IN THE CORONERS COURT
OF VICTORIA
AT MELBOURNE

Court Reference: **COR 2015 5613**

FINDING INTO DEATH WITHOUT INQUEST

Form 38 Rule 60(2)
Section 67 of the Coroners Act 2008
Amended pursuant to Section 76 of the Coroners Act 2008 on 8 August 2019[1]

Findings of:	**MR JOHN OLLE, CORONER**
Deceased:	**ALLISON JUDITH ALLAN**
Date of birth:	**29 JUNE 1972**
Date of death:	**3 NOVEMBER 2015**
Cause of death:	**I(a) HYPOXIC ISCHAEMIC BRAIN INJURY** **I(b) ALCOHOL AND SODIUM VALPROATE TOXICITY**
Place of death:	**SOUTH WEST HEALTHCARE,** **25 RYOT STREET,** **WARNAMBOOL, VICTORIA 3280**

[1] This document is an amended version of the finding into the death of Allison Judith Allan dated 30 July 2019. The sixth and final recommendation was accidentally omitted from the original version and has been added to this version pursuant to Section 76 of the *Coroners Act 2008* (Vic).

HIS HONOUR:

BACKGROUND

1. Allison Judith Allan was born on 29 June 1972. She was 43 years old at the time of her death. Alison lived with her partner Reno Grima in Portland. Allison worked as a disability support officer.

2. Allison had a medical history of epilepsy, depression, alcohol abuse and she had three previous suicide attempts by overdose. Allison's regular medications included the antidepressant desvenlafaxine[2], which was prescribed in July 2015 by General Practitioner Dr Jesse Das due to suicidal depressive symptoms and she was also prescribed the anticonvulsant sodium valproate (Epilim).[3]

3. Dr Das referred Allison to Portland Adult Mental Health Service (MHS) in July 2015. Portland MHS notes by Consultant Psychiatrist Dr Jayanta Deb recorded that within a few weeks of being prescribed desvenlafaxine Allison reported a significant mental health improvement. Allison had returned to work, was no longer depressed, had normal diet and sleep patterns, and improved energy levels. Follow up phone calls and in-person consultations continued with the Portland MHS over the subsequent months, but she cancelled her 21 October 2015 appointment, advising she would reschedule it on another day. Allison did not reschedule her appointment with Portland MHS, and they were unable to contact Allison by telephone on 26 October 2015.

THE PURPOSE OF A CORONIAL INVESTIGATION

4. Allison's death constituted a '*reportable death*' under the *Coroners Act 2008* (Vic), as her death occurred in Victoria, and was both unexpected and unnatural.[4]

5. The jurisdiction of the Coroners Court of Victoria is inquisitorial.[5] The purpose of a coronial investigation is independently to investigate a reportable death to ascertain, if possible, the identity of the deceased person, the cause of death and the circumstances in which death occurred.

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[2] Antidepressant belonging to a group of medications called selective serotonin and norepinephrine reuptake inhibitors (SNRIs).
[3] A common anticonvulsant medication used to reduce seizures in epilepsy.
[4] Section 4, definition of 'Reportable death', *Coroners Act 2008.*
[5] Section 89(4) *Coroners Act 2008.*

6. It is not the role of the coroner to lay or apportion blame, but to establish the facts.[6] It is not the coroner's role to determine criminal or civil liability arising from the death under investigation, or to determine disciplinary matters.

7. The "cause of death" refers to the medical cause of death, incorporating where possible, the mode or mechanism of death.

8. For coronial purposes, the circumstances in which death occurred refers to the context or background and surrounding circumstances of the death. Rather than being a consideration of all circumstances which might form part of a narrative culminating in the death, it is confined to those circumstances which are sufficiently proximate and causally relevant to the death.

9. The broader purpose of coronial investigations is to contribute to a reduction in the number of preventable deaths, both through the observations made in the investigation findings and by the making of recommendations by coroners. This is generally referred to as the 'prevention' role.

10. Coroners are also empowered:

    (a) to report to the Attorney-General on a death;

    (b) to comment on any matter connected with the death they have investigated, including matters of public health or safety and the administration of justice; and

    (c) to make recommendations to any Minister or public statutory authority on any matter connected with the death, including public health or safety or the administration of justice. These powers are the vehicles by which the prevention role may be advanced.

11. All coronial findings must be made based on proof of relevant facts on the balance of probabilities. In determining these matters, I am guided by the principles enunciated in *Briginshaw* v *Briginshaw*.[7] 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 that they caused or contributed to the death.

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[6] *Keown v Khan* (1999) 1 VR 69.
[7] (1938) 60 CLR 336.

MATTERS IN WHICH THE CORONER MUST, IF POSSIBLE, MAKE A FINDING

Identity of the Deceased pursuant to section 67(1)(a) of the *Coroners Act 2008*

12. Allison Judith Allan was visually identified by her partner Reno Grima on 4 November 2015. Identity was not in issue and required no further investigation.

Medical cause of death pursuant to section 67(1)(b) of the *Coroners Act 2008*

13. On 9 November 2015, Dr Victoria Francis, Forensic Pathologist at the Victorian Institute of Forensic Medicine, conducted an examination on Allison's body and provided written report dated 4 March 2016, concluding a reasonable cause of death to be "I(a) Hypoxic ischaemic brain injury I(b) Alcohol and sodium valproate toxicity". I accept her opinion in relation to the cause of death.

14. Toxicological analysis of the ante mortem specimens detected ethanol (0.06 g/100mL), valproic acid (~300 mg/L), morphine free (~0.03 mg/L), midazolam (~0.02 mg/L), the metabolite of venlafaxine, desmethylvenlafaxine in the ante mortem blood (~0.5 mg/L) and in post mortem blood (~0.03 mg/L)

15. Dr Francis noted the post mortem examination revealed hypoxic ischaemic encephalopathy. There were focal acute inflammatory changes within the lungs. There was a moderate degree of coronary artery atherosclerosis (up to 60% stenosis) which a significant degree in someone of this age group

Circumstances in which the death occurred pursuant to section 67(1)(c) of the *Coroners Act 2008*

16. During the afternoon on 29 October 2015, Allison was at home with Reno and her brother, drinking alcohol and putting up decorations. Reno stated that she appeared happy. At approximately 4.00pm Allison said she had a bit of a headache. Consequently, she had a short nap and then went to visit her neighbours and returned approximately half an hour later. Sometime between 5.30pm and 6.30pm, Alison said she was tired and wanted to go to bed. Reno heard tablets "popping" from the kitchen. Reno stated that Alison normally took her medications before bed and he would hear the "popping noise". He went into the kitchen and saw open medications on the bench, Reno went to the bedroom and asked Allison how many tablets she had taken. Allison replied that she had taken her epileptic

tablets. Reno called for an ambulance at 7.51pm and advised that he estimated Allison had consumed 750ml of vodka and 30 x 500mg tablets (15 grams) of sodium valproate.

