CORONER'S COURT OF THE AUSTRALIAN CAPITAL TERRITORY

Matter Title:	**Inquest into the death of Jessica Hilary Bovill**
Citation:	**[2025] ACTCD 1**
Decision Date:	26 February 2025
Before:	Coroner Archer
Findings:	See [89]-[91], [94]-[95], [98]-[102]
Catchwords:	**CORONIAL LAW** – manner and cause of death – severe diffuse hypoxic brain injury – severe hypernatremia and metabolic alkalosis caused by treatment errors – treatment of amitriptyline overdose – failure to consult official toxicology guidelines – improper dosage monitoring – excessive sodium load effects – unrestrained access to sodium bicarbonate
Legislation Cited:	*Coroners Act 1997* (ACT) ss 3BA, 13, 34A, 52, 55, 57
Case Cited:	*R v Doogan; Ex Parte Lucas Smith & Ors* [2005] ACTSC 74
File Number:	CD 334 of 2020

CORONER ARCHER:

1. Jessica Hilary Bovill died at the Canberra Hospital ("TCH") on 11 December 2020.[1] I will, with respect, refer to her as Jessica in these findings.

2. At about 0600 hours on 8 December 2020, Jessica's husband, William Bovill, found her unconscious on the kitchen floor at their home. I will refer to William as Bill in these findings. Jessica had consumed amounts of amitriptyline, a Tricyclic anti-depressant ("TCA"), in overdose quantities. An ambulance was called, and she was admitted to the Emergency Department ("ED") at TCH. During the treatment for that overdose, she was administered sodium bicarbonate in doses that were far in excess of therapeutic requirements. As a result, she suffered brain swelling and consequential neurological injury, which was eventually consistent with brain death. Organ donation was arranged for the following days.

3. In the reasons that follow, I find that an error in sodium bicarbonate dosage caused Jessica's death.

4. Jessica was 43 years old at the time of her death. She is survived by her husband William ("Bill") and her three children.

PART 1 - PROCEDURAL HISTORY

5. Jessica's death was referred to the Coroner on the day of her death, pursuant to s 13(1)(a) of the *Coroners Act 1997* (ACT) ("the Act"), as the circumstances of the death were unknown.

6. At the direction of Coroner James Stewart, a post-mortem examination was conducted by Professor Johan Duflou, forensic pathologist. The examination consisted of an external examination, a toxicological analysis of Jessica's ante-mortem bloods, and a review of the medical records of Jessica's admission and generally. The results of Professor Duflou's examination are addressed below.

Required findings

7. As Jessica's death fell within the terms of s 13(1)(a) of the Act, the Coroner was required to hold an inquest into the manner and cause of Jessica's death and make findings that are required by s 52 of the Act. That section of the Act relevantly provides:

[1] Those responsible for Jessica's care formally determined on that day that there had been an irreversible cessation of all function of her brain. It is likely she had suffered brain death at an earlier time.

52 Coroner's findings

(1) A coroner holding an inquest must find, if possible—

(a) the identity of the deceased; and

(b) when and where the death happened; and

(c) the manner and cause of death; and

(d) in the case of the suspected death of a person—that the person has died.

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(4) The coroner, in the coroner's findings—

(a) must—

(i) state whether a matter of public safety is found to arise in connection with the inquest or inquiry; and

(ii) if a matter of public safety is found to arise—comment on the matter.

8. Coroner Stewart had carriage of the investigation after Jessica's death was referred to the Court. As no findings had been made by the time of my appointment as the Territory's dedicated coroner in March 2022, responsibility fell to me to complete the investigation into Jessica's death and to make the findings required by s 52 of the Act.

Evidence

9. A police coronial report was prepared. Professor Duflou provided an autopsy report. Bill provided a series of documents, including a detailed summary of the events surrounding Jessica's death, her physical and mental health history, and the work-related injuries she had sustained. That document was forwarded to the Court on 24 March 2021. Bill also provided commentary on some of the expert reports the Court had received. Relevant work injury documents were obtained from insurers.

10. The Court obtained two expert reports, from:

(a) Professor Alison Jones, specialist physician and clinical toxicologist; and

(b) Professor Olaf Drummer, forensic pharmacologist, and toxicologist.

11. Through the ACT Government Solicitor's Office ("ACTGS"), a statement dated 4 May 2023 was received from Dr TX, an ED specialist. Dr TX was the clinician responsible for prescribing medications to Jessica upon her admission to TCH (including the administration of sodium bicarbonate). ACTGS also provided information about the operation of the Toxicology Hotline at TCH. Solicitors acting on Bill's behalf in civil proceedings provided a report from Dr George Skowronski, Associate Professor of Critical Care at the St George Clinical School of the University of NSW.

12. Jessica's medical records were also obtained.

Scope of the investigation

13. The investigation I am empowered to conduct in relation to Jessica's death is not without boundaries. As I have noted in recent findings, the concept of "manner and cause" of

someone's death is not capable of precise definition. It does not permit an investigation that deals with all matters having a tangential connection to a death.[2] Concepts of "remoteness" must be borne in mind when matters that properly contextualise the cause of death are considered.

14. Similar considerations also apply in relation to s 3BA of the Act. Pursuant to that provision, I have a responsibility to ensure that the objects of the Act are carried out in a way that recognises that the family and friends of a deceased person have an interest in having all reasonable questions about the circumstances of that person's death answered. However, that responsibility does not set aside the limitations s 52 of the Act places on my power to investigate.

15. In this case, it is clear that Jessica had faced challenges arising from the difficulties she experienced in a number of her workplaces and the way in which Comcare was dealing with those issues. Undoubtedly, the physical and psychological sequalae of those challenges was a factor in the decision Jessica took to commit the act of self-harm that caused her to be taken to TCH. However, other than to establish necessary context for that decision, it is beyond the scope of this inquest to investigate that workplace injury history in any significant detail.

Hearing

16. I was satisfied, for the purposes of s 34A of the Act, that the manner and cause of Jessica's death were sufficiently disclosed, and, therefore, a hearing was unnecessary. My decision not to conduct a hearing and the reasons for that decision were provided to Bill in writing when provisional findings were sent to his solicitors on 13 December 2024. Having arrived at that decision, consistent with my obligations under s 34A(3) of the Act, I also provided the Chief Coroner with a written notice of that decision, including the grounds for it. In the context of the s 55 process (described below), Bill, the Territory, and Dr TX confirmed that a hearing was not required.

Provisional findings & the section 55 process

17. On 13 December 2024, provisional findings were sent to Bill's solicitors and the ACTGS. Those findings contained a comment adverse to a person identifiable from my proposed findings, namely Dr TX and the Territory. Consistent with my obligations under s 55 of the Act, I invited those persons to make a submission in relation to the proposed comment or give me a written statement in relation to it.

[2] See *R v Doogan; Ex Parte Lucas Smith & Ors* [2005] ACTSC 74.

18. By letter dated 16 January 2025, the ACTGS provided a response on behalf of the Territory. The Territory acknowledged and accepted the comments and criticisms made in the provisional findings. A statement from Ms Janet Zagari, Deputy Chief Officer of Canberra Health Services, dated 15 January 2025 was provided to the Court. Pursuant to s 55(3) of the Act, a request was made that I attach to my findings a copy of Ms Zagari's statement. I have acceded to that request but have not included the attachments that were referred to by Ms Zagari.

