cerebral function and consciousness. Diagnosis does not generally present a challenge for acute syndromes, because the exposure and clinical manifestations are likely to be closely linked in time. The treatment of diffuse acute TLE is primarily supportive, starting with removal of the exposure source. For most of the neurotoxins that act diffusely on the brain, recovery from acute exposure is complete.

Opioid toxic encephalopathy

53. As noted above, Dr Iles commented that the changes observed in the brain can be seen following opioid toxicity.

54. A review of the literature revealed the following information:

- In an article entitled Neurotoxicology Syndromes Associated with Drugs of Abuse[33] the syndromes described were acute and chronic problems associated with opioid misuse and opioid withdrawal syndromes. There was no mention of opioid induced TLE as an opioid associated neurotoxicological syndrome.
- In another article entitled Severe Leukoencephalopathy Following Acute Oxycodone Intoxication[34] TLE followed an acute toxic ingestion of oxycodone in a very large dose.
- In "Opioid-induced toxic leukoencephalopathy: A case report and review of the literature"[35] a case report of a 4-year-old boy who took an accidental overdose of

[31] Kim Y, Kim JW. Toxic encephalopathy. Saf Health Work. 2012 Dec;3(4):243-56. doi: 10.5491/SHAW.2012.3.4.243. Epub 2012 Nov 30. PMID: 23251840; PMCID: PMC3521923.

[32] A solvent is a substance that dissolves a solute, resulting in a solution. Specific uses for organic solvents are in dry cleaning (e.g. tetrachloroethylene); as paint thinners (toluene, turpentine); as nail polish removers and solvents of glue (acetone, methyl acetate, ethyl acetate); in spot removers (hexane, petrol ether); in detergents (citrus terpenes); and in perfumes (ethanol).

[33] Rachel A. Caplan MD, Jonah P. Zuflacht MD, Jed A. Barash MD, MHS and Corey R. Fehnel MD, MPH. Neurotoxicology Syndromes Associated with Drugs of Abuse. Neurologic Clinics, 2020-11-01, Volume 38, Issue 4, Pages 983-996.

[34] Yazmin Morales Odia, Madhavi Jinka, Wendy C. Ziai. Severe Leukoencephalopathy Following Acute Oxycodone Intoxication. Neurocrit Care (2010) 13:93–97 DOI 10.1007/s12028-010-9373-y

oxycodone is described. Other paediatric cases in the literature are listed but there is no record of the amount and timing of the ingestion. Presumably in this paediatric population they are accidental toxic ingestions.

- In another article that reviews the pathology and causes of TLE[36] there is no mention of opioid induced TLE.

55. The CPU sought advice from Dr Iles regarding when opioid induced TLE was likely to occur following opioid ingestion and whether it occurred in the setting of overdose only, compared with long term use as in David's case. Dr Iles reported that in her experience she had seen one or two cases where it had occurred as an idiosyncratic reaction in the setting of overdose.

Dr Taverner

56. Dr Taverner provided a detailed statement where he described his extensive experience with pain management and ketamine infusions. He stated that he had been using ketamine infusions for about 10 years and had conducted more than 1500 infusions - over 500 would have been for opioid reduction and cessation. It was his experience that ketamine infusion had been very effective in reducing opioid dependence and dosage.

57. He noted that the primary goal of ketamine treatment is to manage the patient's pain while they go through withdrawal of their opioid dependence.

58. Dr Taverner also advised that he had no prior experience with TLE, and that he understood that TLE is a condition that has been reported in active users of heroin, cocaine, marijuana and a number of other drugs. Dr Taverner conducted a literature review and found no reports of this condition associated with ketamine treatment.

59. Dr Taverner had never experienced a patient with TLE and stated that he discussed David's case with other experienced pain medication specialists, and it appeared that they had no experience of it either. He did not know why David deteriorated, and his deterioration to death was also unexpected as there is a variable presentation and progression and death is not an inevitable outcome of TLE.

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[35] Wheaton T, Toll BJ, Breznak K, Da-Silva S, Melvin J, Misra A, Hwang SW. Opioid-induced toxic leukoencephalopathy: A case report and review of the literature. Heliyon. 2019 Dec 11;5(12):e03005. doi: 10.1016/j.heliyon.2019.e03005. PMID: 31879710; PMCID: PMC6920259.