17. Ambulance paramedics arrived shortly afterwards, and Allison was assessed to have a Glasgow Coma Score (**GCS**) of 15[8] and, except for a slightly elevated heart rate, her vital signs were unremarkable. Allison was transported to Portland District Health (**PDH**) Urgent Care Centre, arriving at 8.27pm on 29 October 2015. The Associate Nurse Unit Manager (nurse) triaged Allison, noting that she was drowsy but conversing, and was advised she had taken a medication overdose and consumed alcohol because she wanted to die. Allison confirmed to the nurse that she believed she had taken as many as all the sodium valproate tablets from the empty foil medication strips found by Reno, equating to 15 grams. The nurse subsequently telephoned the Victorian Poisons Information Centre (**VPIC**), documenting advice she received regarding medical management for sodium valproate overdose and alcohol consumption.

18. From 8.35pm to 10.00pm, half hourly neurological observations recorded that Allison was maintaining a GCS of 15. Allison's alcohol breathalyser reading performed at 8.45pm recorded a level of 0.248 grams/decalitre[9]. Nursing shift change and handover occurred between 10.15pm and 10.30pm. Allison was first documented on the observation chart to be "sleeping" at 10.30pm.

19. At 10.41pm, a doctor documented that she had attempted to speak to Allison, but she was in a deep sleep. With ongoing unremarkable vital signs, she documented that "as per discussion of nurse in charge and poisons centre" the amount of sodium valproate ingested was not a harmful dose, and so blood tests were not required. The medical management plan was to observe Allison for 18 hours with a psychiatry review the following day. The doctor added that Allison "can be very drowsy due to combination of drug and alcohol".

20. The next recorded observations at 12.25am on 30 October 2015 documented that Allison remained asleep and was mildly hypotensive[10]. At 12.45am, Allison's heart rate had increased to 126 beats per minute, her oxygen saturation level had declined to 90 per cent,

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[8] An objective scale of neurological assessment, ranging from three (deep unconsciousness) to fifteen (no impairment). A score of less than eight being commonly accepted as the level of conscious state impairment in which a person is likely to be unable to protect their airway from saliva and other secretions and is at risk of obstructing their airway.

[9] The Australian legal limit for blood alcohol for fully licensed car drivers is 0.05 grams/decalitre or per cent.

[10] Low blood pressure.

and her GCS was recorded to be significantly lower at eight[11]. Supplementary oxygen via nasal prongs was administered effectively.

21. Allison's vital signs then remained unremarkable throughout the remainder of the night, with the exception of an elevated heart rate of 95-115 beats per minute. From 2.00am, Allison's GCS was recorded as seven. Routine blood tests (plus a valproate level) were collected at 2.15am on 30 October 2015 and received for processing at St John of God pathology service at 7.55am. The valproate level was confirmed to be significantly elevated at 3781 micromole/litre[12], as was the ammonia level of 791 micromole/L[13]. The other routine blood test results were unremarkable. At 7.30am Allison was recorded to have a (lowest possible) GCS of three, and her previously normal response pupils were recorded as sluggish or not reacting to light[14].

22. A Computed Tomography (**CT**) brain scan revealed diffuse cerebral oedema[15], a finding commonly associated with severe valproate toxicity. Some cerebellar tonsillar herniation[16] was also identified but suspected to be congenital. There was no evidence of intra-cerebral haemorrhage. Following the CT scan Allison was sedated and intubated[17] at approximately 8.50am on 30 October 2015 to commence mechanical ventilation in response to her poor conscious state. Unremarkable blood gas test results, including a pH of 7.36[18], shortly before the intubation confirmed that Allison had remained in a stable condition.

23. Later that morning, Allison was transferred via air ambulance to Warrnambool Base Hospital (**WBH**) for ongoing management, arriving at the Intensive Care Unit (**ICU**) at approximately 11.35am with vital signs within normal limits other than the persistent mild tachycardia[19]. Allison developed further signs of valproate toxicity over the afternoon, including worsening lactic acidosis[20] (lactate 9.2mmol/L[21], pH 7.22, $PaCO_2$ 37mmHg[22],

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[11] Nil eye opening (1); incomprehensible verbal response (2); localising to pain (5).
[12] St John of God Pathology valproate reference range: 350-700 micromole/litre.
[13] A metabolic consequence of sodium valproate toxicity, excess ammonia can result in altered conscious state. Ammonia normal reference range: < 50 micromole/L.
[14] Both pupils should be the same shape, size and react equally to light. Although not part of the Glasgow Coma Scale, examination of the pupils is an essential adjunct to it, especially when the patient's level of consciousness is impaired. Changes to pupil size or function are associated with several medical conditions, including acute brain injury.
[15] Widespread brain swelling.
[16] A common congenital anomaly whereby the hindbrain (cerebellum) extends down toward the brainstem and spinal cord. Cerebellar tonsillar herniation can also occur in the setting of head trauma due to raised intracranial pressure secondary to a cerebral haematoma or haemorrhage, resulting in pressure on the brain stem and spinal cord, and blockage of cerebrospinal fluid flow.
[17] Insertion of an endotracheal plastic tube to create an artificial airway to maintain airway patency and enable mechanical ventilation of the lungs.
[18] pH is a logarithmic measure of hydrogen iron concentration, that is, the acidity or alkalinity of a solution. The normal pH range in human blood is 7.35-7.45.
[19] Elevated heart rate.
[20] An abnormal increase in the acidity of the fluids in the body, caused by an accumulation of lactic acid, which is common in shock states, and is strongly associated with a worsening prognosis.
[21] Lactate reference range: 0.5-2.0mmol/L.
[22] Arterial blood gas partial pressure of carbon dioxide reference range: 35-45mmHg.