19. An email was received on behalf of Dr TX. The submission contained in it sought an amendment to what is now paragraph 50 of these findings. It was submitted that:

> Dr TX was unaware that the eTG advice had been updated in August 2020 (approximately 3-4 months prior) to include a limitation to the maximum dosing of sodium bicarbonate.

20. That submission caused the Court to request Therapeutic Guidelines,[3] the company responsible for the production and distribution of the guidelines, to produce the relevant versions of the guidelines that operated in effect before and after August 2020. Hitherto, the two versions of the guideline had not been available. The submission made on Dr TX's behalf stands to be considered in light of the fact that there was in the (pre-August 2020) Guidelines a limitation as to maximum dosing.[4] The terms of the pre-August 2020 Guidelines were forwarded to Dr TX with an invitation to make further submissions. The reply that was received indicated that Dr TX "wishes to withdraw the submission made in relation to the Section 55 adverse comment". I find that Dr TX was at all times unaware that the Guidelines (in either their pre or post August 2020 form) contained maximum dosing limitations and that the initial submissions made on her behalf in respect of the s 55 process did not accurately reflect the terms of the change that occurred to the Guidelines in August 2020, though noting that obtaining the point-in-time version of the Guidelines was difficult.

21. Bill provided a notated version of the provisional findings and a detailed analysis of Dr TX's statement.[5] Minor amendments were made to my findings as a result of those submissions.

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[3] Therapeutic Guidelines is an independent not-for-profit organisation. It develops and publishes the *Therapeutic Guidelines*, which is extensively adopted and used in Australian medical and pharmacy schools, public teaching hospitals, and in community medical and pharmacy practices.
[4] See [52].
[5] Bill maintained, in response to the s 55 notice and throughout the inquest, that Jessica would have survived the amitriptyline overdose. He has consistently maintained that it was the negligence of Dr TX that caused Jessica's death.

PART 2 – JESSICA'S BACKGROUND, MENTAL HEALTH HISTORY, AND ACT OF SELF-HARM

22. Jessica was born in Queensland in 1977. Her parents separated when she was young. She remained with her mother in Queensland. She met Bill during her course of study in molecular biology at the University of Queensland in Toowoomba. Their married life was spent living in Queensland and, from 2004, in South Australia. In 2010, they moved to Canberra. They had three children together, born in 2001, 2004, and 2014.

23. In 2017, Jessica began working at the Commonwealth Scientific and Industrial Research Organisation ("CSIRO"). Her work there was exacting and demanding, involving laboratory work both at the Black Mountain facility and the University of Canberra. As a result of that work, Jessica sustained injury, first reported to CSIRO in February 2019. The injury eventually developed into Chronic Regional Pain Syndrome ("CRPS"). Thereafter, Jessica lived with significant pain and other symptoms, including weakness in her right arm and shaking hands. Her ability to perform duties associated with her work and to undertake everyday tasks was significantly compromised.

24. Jessica also had diabetes, which was managed by medication (insulin) and lifestyle modifications.

25. The history of the management of Jessica's CRPS in various workplaces and her interaction with their insurers is complex. Over time,
    (a) the pain associated with the injury itself;
    (b) issues surrounding how her condition was managed or accommodated in the workplace;
    (c) how the injury was assessed; and
    (d) what treatments would be paid for by her employers and their insurers

    caused Jessica considerable distress. In the months before her act of self-harm, Jessica's mental health deteriorated significantly for a variety of reasons, including pressures associated with the Comcare process.

26. Jessica enjoyed a good therapeutic relationship with her GP. She was continuing to see a psychologist, and referrals were made by her to the Access Mental Health Team. In October, she was visited by the Home Assessment Acute Response team ("HAART"), and a detailed assessment was conducted. Amongst other interventions, including referral to counselling services, HAART sought to assist Jessica with dispensing medications.

27. On 2 November 2020, Bill contacted HAART to discuss practicalities associated with Jessica's medication issues. He asked if he could collect a week's worth of supply from the chemist.

28. On 6 November 2020, a letter was sent to her home address by HAART, advising her that her file was being closed and that she should follow up with her GP.

29. On 11 November 2020, Jessica called HAART "upset and teary". She indicated that she was safe and that the Comcare process was stressful. Her file was not re-opened.

30. On 12 November 2020, Jessica attended a regular session with her psychologist. A program of weekly sessions was arranged. Jessica did not attend, and the psychologist contacted Bill, who indicated that he was taking a month of personal leave to spend time with Jessica at home.

31. The responsibility for medication oversight was taken over by Bill. He re-stocked the pill container of the various medications from a supply that was kept hidden from Jessica eventually in a kitchen cupboard, which he thought Jessica would unlikely open.

32. On 6 December 2020, the family spent time at home together and, amongst other things, put up a Christmas tree.

33. On the following day (7 December 2020), Bill and Jessica talked about going to the hospital. According to Bill, he encouraged Jessica to go to the hospital, but she was reluctant to go.

34. At about 0600 hours on 8 December 2020, Bill woke up and found that Jessica was not in their bed. He went looking for her. He found Jessica unconscious on the kitchen floor. She was breathing but unconscious. Bill later reported to the police that he found on top of the microwave a box of 50 gm amitriptyline tablets. He told police that up to 30 tablets were missing.[6] He was of the view that the remainder of the contents of the medication supply was not touched. If this amount of amitriptyline (30 tablets) was consumed, it would amount to a dose of 1.5 grams or 24 mg/kg.[7]

35. A "000" call was made. Bill commenced cardiac compressions. ACT Fire and Rescue arrived at the scene first. CPR was continued, but stopped shortly after as Jessica was breathing. No shocks were delivered.

[6] In submission in the context of the provisional findings process Bill suggested that the number of amitriptyline pills missing was between 21 and 30.
[7] Professor Drummer, Expert Forensic Report in the matter of Jessica Bovill deceased, dated 24 April 2024, [4.3].

36. The police report records that Bill indicated that apart from the amitriptyline, there would have been the following (unconsumed) medications in the bag:

    (a) 40 tablets of 150mg Venlafaxine (Efexor-XR);

    (b) 15 Lantus Solostar insulin pens;

    (c) 15 tablets of 10 mg Ezetimibe;

    (d) 7 tablets of 300mg Gabapentin (Neurontin);

    (e) 24 tablets of 1000mg Metformin (Metex XR); and

    (f) 21 tablets of 100mg Palexia.

37. Jessica was transported to TCH and arrived at the ED of TCH at 0747 hours.

PART 3 – JESSICA'S MEDICATION REGIME – AMITRIPTYLINE

38. Before her overdose, Jessica was taking a range of medications to address her depression and pain. Those drugs included amitriptyline (Endep), which is commonly used for depression and treatment of neuropathic pain. The prescribed dose was 25 mg tablets, up to three tablets at night before bed.