[36] Kim Y, Kim JW. Toxic encephalopathy. Saf Health Work. 2012 Dec;3(4):243-56. doi: 10.5491/SHAW.2012.3.4.243. Epub 2012 Nov 30. PMID: 23251840; PMCID: PMC3521923.

60. Dr Taverner also wrote:

> *I have thought about David's deterioration and death a great deal. We noted at the time of his treatment that David seemed unexpectedly more sensitive to the supplementary opioids we used to ameliorate the opioid step-down and withdrawals. He was sleepier than expected with the ketamine infusion, the opioids were able to be reduced faster than expected, with less distress than expected and less ketamine and other supplementary drugs than expected. His lack of withdrawal symptoms was unusual in the light of his very high opioid dose. I went so far as to question whether he had in fact been opioid dependent at all but his screening tests on admission were positive for opiates in the form of codeine. On discussion with the Dorevitch toxicology technicians I have since learned that the urinary drug screen doesn't measure oxycodone unless specifically requested. The referral letter from Dr Drake makes no mention of Panadeine Forte. The origin of the codeine is unknown. I am thus unable to confirm that he was taking the prescribed Oxycontin.*
>
> *We encountered fluctuating consciousness that was unusual and unexplained by the ketamine which had to be stopped and started several times.*
>
> *Since David's death I am vigilant with monitoring my patients for any similar symptoms. As I still do not know what caused his deterioration, it is difficult to institute changes in response to it.*
>
> *If I did encounter a patient with a similar reaction to David's (exceedingly sleepy, minimal withdrawal symptoms, sensitivity to opioid doses) I would likely arrange an MRI quickly to exclude TLE.*

Conclusion

61. The CPU noted that David developed what remains an unexplained TLE.

62. A review of the literature by Dr Taverner and the CPU cannot identify an obvious cause of the TLE. Nor has ketamine ever been reported as causing TLE. Dr Iles thought the histopathological findings were most consistent with a TLE from opioids, but she could not be certain they were the cause in this case. It is possible that one of the drugs used in the detoxification program may have contributed to the syndrome, but the CPU noted that this remains speculation.

63. The CPU did however consider that Dr Taverners's management and the management at Beleura and Peninsula Private Hospitals were reasonable and could not identify any prevention opportunities (noting that the finding will be published).

64. I accept the CPU's advice in relation to this matter and I am grateful for their assistance with the investigation.

65. I acknowledge that it is disappointing that the coronial investigation was unable to identify the underlying cause of David's TLE, but I hope his mother can take comfort that no issues were identified in relation to David's medical management proximate to his death.

FINDINGS AND CONCLUSION

66. Pursuant to section 67(1) of the Act I make the following findings:

    a. the identity of the Deceased was David John Main, born 13 May 1965;

    b. the death occurred on 14 October 2019 at the Peninsula Private Hospital, 525 McClelland Dr, Frankston, Victoria, from *Toxic leukoencephalopathy (of uncertain aetiology)*, and

    c. the death occurred in the circumstances described above.

67. I convey my sincere condolences to David's family for their loss and the tragic circumstances in which his death occurred.

68. Pursuant to section 73(1B) of the Act, I order that this finding (in a redacted format) be published on the Coroners Court of Victoria website in accordance with the rules.

69. I direct that a copy of this finding be provided to the following:

Mrs Beverley Simpson, Senior Next of Kin

Australian Health Practitioner Regulation Agency (AHPRA)

Dr Murray Grant Taverner, Frankston Pain Management

Dr Peter Drake, Jasper Family Medical Practice

Safer Care Victoria

Leading Senior Constable Emma Rainey, Victoria Police, Coroner's Investigator

Signature:

SARAH GEBERT

Date: 16 August 2021

NOTE: Under section 83 of the ***Coroners Act 2008*** ('the Act'), a person with sufficient interest in an investigation may appeal to the Trial Division of the Supreme Court against the findings of a coroner in respect of a death after an investigation. An appeal must be made within 6 months after the day on which the determination is made, unless the Supreme Court grants leave to appeal out of time under section 86 of the Act.