$HCO3^-$ 15mmol/L[23]) and cardiovascular instability (hypertension[24], brief episodes of non-sustained ventricular tachycardia[25] and a prolonged QTc interval[26] up to 549 milliseconds).

24. The lactic acidosis and cardiac instability resolved by the morning of 31 October 2015, though Allison then developed severe hypernatremia[27] (up to 163mmol/L), another clinical feature of valproate toxicity, which was effectively treated. Allison's pupils were uneven and non-reactive to light. A repeat CT brain scan revealed left cerebellar, occipital and frontal infarcts[28], though no evidence of cerebral oedema. Continuous renal replacement therapy (CRRT)[29] was commenced at 1.20pm on 31 October 2015 to promote valproate and ammonia clearance.

25. On 2 November 2015 a Magnetic Resonance Imaging (**MRI**) brain scan was performed, revealing multiple recent infarcts and evidence of oedema. The MRI report also noted that there was "minor inferior displacement of the cerebellar tonsils likely secondary to the recent infarcts." Allison's condition continued to deteriorate and in the early evening of 3 November 2015, she was assessed and determined to be brain dead. After discussion with her family, mechanical ventilation was discontinued at 9.05pm. Allison was declared deceased at 10.00pm on 3 November 2015.

Review and Assessment of Contributing Factors

Victorian Poisons Information Centre advice

26. VPIC provides wide ranging advice regarding poisons to the state of Victoria. Requests for advice vary from domestic calls to calls from hospitals regarding snake bite or toxic ingestions. The main role of VPIC is to advise whether hospital attendance is required and, based on the knowledge of the pharmacology of the poisons, the likely course and period of observation required. VPIC also have a role in advising whether any decontamination is recommended and whether antidotes are available and recommended. They may provide specific advice in the case of complex problems such as snake bite or ingestions requiring specialist consultation or intensive care management.

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[23] Arterial blood gas bicarbonate reference range: 22-26mmol/L.
[24] Elevated blood pressure.
[25] A potentially lethal cardiac rhythm.
[26] Prolonged cardiac ventricular repolarisation (delayed electrical pathway conduction in the heart) which predisposes to malignant ventricular arrhythmias.
[27] Elevated serum sodium level. Serum sodium reference range: 135-145mmol/L.
[28] Tissue death caused by transient or permanent interruption of blood supply to the affected region.
[29] Renal replacement therapy is a term used to encompass life-supporting treatments for renal failure and includes haemodialysis, peritoneal dialysis and haemofiltration.

27. The advice received in the phone consultation with VPIC documented by the nurse in the PDH Emergency Nursing Record following her triage of Allison on the evening of 29 October 2015 was:

- If patient weighs 70kg (approximate weight) the Epilim dose[30] works out to be 200mg/kg which is not a lot
- Patient may be drowsy
- Patient may be unsteady on her feet
- Patient would have to take twice as much (30g) for there to be any concern
- Not likely to require intubation
- Observe patient until drowsiness / sedation wears off
- Observe overnight and up to 18 hours if required

28. In the VPIC records entry by the call taker/pharmacist at 9pm on 20 October 2015, she noted the estimated 15 grams of sodium valproate and 750ml of vodka reportedly ingested by Allison. The VPIC record also included Allison's weight, age, the time of ingestion, and a description of being "drowsy and drunk". Documentation of the advice provided by the VPIC pharmacist was essentially as detailed above, noting that Allison required "supportive care overnight for 18 hours[31] while she sleeps it off". The VPIC pharmacist also noted that the calculated sodium valproate overdose level was "only just over 200mg/kg" (15,000mg/70kg = 214mg/kg). Allison's post mortem weight was 63kg, which equates to a sodium valproate level of 238mg/kg.

Sodium valproate toxicity and management

29. A statement was provided by VPIC Manager Mr Jeff Robinson. Mr Robinson advised that vodka and sodium valproate cause additive effects on central nervous system (CNS) depression, and confirmed the advice provided by the VPIC pharmacist regarding expected symptoms for Allison was correct. Significant CNS depression (potentially requiring intubation) is expected in cases of sodium valproate overdose where 400mg/kg or more has been ingested.

30. Allison was prescribed the enteric-coated formulation of sodium valproate, which slows release of the medication into the body once it is ingested. Mr Robinson advised that the VPIC pharmacist was aware the sodium valproate was enteric-coated, and her advice to

[30] Amount of Epilim (sodium valproate) ingested.
[31] The pharmacist documented in the VPIC call record that she advised ANUM Taylor to observe Allison for 16 hours, but in a supplied recording of the phone call, The pharmacist actually advised that 18 hours of observation was required.

monitor Allison for 18 hours reflected this understanding. Peak plasma concentrations of enteric-coated sodium valproate occur four to five hours after therapeutic doses, but may be markedly delayed following overdose, occurring typically in the range of 3.5 to 11.3 hours, and up to 18 hours post ingestion.[32] Valproate serum concentrations should be measured when the suspected overdose is greater than 200 mg/kg. Blood samples should be taken on patient presentation, and then repeated at three to four-hour intervals (to identify the onset of peak concentrations and ensure that the serum concentrations are falling).[33]

31. There are no proven antidotes for sodium valproate intoxication. Acute treatment is primarily symptomatic and supportive, focusing mainly on CNS and respiratory depression. Gastrointestinal decontamination with activated charcoal is recommended for a sodium valproate overdose over 400 mg/kg. Naloxone[34] in the setting of CNS depression and L-carnitine[35] in the setting of hyperammonemia[36] have varying reported results in valproate overdose but may be used as adjuncts to standard management. Multiple doses of activated charcoal in addition CRRT may be useful in severe toxicity. Treatment of encephalopathy[37], seizures, hypotension, electrolyte disturbances, thrombocytopenia[38], metabolic acidosis, bone marrow suppression, and hypothermia[39] may sometimes be required following large to massive overdoses. Delayed cerebral oedema may occur, and serum ammonia levels should be checked if encephalopathy is suspected.[40, 41]