39. In his expert report, Professor Drummer described amitriptyline – its characteristics, dosages, and effects – in these terms:

> 5.1. Amitriptyline is a tricyclic antidepressant [TCA] that inhibits the neuronal reuptake of noradrenaline and serotonin. It also has anti-cholinergic activity and affects sodium channels in the myocardium, and in overdose it blocks alpha-adrenoceptors causing hypotension.
>
> 5.2. It is indicated for major depression, panic disorder, neuropathic pain and enuresis.
> …
>
> 5.4. Maximum blood concentrations of the drug after oral ingestion usually occur within 4-6 hours; although ingestion of overdose amounts will prolong absorption due to its anti-cholinergic properties and possibly rate-limiting absorption rate.
> …
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> 5.7. Patients given an oral 150 mg dose daily developed an average amitriptyline and nortriptyline steady-state plasma concentration of about 0.1 mg/L (range 0.04-0.16) and 0.1 mg/L (range 0.02-0.24), respectively.
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> 5.10 Plasma (blood) levels of the drug can be useful to determine the possible extent of poisoning, although there is not a good correlation between plasma concentration and toxicity (including any associated ECG changes), however plasma level monitoring is useful to determine if levels are still rising or falling when patients are in hospital. This can then assist in on-going management of the overdose.
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> …
>
> 5.14. The use of excessive doses can progressively cause anxiety, ataxia, tachycardia, alterations in blood pressure, arrhythmias, seizures, and coma. ECG changes are one of the more symptomatic signs, including prolongation of the QTc and QRS intervals.

> A QRS duration of >100 msec is an indicator of an overdose to drugs like amitriptyline (tricyclic anti-depressants). Ischaemic changes in the CNS or swelling of the brain are not listed as an expected adverse outcome.[8]

40. One of the first diagnostic procedures that was undertaken upon her admission after her overdose was the taking of blood at 0750 hours.

41. At autopsy, the blood taken at 0750 hours was subject to a toxicological analysis. Amitriptyline was found at a level of 1.0 mg/L, Venlafaxine at 0.46 mg/L, and Tapendotal at approximately 0.04 mg/L. According to Professor Drummer, as a general proposition, a level of 1.0 mg/L of amitriptyline was 4 to 10 times above the therapeutic level.

PART 4 – TREATMENT OF THE OVERDOSE

42. The description of the clinical course that followed after Jessica's admission to the ED is derived from the account subsequently given by Dr TX and the clinical records.

43. Dr TX was what was styled as a "D1 Consultant" at the ED. This meant that she was on-call at home prior to the commencement of her shift at 0800 hours on 8 December 2020.

44. At 0733 hours (she was not at TCH), Dr TX received a call from an ED Registrar, who told her that the ACT Ambulance Service ("ACTAS") had alerted the ED to an incoming patient, who, as they understood, had taken a tricyclic anti-depressant overdose. It was unclear how much of the tricyclic anti-depressant she had taken, or when it had been consumed. She was told that Jessica had been assessed as being 8/15 on the Glasgow Coma Scale, which, according to Dr TX, is at the threshold of a patient requiring intubation and mechanical ventilation.

45. Dr TX assessed that Jessica had ingested an overdose of amitriptyline. In her statement, Dr TX indicated that she was "familiar with the principles of TCA overdose",[9] and the last case of TCA overdose she had been involved in was approximately 12 months ago. She said she consulted the "relevant literature"[10] to ensure that there had been "no changes to treatment/management recommendations" since she dealt with a TCA overdose 12 months ago.[11] The literature she consulted online and before arriving at TCH was a publicly accessible website called "LITFL" (Life in the Fast Lane), which, according to Dr

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[8] The QRS or QRS complex is the combination of three of the graphical deflections seen on a typical electrocardiogram (ECG). It corresponds to the depolarization of the right and left ventricles of the heart and contraction of the large ventricular muscles. Ventricular depolarization refers to the time taken for the electrical stimulation and relaxation of the ventricles during a complete cycle of heart contraction and relaxation. In adults, the QRS complex normally lasts 60 to 100 msec: Yasar Sattar and Lovely Chhabra, *Electrocardiogram* (StatPearls Publishing, 5 Jun 2023).
[9] Statement of Dr TX dated 4 May 2023, [17].
[10] Ibid.
[11] However, see paragraphs [50]-[52] and footnote 20.

TX, is "the internet presence of a community of practice of Australasian emergency specialists".[12] Dr TX summarised the advice given on the website in the following terms:

> Relevantly, LITFL advised that in cases of significant TCA overdose, treatment included cardiac monitoring with the administration of doses of intravenous 2 meq / kg of sodium bicarbonate, repeated 3-5 minutely until such time as the ECG QRS complex narrowed (i.e. QRS narrowing was the endpoint of treatment).[13]

46. Dr TX said that advice was "consistent with [her] understanding of the treatment principles of tricyclic overdose". She went on to explain:

> Sodium bicarbonate bottles each contain 100 mls of an 8.4% solution of sodium bicarbonate. Each 1 ml contains 1 mmol bicarbonate or 1 meq bicarbonate. This meant that in the case of a 70 kg adult, each 'dose' would be approximately 1.5 x 100 ml bottles (specifically 150 ml), repeated every 3-5 minutes, until the ECG QRS complex narrowed.[14]

47. Her statement included a screenshot of the relevant page on LITFL. A significant additional patient management issue listed on that page but not referred to by Dr TX was to "hyperventilate to maintain a pH of 7.50-7.55". As it was to be seen, the administration of sodium bicarbonate would normally be associated with an increase in pH levels.

48. Referencing other sources, Dr TX said in her statement:

> In a TCA overdose, if widening of the QRS is > 100m/s, treatment with sodium bicarbonate is recommended. A wider QRS again (above 160ms) indicates an increased risk of lethal ventricular arrhythmias. There are also other QRS abnormalities typically seen in TCA overdose cardiac toxicity.[15]

49. When Dr TX arrived at the ED and assessed Jessica, she noted, amongst other things, the following:
    (a) that the ECG monitoring indicated an abnormally widened QRS;[16]
    (b) "abnormal involuntary movements", "consistent with episodic posturing" (indicating, in her view that "severe brain injury may have already occurred" "by some mechanism");[17]

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[12] Statement of Dr TX, dated 4 May 2023 [17.1].
[13] Ibid [17.3].
[14] Ibid [17.4].
[15] Ibid [17.7].
[16] Ibid [19.7].
[17] Ibid [19.8]. The notes made by Dr TX (prepared in retrospect) refer to shaking consistent with seizures at the time of her initial assessment. The Registrar's notes do not mention seizures at that point of time. Neither ACTAS nor nursing records contain such a reference. Whilst seizures are a common feature of amitriptyline overdoses seizures are also a feature of the disturbance caused by sodium bicarbonate overdose (noting that the administration of sodium bicarbonate began at 0755 hours). The presence of seizures does not necessarily indicate that brain damage has occurred.

(c) a deteriorating GCS level (now 4/15);[18] and

(d) that she was tachycardiac.[19]

50. Dr TX did not seek further guidance from the digital treatment guidelines that were available at TCH's computer system as to how to treat critically unwell patients who had suffered TCA overdoses. Those guidelines were entitled "*Therapeutic Guidelines: Toxicology and Toxinology, Tricyclic antidepressant (TCA) poisoning*" ("the Guidelines"). They relevantly identified the key investigations for TCA poisoning, namely ECG, blood gas analysis, and, significantly, serum potassium concentration in patients treated with serum alkalinisation (sodium bicarbonate). Serum alkalinisation was recommended when QRS widening was progressive and associated with symptoms such as breathing or circulatory compromise (for example, arrythmias, hypotension) or central nervous system depression. As to how much sodium bicarbonate to use, the advice was:

> For serum alkalinisation, in adults and children, use:
>
> sodium bicarbonate 8.4% 1 to 2 mL/kg up to 100 mL (1 to 2 mmol/kg up to 100 mmol) intravenously, every 3 to 5 minutes, titrated to a narrowing of the QRS complex and aiming for a serum pH between 7.45 and 7.55. Maximum total dose is 6 mL/kg (6 mmol/kg). Urgently seek advice from a clinical toxicologist if there is inadequate response to the maximum total dose.[20]
>
> PLUS
>
> hyperventilation by intubation and mechanical ventilation, aiming for a serum pH between 7.45 and 7.55 (typically by maintaining partial pressure of arterial carbon dioxide [$PaCO_2$] between 30 and 35 mmHg).

51. The warning that "serum alkalinisation can cause hypokalaemia due to intracellular potassium shift – monitor the serum potassium concentration and replace potassium if required, aiming for normal serum potassium concentration" was repeated.

52. Prior to August 2020, the relevant part of the Guidelines read:

> QRS widening and sodium channel blockade
>
> Continuous ECG monitoring and regular 12-lead ECGs are required to assess QRS widening. When QRS widening is progressive and associated with a decompensation in airways, breathing or circulation (see #[Toxicology: general approach/QRS widening]), intravenous boluses of sodium bicarbonate and concurrent hyperventilation therapy (by intubation and mechanical ventilation) should be used to increase the pH to a target of 7.45 to 7.55. Use:

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[18] Ibid [19.9].
[19] Ibid [19.10]. Meaning a fast heart rate.
[20] The guidelines had been amended in August 2020 to include this maximum dosage guidance. The guidelines were not accessible to Dr TX on her phone, but they were accessible online at TCH. Emphasis added.

sodium bicarbonate 8.4% (= 1 mmol/mL) 1 to 2 mmol/kg IV bolus, every 3 to 5 minutes, titrated to a narrowing of the QRS complex. If no response, urgently seek advice from a poisons information centre. Maximum total dose 10 mmol/kg

PLUS

hyperventilation following intubation and mechanical ventilation (where indicated).

53. At the time of Jessica's death there was no onsite toxicology service available at TCH. However, at the time and today doctors at TCH had/have access at any time of day to a Toxicology Hotline run by the Westmead Hospital Poisons Information Centre. The Poisons Information Centre is staffed by pharmacists and scientists with poisons specialisations. The Poisons Information Centre also employs clinical toxicologists from around Australia who provide specialised advice in respect of the medical management of human poisoning[21]. Dr TX did not consult that service.

54. Dr TX gave directions to begin the infusion of sodium bicarbonate and to insert a second IV canula in Jessica's other arm to facilitate the administration of sodium bicarbonate through both arms so as "to more rapidly facilitate the appropriate initial dose of approximately 150 meq".[22] No total dose of sodium bicarbonate was directed, and nursing staff were told to give sodium bicarbonate until they were told to stop. She directed that the administration of IV sodium bicarbonate be continued with 100 ml vials, rather than the smaller 10 ml vials. That started at 0751 hours. The evidence in the inquest suggests that vials of sodium bicarbonate were obtained from a variety of locations, including the resuscitation trolley, trolleys elsewhere in ED, and ACTAS staff (who obtained them from their re-stocking cupboard).

55. In her statement, Dr TX described what the process of treatment then was. That description was generally by reference to events rather than time, making more difficult an assessment of the objective measures that existed of Jessica's metabolic status when treatment decisions were made.

56. The records suggest that Jessica was commenced on sodium bicarbonate at 0755 hours. A venous blood gas sample[23] reported at 0755 hours showed: pH 7.24; pCO2 56 mmHg; pO2 26 mmHg; Na+ 132 mmol/L; K+ 4.5 mmol/L; Cl- 101 mmol/L; Ca++ 1.09mmol/L; Glucose 15.2 mmol/L; Lactate 3.3 mmol/L; and HCO3 19.7 mmol/L. The report showed reference levels identifying low and high ranges. The pH result was

[21] Information provided by ACTGS by letter dated 5 November 2024. This was the service that Dr KD consulted.
[22] Statement of Dr TX, dated 4 May 2023 [24.2].
[23] A blood gas test measures oxygen and carbon dioxide levels in the blood. The test can also show blood pH levels and lung function.

between the low and critically low levels. The Na+ (sodium) level was also between the low and critically low levels. Potassium (K+) was around normal.

57. At 0858 hours, blood gases were reported in these ranges: pH 7.64; pCO_2 64 mmHg; pO_2 41 mmHg; Na^+ 156 mmol/L; K^+ 3.4.mmol/L; Cl^- 99 mmol/L; Ca^{++} 0.84mmol/L; Glucose 11.1 mmol/L; Lactate 4.9 mmol/L; and HCO_3 19.7 mmol/L. The pH result was beyond the critically high figure of 7.55. The Na+ (sodium) level was also beyond the critically high level of 155. Potassium was just below the low (3.5) reference range.

58. It appears that Dr TX reviewed this result. She noted that "[Jessica] had become metabolically alkalotic with the administration of the sodium bicarbonate". She noted that the QRS "remained prolonged", although it had narrowed. She noted Jessica's serum sodium level to be elevated at 160 mmol/L,[24] "likely as a result of the sodium load in the sodium bicarbonate". The readings did not prompt a review of the treatment being administered notwithstanding the targets outlined in the Guidelines. The rationale for that approach was that the QRS "remained prolonged although it appeared narrower than previously (it was 123 milliseconds)".[25]

59. At 0910 and 0911 hours the administration of two further bags of bicarbonate was commenced. Dr TX suggested that around 0915 to 0920 hours, she was asked by a nurse whether further bicarbonate bottles should be administered:[26]

> 42.2. I considered this issue in light of my initial recommendation to continue administration of sodium bicarbonate until the QRS narrowed. The QRS had narrowed, but was still abnormally wide; the patient was now intubated; she had maintained greater Cardiovascular stability; and was now alkalotic.
>
> 42.3. I advised that I did not think further bicarbonate was indicated at this time.

60. She spoke to a colleague, Dr KD, after the last bags were administered:

> 44. Dr KD and I had a brief discussion about the elevated sodium level on the gas and the persistently prolonged QRS and QT interval on the cardiac monitor. Dr KD advised she would ring the Toxicology Hotline to discuss the situation and obtain guidance on further best practice management.

61. Dr TX then left the area briefly to attend to other tasks. Dr TX described what then happened (noting there was no timing references):

> 47. On my return I saw Dr KD sitting at the Resus staff station with a number of blood gases and ECGs in front of her. I have worked with Dr KD for 15 years and thought she looked very concerned. She advised me that it appeared that a total of 16 of the 100 ml sodium bicarbonate bottles had been administered, and that on discussion

[24] The printout appearing in the TCH records (at page 182) is timed at 0858 hours and suggests the reading was 156.
[25] Statement of Dr TX, dated 4 May 2023, [41].
[26] Ibid [42.1].

with the Toxicology Hotline staff, they were concerned that this represented the administration of a critical sodium load, particularly in the context of the raised sodium level on the blood gas. The concern was whether this placed Ms Bovill at risk of central pontine myelinolysis (CPM), a condition where the nerve sheath of brainstem cells becomes damaged with the rapid shift of water out of the cells into the more salt concentrated blood, where the sodium level is high.

48. Up until that point, I had lost situational awareness of how much sodium bicarbonate had been administered, given the other issues I was addressing. I had not considered the implications of the corresponding salt load. My focus had been on ensuring we managed the acute and immediately life-threatening risk of cardiac instability with a primary goal of narrowing the QRS using sodium bicarbonate.