VPIC internal review

32. Mr Robinson explained that the medical management advice provided by the VPIC pharmacist to the nurse was mostly correct. However, Mr Robinson explained that serial valproate serum concentrations should be measured in all cases of deliberate self-poisoning, and therefore laboratory investigations should have been recommended by the VPIC pharmacist. Mr Robinson advised that the VPIC pharmacist's decision not to recommend the serial blood tests was based on the history provided by the nurse, who was confident that only a maximum of 30 x 500mg tablets had been ingested. Additionally, further shortcomings identified by Mr Robinson included an absence of detail regarding symptom documentation by the VPIC pharmacist, as well as not advising the nurse to call back if

[32] Sztajnkrycer, M, "Valproic acid poisoning", Up To Date, Version 19.0. Accessed online 4 May 2017.
[33] Sodium valproate, Toxins Poisons Information Database. Accessed online 11 May 2017.
[34] An opioid reversal agent commonly used in the setting of CNS and respiratory depression following an overdose.
[35] An amino acid derivative that is thought to limit production of the toxic metabolites in the setting of a sodium valproate overdose.
[36] Elevated serum ammonia level.
[37] Abnormal brain function.
[38] Low blood platelet count, resulting in predisposition to bleeding.
[39] Low core body temperature.
[40] Sodium valproate, Toxins Poisons Information Database. Accessed online 11 May 2017.
[41] Sztajnkrycer, op. cit.

Allison developed symptoms that did not match the expected benign course. These identified issues were subsequently discussed with the VPIC pharmacist.

33. Mr Robinson advised that the VPIC pharmacist's VPIC shift finished at 9.30pm on 29 October 2015, and her telephone consultation with the nurse was reviewed by Mr Robinson approximately 11.5 hours after the consultation, as all VPIC calls are internally reviewed by a second staff member at the start of the next shift. On this date, the hotline had been diverted to the NSW Poisons Information Centre from 9.30pm, with the next shift commencing at 8am on 30 October 2015. Mr Robinson reviewed the VPIC pharmacist's advice at approximately 8.30am on 30 October 2015 but did not call PDH back as it was then 13.5 hours after the overdose and any drug toxicity "would have well and truly declared by this time". Allison was intubated at 8.50am on 30 October 2015.

34. Mr Robinson noted that due to current VPIC staffing resources, it is not possible to improve or rectify the delayed internal call review times, which can occur up to 17 hours later. Mr Robinson added that there has been no increase in VPIC staffing levels for 15 years, with just one poisons information specialist rostered to work evenings, weekends and overnight, and no designated breaks. The VPIC workload and call complexity has increased substantially in recent years, which can result in incorrect or inadequate advice due to increased work pressure and fatigue. Mr Robinson explained that the VPIC pharmacist's shift on 29 October 2015 was very busy, with 63 calls in 6.5 hours including 10 complex calls from hospitals (a typical VPIC evening shift would be 55 calls including one or two from hospitals). In the two hours prior to The VPIC pharmacist's phone consultation with the nurse, the VPIC pharmacist had answered 29 calls, which Mr Robinson described as "an extraordinarily high number".

35. The VPIC is currently preparing a request to the Victorian Department of Health and Human Services for additional funding for extra staff.

Portland District Health Urgent Care Centre medical and nursing management

36. A joint statement from PDH was provided by Director of Medical Services Cathy Bones, Quality Manager Loren Drought, and Director of Quality Services Ros Jones. The joint statement advised that PDH does not have a policy specific to management of sodium valproate overdose but does has a general policy on poisons substance abuse, which instructs staff to contact VPIC for advice.

37. Frequent neurological observations (half hourly to hourly) were performed on Allison from her arrival at PDH until the following morning when she was intubated, except for the period between the observations performed at 10pm on 29 October 2015 and 12.45am on 30 October 2015. Observations performed in this period at 10.30pm and 12.25am simply noted that Allison was sleeping. A doctor also noted at 10.41pm that Allison was in a deep sleep following her assessment. The PDH joint statement confirmed that neurological observations should have been continued during this period.

38. At 12.45am on 30 October 2015 following Allison's documented significant decline in GCS as well as her tachycardia, hypotension and oxygen desaturation, the nurse immediately reported these changes and Allison was reviewed by a doctor. Supplemental oxygen therapy and intravenous fluids were commenced, which resolved all the clinical concerns other than the expected low conscious state, as per the VPIC advice.

39. The PDH joint statement explained that Allison's deteriorating condition was not initially escalated to the on-call physician, who was contacted at 7.15am on 30 October 2015 around the time of Allison's further neurological decline. The on-call physician requested that blood tests be performed and to contact the anaesthetist for intubation. Allison was intubated following her CT brain scan and was subsequently transferred to WBH. The PDH joint statement advised that Allison was not intubated earlier (for example, when her GCS was first noted to have declined to seven), as the doctor and nurse-in-charge considered that alcohol was the main cause for the drowsiness, and symptoms were in keeping with the advice provided by VPIC. The anaesthetist escorted Allison to her CT scan to monitor her airway, which remained patent throughout the night. Except for the brief period of moderate tachycardia, mild hypotension and mild oxygen desaturation around 12.45am, regular assessments overnight confirmed that Allison's vital signs, including her respiratory rate and oxygen saturations, were otherwise within normal limits.

PDH internal review

40. A case review of the medical and nursing management of Allison was undertaken at PDH on 7 February 2017. The PDH case review identified the following issues in patient management:

- No confirmation of accurate patient weight;
- No confirmation of accuracy of drugs ingested;

- The PDH doctor did not speak directly to the VPIC;
- The night shift doctor was given reassurance regarding the advice from VPIC by the nurse-in-charge and the evening shift doctor. The evening shift doctor had not taken a history from Allison;
- The intubation of Allison should have occurred much earlier;
- The on-call physician was not made aware of Allison;
- The PDH Neurological Observations policy was not adhered to;
- Patient allocation between junior and senior Urgent Care Centre nursing staff was inequitable;
- Recognising and responding to the deteriorating patient did not occur in a timely manner by the Urgent Care Centre nurse-in-charge and doctor;
- It was assumed that alcohol was the cause of Allison's drowsiness;
- The PDH process of escalation was not initiated in a timely manner by the Urgent Care Centre nurse-in-charge or doctor; and
- This case was not reviewed earlier at PDH, as at the time of Allison's hospitalisation the Mortality and Transfer Committee did not review cases of non-admitted patients who were transferred to another health service.