62. Given her distress from the mistake she realised she had made, Dr TX stepped out of her care role, and the responsibility for Jessica's care was assumed by others.

63. At 0951 hours (it is assumed after Dr TX had stepped aside from Jessica's care) blood gases were reported in these ranges: pH 7.76; pCO_2 54 mmHg; pO_2 38 mmHg; Na^+ 168 mmol/L; K^+ 2.2 mmol/L; Cl^- 99 mmol/L; Ca^{++} 0.69 mmol/L; Glucose 11.3 mmol/L; Lactate 9.8 mmol/L; and HCO_3 63.1mmol/L. The pH level was above the critical range (7.55). Potassium was significantly below the critically low reference range of 2.9. Sodium was above the critical level (155).

64. It is not clear from the evidence as to how much sodium bicarbonate was eventually administered and Dr TX was unable to say with certainty.[27] There was a discrepancy between the medication chart (1180 mmols), the fluid balance chart (1500 mmols), and the clinical notes (1600 mmols).

[27] Ibid [48.1].

65. As a result of the administration of this amount of sodium bicarbonate,[28] Jessica suffered severe hypernatremia[29] and metabolic alkalosis.[30] The metabolic alkalosis in turn resulted in hypokalaemia.[31] An attempt was made to address those issues. It was known that there were significant risks associated with treating the overdose of sodium bicarbonate either conservatively or more aggressively. The risks and benefits of treatment options were discussed within the treating team.

66. Potassium was then administered. Jessica was transferred to the ICU, where she remained sedated and intubated. She was cooled, given insulin, administered IV noradrenaline, and commenced on dialysis. A brain scan was conducted at 2218 hours on 8 December 2020. It showed possible ischaemic changes without oedema. A further scan, conducted at 0630 hours on 9 December 2020, showed "rapid interval development of diffuse cerebral and cerebellar oedema" with uncal herniation and compression of the midbrain. A further MRI scan, conducted at 1046 hours on 10 December 2020, showed diffuse hypoxic ischaemic injury with uncal and tonsillar herniation. Neurological advice was that no intervention was possible.

67. Brain death was declared by the ICU consultant at 1545 hours on 11 December 2020.

68. A note made by Dr NT, ICU staff specialist, at 1747 hours (with errors included) summarised the advice given to Jessica's family in the following terms:

> Written in retrospect
>
> I met with the family at 16:00 hours after the perfusion scan was reported and was consistent with Brain death. Jessica was declared brain dead at 15:45 when the second specialist reviewed the scans and certified her dead.

---

[28] Advice was received from Professor Drummers as to the extent of the overdose:

> The amount of bicarbonate required will depend on a patient's clinical state that includes monitoring her blood pH, ECG and her electrolytes and while recommendations are for a maximum bicarbonate dose, such as 396 mL (6 mmol/kg x 66 kg body weight) for the current version of eTG or 660 mL for the previous version in 2020; it is vital that titration of bicarbonate infusion occurs and stops when her blood pH, ECG and electrolytes have improved sufficiently. This process is likely to take some time to allow for appropriate monitoring of these vital signs and allow her body to respond to the bicarbonate infusion(s).
>
> It is not possible to come up with a number of how many times her bicarbonate dose was exceeded given we don't know what bicarbonate dose she needed; arguably she may not have needed much, if any. Based on the maximum recommended dose using the 2020 eTG guideline (660 mL) and assuming she was only given 1180 mL bicarbonate her dose was 1.8 times the recommended, or almost 3 times the maximum recommended dose using the amended eTG guideline.

[29] High sodium levels in the blood. Here it peaked at 169 mmol/L. A normal blood sodium level is between 135 and 155 milliequivalents per litre (mEq/L). Severe hypernatremia is considered to be >160.

[30] Metabolic alkalosis is a disorder whereby the pH of tissue is elevated beyond the normal range (7.35–7.44 to 7.45).

[31] A low level of potassium.

I first introduced myself to the family and later updated again all the events and progress from the time she was found unconscious in the Kitchen at home. I told them Jessica presented with significant signs and symptoms of severe TCA overdose. I explained them these signs and the ECG changes she had on her presentation.

I also explained the potential neurotoxicity and cardiac toxicity of a severe TCA overdose. I told them the doctors in ED noticed that Jessica had seizures as they were planning to intubate her and that she was at risk of arrhythmias as well. She was treated with bicarb injections to stabilize her ECG changes. During this she did get a very high dose of bicarb in a very short time which let to severe derrangements in her acid base and electrolytes. Toxicology were consulted after this and they also mentioned that Jessica had very high dose of bicarb therapy. Their advise was to get renal specialist input for urgent dialysis.

The Renal specialist also expressed his concerns about the potential risks benefits of IHD vs, conservative therapy. There were challenges on both ways, with conservative fluid therapy with dextrose would mean that Jessica would need insulin therapy and that would push her K down further and increase risk of arrhythmias. It was felt, given her Na rise was acute (few hours), it would be more safer to treat the severe electrolyte/acid base derrangements with dialysis.

I also explained them given that Jessica had seizures in ED, with the electrolyte derrangements (low Ca), could have worsen her seizures which given that she was sedated and paralysed would be difficult to pick (Non convulsive status). This in addition to the rapid acid-base, osmolar shifts from high dose bicarb, put Jessica at risk of brain swelling, which unfortunately she suffered.

I then explained to the family that given worsening brain swelling, led to decrease and later no blood flow in her brain given the pressures in the skull would rise from the brain swelling. I explained the MRI findings from yesterday, they said that Jessica was having one but were not aware of the reports.

I than explained why we did a nuclear perfusion scan. I than explained them the results and that she was pronounced death at 15:45 hours.

I also later told them we were quite concerned how Jessica got high dose of bicarb and that the hospital has already started a root cause analysis on how someone would get this high dose of bicarb. I told them if they wish to speak to a hospital representative, they were happy to come and discuss the matter with them. Bill at this point expressed that he read about the TCA overdose, and said, people do survive even with 6gm OD of TCA and Jessica had taken about 1.5 gm. He expected Jessica to get better and return home. I said, I agree with his assessment, but unfortunately, things have not turned out that way.

I also explained to the family, given that there was a medication error where Jessica got very high dose bicarb therapy, we will refer her to the coroner who will conduct their own investigation to assess how much the above error contributed to her death. The hospital will continue with its own investigation and that they will be updated the finding of the same.

69. The family agreed to an organ donation process.

PART 5 – EXPERT ANALYSIS OF TREATMENT

70. The following part sets out the views of the experts and my findings in respect of three issues:

    (a) Was the amitriptyline overdose appropriately treated?

    (b) What caused Jessica's death?

    (c) Would Jessica have survived but for the overdose of sodium bicarbonate?

A. Was the amitriptyline overdose appropriately treated?

71. In her expert report, Professor Jones summarised the central features of TCA overdose management in the following way:

    (a) Appropriate clinical risk assessment of the likely timing and estimated total amount ingested;

    (b) Proper management of the airway (including intubation) to address central nervous system depression;

    (c) Administration of activated charcoal within 4 hours of ingestion to inhibit the absorption of the medication;

    (d) Prevention and management of cardiac arrythmias. This involves correcting any hypoxia, addressing electrolyte abnormalities, hypotension,[32] and acidosis.[33] Intravenous sodium bicarbonate "is the mainstay of treatment in tricyclic antidepressant poisoning as it resolves cardiac arrhythmias".

72. Professor Jones then explained how cardiac arrythmias may emerge:

> The principal mechanism of amitriptyline cardiotoxicity is sodium channel blockage resulting in slowing of depolarisation of the cardiac action potential through the myocardium and conducting tissue. Clinically this may present as prolongation of the QRS complex on the ECG (> 120 msec), QTc prolongation on the ECG and as potentially life-threatening arrhythmias.

73. In Professor Jones' opinion, each of these issues was addressed in Jessica's care:

    (a) She was identified as having an airway at risk and was correctly intubated and ventilated.

    (b) She was assessed by ECG for cardiac arrythmia and had her blood pressure and heart rate continuously monitored.

    (c) She was administered activated charcoal. Whilst it is likely that Jessica had presented 4 hours after ingestion of the amitriptyline overdose, use of this treatment was unlikely to have caused Jessica any harm.

[32] Low blood pressure.
[33] The blood becoming too acidic.

(d) She was administered sodium bicarbonate.[34]

74. Professor Jones and the other experts agreed that the amount of sodium bicarbonate that was administered was excessive. Associate Professor Skowronski indicated that:

> The generally recommended target is to achieve a blood pH of around 7.5, using a combination of bicarbonate and mild hyperventilation, which also raises pH by increasing the removal of CO2 from the blood. Most guidelines recommend a loading dose of 1-2mmol/kg body weight – around 100mmol in an average adult, followed, if needed, by a further 100mmol by infusion over some hours.

75. Consistent with the opinion of the experts, I find that although the broad parameters of the treatment of the amitriptyline overdose were appropriate, an excessive amount of sodium bicarbonate was administered.

B. What caused Jessica's death?

76. It was realised by treating doctors that sodium bicarbonate had been infused in overdose quantities. The subsequent clinical course was consistent with that fact. Treatments were directed at reversing the effects of the sodium bicarbonate overdose. At the time, it was the opinion of treating clinicians that there was a causative association between the excessive administration of sodium bicarbonate (and attempts to reverse it) and Jessica's terminal decline.

77. Associate Professor Skowronski described Jessica's decline and then death in these terms (after noting the volume of sodium bicarbonate that had been administered):

> This was a very large quantity of sodium bicarbonate, given over a short time (7.55 am to 9.11 am). It resulted in the expected sequelae of severe hypokalaemia, hypernatremia, hypocalcaemia and metabolic alkalosis. Of these, the hypernatremia, peaking at a serum sodium concentration of 169mmol/l between 11.29 am and 12.11 pm, is likely to have been the main contributor to Mrs Bovill's brain injury, as this is a well-known complication of severe hypernatremia.
>
> In brief, the main mechanism of injury is that this very high concentration of blood sodium draws water out of brain cells by osmosis, causing shrinkage. As the high sodium level is corrected with treatment, brain cells may swell to significantly larger than their original size. This brain swelling raises the pressure inside the skull, impeding blood flow and hence oxygen supply to the brain. It is this limitation of blood flow and oxygen supply that results in damage, or even death, of the brain.

[34] Associate Professor Skowronski noted that "most guidelines recommend bicarbonate treatment, given the ECG changes she exhibited and the transient hypotension, though some intensivist would be more conservative, reserving bicarbonate for clinically significant manifestations of cardiotoxicity". He went on to say that had he been treating Jessica "I would likely have withheld it in the absence of persistent hypotension or cardiac rhythm disturbance, though I acknowledge this would put me at odds with many current guidelines".

> The rapidity of correction can also be a factor in the development of brain swelling, though this is generally considered to be a more important issue when the hypernatremia has developed over a long period.

78. An alternative hypothesis explored during the inquest was that Jessica's death was directly caused by the original amitriptyline overdose. For that to be so, symptomology referable to the original overdose must have caused the hypoxic brain injury that was the cause of her death. Professor Jones discussed this possibility in her report, although she did so only at an abstract level. She indicated that it was possible that respiratory depression and the coma Jessica suffered might have caused her death if she had not been intubated and cared for at TCH. Those factors could have caused hypoxia. Hypotension and cardiac arrythmias (noting her prolonged QRS and QTc indicating risk) are an additional potential cause of death in amitriptyline poisoning. Professor Jones indicated that "additional expertise from ICU and renal physicians is required to comment on the effects of fluid, pH and tonicity shifts on possible cerebral oedema, as a consequence of excess intravenous bicarbonate administration in this case".

79. I am of the view that Associate Professor Skowronski was qualified to provide that commentary. Apart from the passages already quoted as to the cause of Jessica's death, he was asked specifically to consider whether Jessica would have suffered the same neurological injury if she had not been administered the sodium bicarbonate. He regarded that as very unlikely:

> In my opinion it is very unlikely, for reasons I have alluded to previously. In summary:
> - This was not a massive overdose, and the expected mortality would be low. In a 2019 national audit of 2381 US cases of amitriptyline overdose, only 10 deaths were reported.
> - Brain swelling is not a characteristic feature of tricyclic overdose, unless complicated by a major hypoxic insult.
> - There is no history in her presentation of cardiac or respiratory instability sufficient to explain hypoxic brain injury of this severity.
> - The amount of sodium bicarbonate administered greatly exceeds the usual clinical dosage.
> - The pattern of neurological injury is completely consistent with the severe electrolyte disturbance seen in this case, which in turn is an expected consequence of the very large dose of sodium bicarbonate administered.

80. It should be added that the heart arrhythmias that can be associated with deaths from amitriptyline overdoses were not seen in this case, despite the abnormal ECGs.

81. Consistent with that opinion, I find that the cause of Jessica's death was the neurological injury caused by the administration of the very large dose of sodium bicarbonate.

C. Would Jessica have survived but for the overdose of sodium bicarbonate?

82. Obviously, this question substantially overlaps the question addressed above and clearly involves an element of speculation. However, there are observations that can be made generally of amitriptyline overdoses that put Jessica's overdose into a statistical context.

83. In his expert report, Professor Drummer stated the following propositions:

    (a) The mortality from amitriptyline overdose is higher than for most drugs, although this is very much dependent on the dose, co-morbidities, type of treatment received in hospital and how soon after ingestion medical treatment commenced.

    (b) There is no dose or blood concentration of amitriptyline that will necessarily not be fatal or indeed cause death. While the dose is an important factor individual sensitivities (age, co-morbidities, other drugs etc) play a role, but importantly appropriate early medical intervention is critical, particularly when large doses are consumed.

    (c) When taken well in excess of a recommended dose, amitriptyline can cause death. The greatest risk of life-threatening symptoms usually occurs at doses in excess of 10 mg/kg, although lower doses can be toxic, particularly when acute medical attention is not sought and in medically vulnerable people, including those on other drugs.

    (d) The use of excessive doses can progressively cause anxiety, ataxia, tachycardia, alterations in blood pressure, arrhythmias, seizures, and coma. ECG changes are one of the more symptomatic signs, including prolongation of the QTc and QRS intervals. A QRS duration of >100 msec is an indicator of an overdose to drugs like amitriptyline (tricyclic anti-depressants). Ischaemic changes in the CNS or swelling of the brain are not listed as an expected adverse outcome.

84. Professor Drummer also reviewed a number of studies where amitriptyline overdoses had occurred:

    (a) In one review, initial signs seen in emergency departments that were associated with a fatal outcome were coma (61%), tachycardia (55%), hypotension (50%), respiratory depression (39%), and seizures (39%) with QRS prolongation present in 28% and major arrhythmias in only 1.5%.[35]

[35] Professor Drummer, Expert Forensic Report in the matter of Jessica Bovill deceased, dated 24 April 2024, [5.15].