41. The PDH recommendations for improvement following their internal review were:

- The Urgent Care Centre doctor is to now make the initial call to VPIC;
- Overdose patients are to be either admitted to the PDH Short Stay Unit or a ward rather than remaining in Urgent Care Centre for monitoring. Patient admission requires a mandatory consultation with the on-call physician;
- The PDH neurological observations policy will be reviewed and amended, to include:
    (a) mandatory reporting of patients with a GCS less than 10 to the on-call physician
    (b) In patients with a drug overdose, a drug screen pathology test must be performed, and accurate weight must be obtained and recorded

- PDH Urgent Care Centre staff education to be actioned regarding:
    (a) managing the unconscious patient
    (b) patient care escalation processes
    (c) PDH Adverse Event (recognition and response) policy
- The following PDH policies and processes will be reviewed:
    (a) The Recognising and Responding to the Deteriorating Patient policy
    (b) Bedside handover process for unconscious or critically ill patients
    (c) Handover times and processes

42. The PDH case review process has now changed, with the Mortality and Transfer Committee reviewing all patients who were transferred from PDH, regardless of whether or not they were admitted to PDH.

Victorian Poisons Information Centre

43. I reviewed the call recording to VPIC from the nurse at PDH, and it is clear that the VPIC pharmacist was under pressure. The VPIC pharmacist expressed that she had been extremely busy with a succession of complex calls, but then advised she had composed herself and was ready for the consultation.

44. Allison was standing in front of the nurse while she made the call to VPIC. The focus of the conversation was on the amount of sodium valproate ingested and the degree of associated toxicity risk, with the significant alcohol ingestion a secondary issue. A breath alcohol concentration measurement had yet to be performed. While the VPIC pharmacist acknowledged the reported quantity of alcohol ingested, it is possible that she may not have appreciated the significance of the amount, as the advice provided does not appear to have raised an appropriate level of concern regarding its co-ingestion along with the sodium valproate. The VPIC pharmacist conveyed that supportive care was all that was required at the level of ingestion reported, and that the alcohol would add to Allison's sleepiness.

45. While Mr Robinson confirmed that the VPIC pharmacist should have recommended serial serum valproate levels during the call, in this night time regional healthcare setting, it is likely that such blood tests would not have been processed overnight. The other incomplete aspects of the VPIC pharmacist's advice was not requesting comprehensive clinical

information, and not advising PDH to call back with any further issues. Had VPIC had normal overnight staffing, with calls not being redirected to the NSW service overnight, it is likely the VPIC pharmacist's advice would have been promptly reviewed, the omissions recognised and a follow up phone call to PDH likely would have occurred, which may have triggered a re-evaluation of Allison's clinical condition, provision of further information to VPIC and subsequent updated advice to the hospital. From review of the PDH medical records and PDH joint statement, it is apparent that the VPIC pharmacist's advice as well as VPIC's inability to review the phone consultation in a timely manner on this occasion influenced the PDH medical and nursing care that followed.

Portland District Health

46. The nurse at PDH and Reno were confident of the amount of sodium valproate ingested by Allison. In the setting of acute valproate toxicity, cerebral oedema generally occurs between 12 hours to four days later, and so there is usually little utility in undertaking a CT brain scan in the immediate post overdose period.[42] However, from 10.30pm, approximately two hours after her arrival at the Urgent Care Centre, Allison's GCS was unknown due to a period of inadequate neurological assessment by PDH nursing staff, who simply documented that Allison was sleeping. Formally reassessed once again at 12.45am on 30 October 2015 with a significantly lower GCS of eight which declined to seven by 2am, this was the earliest opportunity for nursing staff to report this change to the medical staff, who should have considered intubation and whether a CT brain scan was required at that stage.

47. The advice from VPIC predicting that Allison's clinical course of symptoms and management following her overdose required observation only was excessively relied upon by the PDH medical and nursing staff. It is likely that the PDH staff were reassured by the information provided by VPIC, and consequently their usual professionalism and vigilance were diminished. Despite the issue of the slightly suboptimal advice provided by VPIC, the onus for evaluation (and re-evaluation) and management of the patient lies with the treating clinicians.

48. It is reasonable to assume that the delayed availability of blood test results made the assessment of the severity of Allison's presentation more difficult. Whilst management of sodium valproate overdose is managed largely on the clinical presentation, serum levels may increase the level of clinician concern depending on their absolute and serial values.

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[42] Sztajnkrycer, M, op. cit.

49. Of the array of deficiencies in staff practices identified by PDH following their internal review, I consider the most significant the inadequate neurological observations, failure to recognise clinical deterioration, delayed escalation to senior staff, delayed intubation,in conjunction with an apparent underappreciation of the significance of the alcohol co-ingestion, and ability of alcohol to potentiate the CNS depression caused by valproate, and failure by PDH staff to re-evaluate the advice given by VPIC when Allison's condition declined.

50. Absent speculation, I am unable to find all of any of the above deficiencies were causative. However, I am satisfied they potentially amounted to a missed opportunity to avert the tragic outcome.

Warrnambool Base Hospital

51. In his coronial brief statement, WBH ICU Consultant Physician Mr John Hounsell explained that management of Allison was repeatedly discussed with the ICU Consultant at St Vincent's Hospital Melbourne, as is the usual practice for complex cases. WBH ICU did not, however, consult with a clinical toxicologist at St Vincent's Hospital ICU, nor did they consult with VPIC. According to Austin Clinical Toxicology Service guidelines for elimination of sodium valproate (and ammonia) from the body, haemodialysis (or CRRT) should be considered in the presence of cardiovascular instability, cerebral oedema or significant metabolic acidosis (pH < 7.1)[43].

52. On 31 October 2015 at 11.20am, a WBH ICU doctor documented that after the repeat CT brain scan performed that morning revealed multiple infarcts but no cerebral oedema, it was "unclear at this point what Allison's recoverable neurological function is but [there is] no concrete evidence at this stage of irreversible damage". In light of Allison's cardiovascular instability and known cerebral oedema on 30 October 2015, it is possible, though not definitive, that early commencement of CRRT may have improved her outcome. CRRT was eventually commenced over 24 hours after Allison's transfer to WBH.

[43] Sodium valproate, Victorian Poisons Information Centre / Austin Clinical Toxicology Service Guidelines, Austin Health. Accessed online 23 May 2017.

Expert opinion – Clinical Toxicologist Dr Betty Chan

53. I asked Clinical Toxicologist Dr Betty Chan[44] to provide an expert opinion statement in response to questions about valproate overdose and its management. The key points from Dr Chan's statement are summarised below.

Valproate overdose

54. In patients with a valproate overdose of less than 400mg/kg, the effects are usually fairly benign.[45] While the concurrent excessive ethanol consumption of 0.25g/dL could have exacerbated drowsiness, it could also mislead clinicians to blame the ethanol as the cause of a coma. The fact that Allison deteriorated with time should be attributed to valproate overdose and not ethanol.

55. Allison's highest recorded serum valproate level was 3781 micromole/litre which is moderately elevated, and does not usually warrant any active treatment if otherwise well clinically. However, she developed valproate-induced hyperammonaemic encephalopathy (VHE), which is an uncommon but serious complication. The main clinical features are drowsiness and coma, followed by respiratory failure and then multi-organ failure.

Victorian Poisons Information Centre

56. In addition to the shortcomings already identified regarding the phone consultation advice provided by the Victorian Poisons Information Centre (VPIC) to Portland District Health (PDH), one further deficiency related to the expected symptom of non-specific drowsiness. While alcohol co-ingestion with valproate overdose can exacerbate drowsiness, as Allison was drowsy but had a Glasgow Coma Score of 15[46] on presentation to the Urgent Care Centre at Portland District Health, her level of alertness should have improved within two to four hours rather than deteriorate with time.

Portland District Health

57. The main deficiency in the care provided at PDH was that Allison's clear clinical deterioration in conscious state from 2.00am on 30 October 2015 did not result in the treating doctor notifying the on-call consultant nor seeking further advice from VPIC.

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[44] Dr Chan is an emergency physician and clinical toxicologist who is the Head of Clinical Toxicology at Prince of Wales Hospital, Randwick, New South Wales, and works as a toxicologist for the NSW Poisons Information Centre.

[45] Based on post mortem weight and the reported quantity ingested, Allison's valproate overdose equated to 238mg/kg.

[46] An objective scale of neurological assessment, ranging from three (deep unconsciousness) to fifteen (no impairment). A score of less than eight being commonly accepted as the level of conscious state impairment in which a person is likely to be unable to protect their airway from saliva and other secretions and is at risk of obstructing their airway.

Ideally at this time, Allison should have been intubated[47], a CT brain scan performed, and rapid transfer to a regional hospital arranged.

58. Regarding the recommendations and practice changes implemented by PDH following their internal review, Dr Chan noted that regardless of where a patient is located in the hospital, with any sudden and significant deterioration in clinical status, the on-call consultant should be notified. Additionally, it is acceptable for a nurse to initiate a call to VPIC, but the treating doctor should consult VPIC to confirm the advice and make subsequent contact with VPIC to discuss treatment if there is a deterioration in consciousness of clinical state.

Warrnambool Base Hospital

59. It is not possible to determine whether Allison's brain injury from cerebral oedema secondary to valproate and hyperammonaemia was reversible by the time she arrived at Warrnambool Base Hospital (WBH). However, the diagnosis of cerebral oedema was clear on the CT brain scan performed earlier in the day at PDH prior to the transfer. Therefore, every attempt should be made to manage the cerebral oedema. Current recommendations are for urgent intermittent haemodialysis or continuous veno-venous haemodiafiltration (referred to as continuous renal replacement therapy, or CRRT). Both modes of dialysis can also clear high levels of ammonia and resolve acidosis. Dialysis treatment should have been commenced as soon as Allison arrived at the Intensive Care Unit (ICU) at approximately 11.35am on 30 October 2015. Dialysis was actually commenced at approximately 1.20pm on 31 October 2015.Treatment with carnitine[48] for management of the hyperammonaemia would also have been appropriate, but the evidence for its efficacy is not strong.

60. Advice from a toxicologist should have been obtained upon Allison's admission to the ICU. Despite the progress notes of Allison's medical admission to the ICU noting her fixed and dilated pupils[49], there was no mention of the CT brain scan performed at PDH earlier that morning nor the finding of generalised cerebral oedema[50]. It is unclear if WBH doctors knew of the CT brain scan result performed at PDH hours before Allison's transfer.

61. There were multiple medical progress note entries regarding the management of Allison, including references to her worsening lactic acidosis and high ammonia level, both of which should have been further triggers for the need for urgent dialysis. However, consultations with St Vincent's Hospital Melbourne ICU and the senior medical unit doctor at WBH did

[47] Insertion of a tube through the mouth or the nose and into a patient's trachea to maintain a patent airway and assist with ventilation.
[48] An amino acid derivative that may reduce the side effects of valproate overdose.
[49] A commonly identified endpoint in medical assessment to signify hypoxic (inadequate oxygen) brain injury.
[50] Brain swelling.

not result in any recommendations for immediate alterations to the treatment provided until the day after Allison's admission to WBH. The delay to commencement of dialysis at WBH was significant, as earlier initiation of this therapy could have changed the outcome in this case.

62. I accept Dr Chan's conclusion, that while one cannot be certain, Allison's death may have been prevented if she had been provided early airway protection (intubation), hyperventilation, careful fluid balancing, early advice from a toxicologist and, most importantly, early institution of dialysis upon arrival at WBH.

Additional statement from Warrnambool Base Hospital

63. After receiving Dr Chan's expert opinion statement, I requested an additional statement from the treating medical team at WBH. A statement was provided by WBH Consultant Physician and Haematologist Dr John Hounsell, with the key points summarised below.