(b) A study of 40 consecutively hospitalised patients due to tricyclic anti-depressants, including amitriptyline, monitored 13 patients with levels greater than 1 mg/L which is about 4-10 times the optimal concentration for therapeutic treatment. Of these 13 patients 7 had ingested amitriptyline alone with doses ranging from 1.1 to 2.2 grams and all had a QRS interval of >100 msec. Other cardiac abnormalities reported included ventricular tachycardia (>120 bpm, n=7), right bundle-branch block (n=4), atrial and ventricular pre-mature contractions, junctional arrhythmia, and idioventricular arrhythmias. Ten became unconscious and 3 had cardiac arrests. Sodium bicarbonate infusion was apparently not used in these patients. Only two of the patients died. There was no mention of cerebral ischemia or oedema in these patients.[36]

(c) Another publication involved a review of 110 patients who had overdosed on amitriptyline and were admitted to hospital. The most common symptoms were sinus tachycardia, altered mental state and hypotension followed by respiratory depression requiring ventilatory support. About half of these patients had taken over 500 mg amitriptyline, although individual doses were not reported, and plasma measurements were not conducted. Bicarbonate was not administered to any of these patients, and no patient died.[37]

85. Professor Drummer's conclusion was:

> 7.10. Death from amitriptyline overdose can occur, even with proper medical intervention in hospitals even with lower doses, however most overdose admissions to hospital for this drug do survive. However, what cannot be ascertained with any certainty is whether survival would necessarily have occurred in Ms Bovill's case if bicarbonate was not given. The blood concentration of amitriptyline, while very high, could have risen further on the day of her admission due to continuing absorption, or if the amount ingested was less and/or she had taken the drug much earlier in the night it may have been already near the peak concentration.

86. I conclude from this overview and the information provided by Associate Professor Skowronski that amitriptyline overdoses can be fatal. We do not know when Jessica ingested the amitriptyline. A significant delay between ingestion and treatment can be a factor relevant to whether the person survives. The amount of amitriptyline taken by Jessica was significant and, without treatment, life-threatening, but it was not as high as it was in other cases involving overdoses not leading to death. Her condition when admitted to TCH was serious. The symptoms that were suffered by Jessica at the time

[36] John Biggs et al, 'Tricyclic Antidepressant Overdose: Incidence of Symptoms' (1977) 238(2) *JAMA* 135.
[37] Cahfer Güloglu et al, 'Analysis of Amitriptyline Overdose in Emergency Medicine' (2011) 28(4) *Emergency Medicine Journal* 296.

of her admission are associated with the symptoms of patients who have died from amitriptyline overdoses and those who have survived. In the quoted reviews, the incidence of fatal outcomes of those treated in a hospital setting is a relatively small percentage of cases, even when serum levels of amitriptyline are higher (and in some cases much higher) than those in Jessica's case.

87. In the quoted reviews, where overdose has resulted in death, death has not been attributed to, or associated with, cerebral ischemia or oedema.

88. I note and accept Associate Professor Skowronski's opinion that it is likely that Jessica would have survived but for the sodium bicarbonate overdose. However, I cannot exclude the possibility that Jessica would still have died from the amitriptyline overdose if she had been appropriately treated at TCH. The reasonable survival prospects she had were lost because of the administration of sodium bicarbonate in excessive quantities.

PART 6 – FORMAL FINDINGS

89. On the basis of the material available to me in the inquest, and particularly in light of the opinions expressed by Associate Professor Skowronski, I find that Jessica's death was a sequela of the use of an excessive amount of sodium bicarbonate in the treatment of her initial overdose of amitriptyline. I find that there was no suggestion of symptoms prior to admission of a severity that could have caused hypoxic brain injury of the type that caused Jessica's death. Such brain injury is not associated in any published reviews with deaths caused by TCA overdose.

90. I do not accept the suggestion that hypoxic brain injury had occurred prior to the administration of sodium bicarbonate at the direction of Dr TX. If seizures were present at the time of Jessica's admission to TCH, that is a symptom consistent with TCA overdose generally.

91. Accordingly, for the purposes of s 52(1) of the Act, I find that:

> Jessica Hilary Bovill died at the Canberra Hospital at about 1545 hours on or about 11 December 2020. She died of severe diffuse hypoxic brain injury caused by the administration of a supratherapeutic level of sodium bicarbonate in the context of the treatment of a self-inflicted and deliberate overdose of amitriptyline.

PART 7 – MATTERS OF PUBLIC SAFETY

Dr TX's standard of clinical practice

92. In her statement, Dr TX drew attention to the fact that the outcomes in Jessica's case had been the subject of review by practitioners outside of TCH and associated with the Poisons Information Centres in Queensland and NSW.[38] An article was published in 2022, noting "a concerning trend of iatrogenic bicarbonate poisoning occurring in patients presenting following overdose of drugs with sodium blocking action".[39] Jessica's case (de-identified) was one of the cases used to highlight that trend. From those case studies and information available to the Poison Information Centres, the authors identified the following common errors in the treatment of TCA overdoses:

    (a) Forgetting the importance of ventilation when giving bicarbonate therapy. Bicarbonate may paradoxically worsen the pH with rising pCO2 in the unventilated patient. Gently hyperventilate, aiming for a low normal pCO2 to assist in reaching the target pH range.

    (b) Aiming for an unrealistic QRS target (e.g. 100–120 ms) rather than a target pH range. The QRS often remains prolonged (e.g. 120–140 ms) for 24–48 h following severe TCA overdose. Furthermore, in sodium channel blockade because of non-TCA medications, the QRS prolongation may not respond to bicarbonate at all.

    (c) Not performing blood gas analysis regularly to ensure the target pH and pCO2 are met and iatrogenic harm is avoided. Complications like severe hypokalaemia can often go unrecognised and can contribute to haemodynamic instability.

    (d) Treating a broad complex QRS that is not secondary to sodium channel blockade but is rather a pre-existing interventricular conduction delay.

93. The authors went on to recommend that practitioners dealing with TCA overdoses use the guidance provided by the Guidelines, and that they discuss such cases with the poisons centre or a local toxicologist.

94. Dr TX, whilst undoubtedly busy, made clinical decisions based on inadequate consultation and without reference to available and authoritative clinical guidance and the Poisons Information Centre. Whilst the online guide (LITFL) was no doubt useful in its content, it did not contain the explicit warnings that were contained in the Guidelines. The Guidelines were available to practitioners at TCH and should have been consulted. Advice should have been sought from a toxicologist much earlier. The amount of sodium bicarbonate administered was far in excess of the suggested maximum dosage. The level of serum alkalinisation was not appropriately monitored, and the continued

[38] Katherine Isoardi and Angela Chiew, 'Too Much of a Good Thing: Bicarbonate Toxicity following Treatment of Sodium Channel Blocker Overdose' (2022) 34 *Emergency Medicine Australasia* 639.
[39] Ibid.

administration of sodium bicarbonate occurred in the face of blood gas results suggesting they had already reached critical levels.

95. I find that the actions of Dr TX contributed to the cause of Jessica's death.

96. The information provided to me does not enable me to say whether Dr TX's clinical practice on that day was reflective of a general shortfall in professional competence and, therefore, giving rise to a matter of public safety.