64. WBH Intensive Care Unit (**ICU**) is a regional ICU run by the on-call specialist general physician, who is also responsible for the general hospital and all medical admissions. There are no dedicated ICU consultants, with junior medical registrars the resident staff in the ICU. For medical management advice, WBH on-call specialist general physicians rely on their individual network of expert colleagues, with most having a strong affiliation with St Vincent's Hospital, where they undertook their training. While there are no formal arrangements regarding this process, the St Vincent's Hospital on-call ICU consultants are always very willing to assist WBH on-call physicians with medical management advice.

65. When accepting transfer of Allison to WBH ICU from PDH the Emergency Consultant was made aware of the VPIC advice that with a modest valproate level and high ethanol level, supportive care was recommended, with valproate-specific complications unlikely. Neither VPIC nor the Emergency Consultant suggested the possibility of urgent dialysis or the need to measure ammonia levels, and VHE was not mentioned at any time. Consequently, the focus of the medical management at WBH was ventilatory support and fluid / electrolyte management.

66. Dr Hounsell only became aware of VHE as a rare complication of valproate overdose after his own research on the day of Allison's admission. Had he known of this complication before the transfer, Dr Hounsell advised that he would never have accepted a patient who might need urgent dialysis. The ICU did not have the capacity for continuous veno-venous

haemodialysis at the time of Allison's admission, as the only two physicians at WBH with such training were not available that day.

67. In a phone discussion with the St Vincent's Hospital on-call ICU consultant on the evening of 30 October 2015, Dr Hounsell raised the treatment option of dialysis, but was advised to continue supportive care and consider dialysis if the acidosis worsened. The following morning, care was handed over to a general and renal physician, who also consulted the St Vincent's Hospital on-call ICU consultant and was advised to undertake a repeat CT brain scan and then commence dialysis.

68. Dr Hounsell accepted Dr Chan's expert opinion of the medical management in this case but highlighted that WBH medical staff believed they were acting on expert advice for a rare condition with which Dr Hounsell had no professional experience. Dr Hounsell advised that while it is unclear from review of the medical records if WBH medical staff were aware of the CT brain scan result performed at PDH on the morning of 30 October 2015, it would be unusual if the result had not been communicated. Dr Hounsell added that a degree of cerebral oedema is a non-specific finding and is also seen in the setting of hypoxic brain injury, which Allison could have sustained. The follow up CT brain scan undertaken at WBH the next day identified multiple infarcts[51] but no cerebral oedema, more in keeping with a hypoxic[52] / hypotensive[53] insult.

69. Dr Hounsell stated that VPIC were not consulted once Allison was admitted to WBH as the advice provided by VPIC was clearly documented in progress notes as well as verbally handed over. Once a patient is in the ICU at WBH, any further medical management advice is referred to an ICU consultant (such as St Vincent's Hospital on-call ICU consultant), who Dr Hounsell considers are experts in toxicology[54] as part of their specialist training. Allison's case was not originally reviewed at a formal WBH morbidity and mortality meeting. Since receiving Dr Chan's report (in January 2018), an education session has been undertaken for all WBH physicians regarding the use of dialysis in the setting of drug overdose, and in particular the signs and management of VHE. And the upskilling of WBH physicians to allow use of continuous veno-venous haemodialysis after hours continues.

[51] Tissue death (necrosis) caused by an obstruction of the tissue's blood supply.
[52] Deficiency in the amount of oxygen reaching body tissues.
[53] Low blood pressure.
[54] The study of the adverse effects of chemicals (poisons) on living organisms.

Conclusions

70. "Death [from valproate toxicity] is rare, and usually results from cardiac or respiratory arrest"[55]. A person who had ingested alcohol and valproate at the levels reportedly ingested by Allison would be expected to survive if they reached medical care, as treatment is mostly supportive, that is, support of the airway and breathing should conscious state deteriorate. The amount of sodium valproate ingested in Allison's case would not normally be expected to require intervention, however the ingestion of a large amount of alcohol complicated the presentation and compounded the CNS depressant effects of the valproate. Furthermore, cerebral oedema associated with acute valproate toxicity is not clearly correlated with the size of the dose ingested.[56]

71. It remains unclear whether Allison's hypoxia ischaemic brain injury was caused either directly by the valproate toxicity; due to potential apnoea[57] / airway occlusion as a result of the respiratory and conscious state impairment due to the effects of the excess serum valproate, ammonia and alcohol levels; or from the combined effects of both. Valproate toxicity can result in cerebral oedema and lead to a hypoxia ischaemic brain injury, and likewise, inadequate respiration can result in a hypoxic brain injury which can lead to cerebral oedema.

72. With the exception of the temporary mildly decreased oxygen saturation level of 90 per cent recorded at 12.45am on 30 October 2015, Allison's documented respiratory rate and oxygen saturation levels were unremarkable and not suggestive of respiratory compromise. Vital signs were generally recorded half hourly to hourly at PDH, however there were no vital signs documented in the following periods: between 10.30pm on 29 October 2015 and 12.25am on 30 October 2015 when the GCS was also not recorded as Allison was "sleeping"; and from 6.30am until 8.55am on 30 October 2015, during which time Allison's GCS declined to three (Allison was intubated at approximately 8.50am on 30 October 2015). In the absence of continuous monitoring of Allison's oxygen saturation and respiratory rate at PDH, it is possible that Allison experienced respiratory compromise in between the times of the unremarkable vital signs recorded.

73. Allison's calculated sodium valproate toxicity based on the reported amount ingested and her estimated weight was calculated to be 214mg/kg. The corrected estimate based on the post-mortem weight was 238mg/kg. Sodium valproate overdose that results in moderate

[55] Sodium valproate, Toxinz Poisons Information Database. Accessed online 18 May 2017.
[56] Sztajnkrycer, op. cit.
[57] Temporary cessation of breathing.