97. I shall cause my findings to be forwarded to the Australian Health Practitioner Regulation Agency ("AHPRA"), so that issue can be appropriately investigated.

Other matters of public safety

98. I otherwise find that issues of public safety do arise from the inquest I have conducted. I make the following comments and recommendations.

(i) Guidance and advice in treating TCA overdoses

99. There was a lack of mandatory requirement to seek toxicological advice from the Poisons Information Centre in respect of TCA overdoses, and to consult the Guidelines in the case of a TCA overdose.

100. I recommend that processes at TCH be reviewed to ensure that ED staff are alerted to the need to:
    (a) seek out any such advice from a toxicology consultant at the Poisons Information Centre when patients present to the ED with a potential TCA overdose; and/or
    (b) consult the Guidelines or other officially approved advice as to the treatment of such cases.

(ii) Access to sodium bicarbonate

101. In the course of Jessica's treatment, there appeared to have been unrestrained access to large amounts of sodium bicarbonate from a variety of sources. This reality may not have provided a situational alert to nursing staff that a large amount of sodium bicarbonate was being administered.

102. I recommend that processes be reviewed with a view to limiting the availability of sodium bicarbonate to a central point of supply.

103. In respect of these recommendations, I note the contents of Ms Zagari's statement. My findings will, for the purposes of s 57 of the Act, constitute my report to the Attorney-General of the inquest I have conducted. Any response by the Attorney-General or

another Minister to my report must be made in accordance with the terms of s 57(4) of the Act.

POSTSCRIPT

104. I express my condolences to Jessica's family and apologise to them for the time taken to publish these findings. I acknowledge that a delay in the resolution of coronial proceedings has added to the trauma of the family associated with the passing of a much-loved wife and mother.

I certify that the preceding one hundred and four [104] numbered paragraphs are a true copy of the reasons for findings of his Honour Coroner Archer.

Associate: Markus Ching

Date: 26 February 2025

IN THE CORONERS COURT)
AT CANBERRA IN THE) CD 334/2020
AUSTRALIAN CAPITAL TERRITORY)

Inquest into the death of
JESSICA HILARY BOVILL

Witness Statement – Janet Zagari
Deputy Chief Executive Officer, Canberra Health Services

1. This statement made by me accurately sets out the evidence that I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief.

2. This statement is provided pursuant to section 55(1)(b) of the *Coroners Act 1997* (ACT) (**the Act**) in response to the s 55 Notice addressed to the Australian Capital Territory that was accompanied by the Provisional Findings of Coroner Archer issued on 13 December 2024 (**Provisional Findings**). For the purposes of preparing this statement, I have been provided with and have reviewed the s 55 Notice and the Provisional Findings.

3. My full name is Janet Leigh Zagari. I am currently employed at Canberra Health Services (**CHS**) as the Deputy Chief Executive Officer located at 2 Bowes Street, Woden. I have been employed at CHS since 1 July 2022.

4. I am authorised to make this statement on behalf of the Australian Capital Territory (the **Territory**).

5. I prepared this Statement from my own knowledge, information and belief and by making inquiries of relevant persons. Where I have relied upon information provided by others, I believe that information to be true and correct.

6. The Territory acknowledges and accepts the comments and criticisms made by Coroner Archer in the Provisional Findings and recorded in the s 55 Notice in relation to the care that Ms Jessica Bovill (**Ms Bovill**) received at The Canberra Hospital.

7. This statement is intended in the absence of a hearing and submissions to provide context in relation to CHS changes in response to Ms Bovill's death. It is a short summary of evidence that would have been provided to the Coroner and Counsel Assisting on those issues. For ease of reference, it refers to the relevant paragraphs in the provisional findings.

8. I provide this statement to provide updated context in relation to paragraphs 95 and 97 of the Provisional findings.

Changes to Policy and Practices

9. In response to Ms Bovill's death, the policy known as '*Emergency Department presentations of Life-Threatening Poisoning – CHS21/507*" (**the policy**) was developed and issued on 9 September 2021. Annexed hereto and marked with the letter "**A**" is a copy of the policy.

10. Section 2 of the policy states:

> "*Specific situations mandating a discussion, and documentation of the discussion, with the* ***Poisons Information Centre on 131 126*** *are as follows:*
>
> 1. *Any poisoning requiring an antidote (except N-acetyl cysteine or naloxone)*
> 2. *Any poisoning requiring admission to hospital to manage the poisoning specifically (does not include those admitted for the social circumstances or mental health issues prompting the poisoning)*
> 3. *Any poisoning with ECG changes (for instance, broadened QRS, R wave in AVR, QT prolongation, any degree heart block, bradycardia)*
> 4. *Any poisoning with significant derangement on venous or arterial blood gas*
> 5. *Any poisoning with haemodynamic instability."*

11. Further, in response to Ms Bovill's death, CHS has made changes such that sodium bicarbonate is now more restricted in both availability and the mechanisms for prescription.

12. Sodium bicarbonate is first line treatment for overdose with calcium channel blockade and widened QRS complex. Treatment is time critical, as such it is necessary to continue to have small quantities of sodium bicarbonate in the Resuscitation trolleys in each Resuscitation room. There are other critical uses which require immediate availability in specific areas of the hospital such as ICU. CHS has taken steps to appropriately limit the amount which is directly available.

13. At the time of Ms Bovill's admission, sodium bicarbonate could be administered by verbal order in the case of time critical interventions. At that time, these orders were recorded on the paper medication chart or intravenous orders chart. Since that time the Digital Medical Record (**DHR**) has been implemented across CHS.

14. As a result of Ms Bovill's death, sodium bicarbonate has been excluded from the list of medications which can be administered in ED on the basis of a verbal order. Verbal orders can only be recorded in DHR using the 'ED Narrator' function and is limited to a specific list of medicines. Sodium bicarbonate is expressly excluded and requires a documented order from the doctor before it can be administered. Annexed hereto and marked with the letter "**B**" is a screenshot of the ED Narrator showing the exclusion of sodium bicarbonate from the list.

15. All prescribing of sodium bicarbonate now occurs electronically for each dose. The default ordering dose for sodium bicarbonate is "Once". Annexed hereto and marked with the letter "**C**" is a screenshot from DHR showing the default dose. As a consequence of this change, the DHR alerts clinical staff when a dose has already been given, a purple warning will appear. If a user attempts to proceed to sign the order in spite of the warning, an additional prompt will present on screen entitled "*Duplicate Medication Therapy*" requiring the clinician to enter a reason as to why the order for the medication is being duplicated. Annexed hereto and marked with the letter "**D**" is a screenshot of the alert and warning.

16. Any and all doses of sodium bicarbonate that have already been administered are visible to all medical staff in DHR. Additionally, a nurse is required to enter/record an ordering and authorising clinician prior to the medication being ordered and available for administration. Annexed hereto and marked with the letter "**E**" is a screenshot of the ordering information required.

17. On 26 November 2021, the "*ED Nursing Education Booklet Green Level V9.1*" was updated to include the line "*Important – the same medication cannot be administered concurrently through multiple lines*", to prevent large doses of medication not being identified because simultaneous lines are running. Annexed hereto and marked with the letter "**F**" is a copy of page 24 of the ED Nursing Education Booklet Green Level V9.1.

(Signature)

Janet Zagari

Signature witnessed by me about 15:00 on
15/01/2025 at 2 Bowes Street, Phillip in the Australian Capital Territory.

(Signature)

Courtney Malyszko