CNS depression is known to occur in patients who ingest 200 – 400mg/kg.[58] The table Allison's highest recorded serum valproate level was 545mg/L approximately eight hours post ingestion, well above the therapeutic range of 40-100mg/L, and indicative of moderate toxicity (coma +/- other organ effects). It is not possible to determine if this highest recorded serum valproate level of Allison's was a peak, on the rise, or falling, with the subsequent valproate level result of 300mg/L (performed by VIFM) taken from blood collected approximately 10 hours after the earlier result, and approximately 18 hours post ingestion. Furthermore, the possibility that Allison ingested more than the reported 15g of sodium valproate cannot be completely discounted.

74. The deficiencies in practice at VPIC and PDH were well identified in the internal health service reviews detailed in the statements provided. Further analysis of the medical, nursing and allied health management provided to Allison following her valproate and alcohol overdose is provided below.

75. While the existing working relationship between the WBH general physicians and the on-call specialist ICU consultants at St Vincent's Hospital is an important one, it is also important that both parties recognise there are limitations to the clinical expertise of an ICU consultant. Clinical toxicology is a medical sub-specialty, requiring completion of a two year masters degree, two year fellowship, management of over 300 peer-reviewed cases and then ongoing employment in a clinical toxicology unit or poisons information service. As such, medical practitioners managing overdose patients should seek assistance from an appropriate specialist or specialty service, unless they are thoroughly familiar with all aspects of care required.

76. VPIC staff are pharmacists trained in risk assessment who provide advice assisted by Austin Hospital Clinical Toxicology Service Guidelines. In complicated and/or severe poisoning cases, VPIC staff have 24-hour access to the Austin Hospital toxicology registrar and on-call toxicology consultants. A range of external consultants (clinical pharmacologists, mycologists, botanists and toxicologists) are also available to provide specialist advice if needed[59].

77. Treating doctors at PDH and WBH inappropriately continued to rely on the initial advice provided by VPIC at the time of Allison's presentation to hospital following her overdose, despite significant patient changes and clinical deterioration over the subsequent hours and

[58] Sodium valproate, Victorian Poisons Information Centre / Austin Clinical Toxicology Service Guidelines, Austin Health. Accessed online 23 May 2017.
[59] "Calling the VPIC", Austin Health. <http://www.austin.org.au/page?ID=523>. Accessed online 18 April 2018.

days. Advice by the VPIC pharmacist was mostly appropriate and based on the clinical scenario in the early hours following Allison's overdose. While the VPIC pharmacist failed to advise PDH triage nurse Ms Taylor that treating staff should call VPIC back if Allison's symptoms deviated from the expected benign recovery pathway, PDH and WBH staff should also have taken the initiative in recognising that further assistance from the VPIC service was required as she deteriorated.

FINDINGS

78. Having investigated the death of Allison Judith Allan and having considered all of the available evidence, I am satisfied that no further investigation is required.

79. On the basis of the evidence, I am unable to make a determination whether Allison had the capacity to form the intention to end her life and therefore make no finding as to intention.

80. I make the following findings, pursuant to section 67(1) of the *Coroners Act 2008*:

    (a) that the identity of the deceased was Allison Judith Allan, born 26 June 1972;

    (b) that Allison Judith Allan died on 3 November 2015, at Warrnambool Base Hospital 25 Ryot Street, Warrnambool, Victoria from hypoxic ischaemic brain injury resulting from alcohol and sodium valproate toxicity; and

    (c) that the death occurred in the circumstances described in the paragraphs above.

RECOMMENDATIONS

81. Pursuant to section 72(2) of the ***Coroners Act 2008***, I make the following recommendations connected with the death:

    1. Portland District Health (**PDH**) implement an operational change in their Urgent Care Centre in response to their review of Allison's death, whereby medical staff must make initial contact with Victorian Poisons Information Centre (**VPIC**). I recommend that this recent change in practice be reviewed by PDH, as unless a doctor is available to consult with VPIC at the time of the patient presentation, it would be appropriate for the triage nurse to still seek early toxicological advice. The advice provided by VPIC may impact the allocated Australasian Triage Scale[60]

[60] The role of the Australasian Triage Scale is as a clinical tool for ensuring that patients are seen in a timely manner, commensurate with their clinical urgency.

category as well as preliminary assessments and investigations prior to medical review. The treating doctor and/or medical team should re-contact VPIC for advice on patient management once they are available, and if appropriate, should also re-contact VPIC with further detailed information to confirm their management is correct.

2. PDH should review their current arrangements with existing pathology service/s, in relation to their capacity to provide optimal care for after-hours patient presentations to their Urgent Care Centre.

3. PDH should enter into an agreement with the nearest health service that provides a higher level of care, to develop a memorandum of understanding (MOU) regarding acute management of patients, such as toxicology patients, who have the potential to deteriorate post presentation. The purpose of the MOU – if such an arrangement does not already exist – would be to detail appropriate escalation of care in such circumstances, if required. Consultation between health services should be a phone conversation between consultant physicians, removing the reliance on junior doctors to appropriately refer or accept patient transfers.

4. Both PDH and Warrnambool Base Hospital (**WBH**) provide internal education to relevant medical staff regarding the importance of:

   (a) having a low threshold for utilising the VPIC service as a primary resource for the medical management of patients who present following overdose, envenomation or poisons exposure, when the treating practitioner does not have a clinical toxicology background or strong understanding of the expected clinical features and management required.

   (b) Treating practitioners re-consulting the VPIC service for any ongoing medical management concerns in such patients, or when the expected clinical course is not followed.

   (c) Where applicable, include reference to the above recommendations in relevant health service policy or guidelines.

5. St Vincent's Hospital Melbourne on-call ICU consultants should, as part of their informal arrangement of providing advice to WBH general physicians, recommend utilisation of the VPIC service as a primary resource for the medical management of drug overdose, envenomation or poisons exposure patients.

6. The Department of Health and Human Services address the staffing shortage at the VPIC referred to in this finding, in particular paragraph 34, through the provision of additional funding.

81. I convey my sincerest sympathy to Allison's family and friends.

82. Pursuant to section 73(1) of the *Coroners Act 2008,* I order that this Finding be published on the internet.

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

(a) Allison's family, senior next of kin;

(b) Investigating Member, Victoria Police; and

(c) Interested Parties.

Signature:

MR JOHN OLLE
CORONER
Date: 30 July 2019