CORONER'S COURT
OF NEW SOUTH WALES

Inquest:	Inquest into the death of OS
Hearing dates:	25 to 28 December 2019
Date of findings:	31 January 2020
Place of findings:	Coroner's Court of New South Wales at Lidcombe
Findings of:	Magistrate Derek Lee, Deputy State Coroner
Catchwords:	CORONIAL LAW - cause and manner of death, oral sedation, observations, respiratory depression, SUDEP, valproic acid, bioavailability of morphine
File number:	2015/91895
Representation:	Ms M Gerace, Counsel Assisting, instructed by Ms B Holliday-O'Brien (Office of the General Counsel, New South Wales Department of Justice and Communities) Mr N Abdi (Aboriginal Legal Service NSW/ACT) for Ms K Simpson Ms T Berberian for Dr S Ardern-Holmes & Dr L Chan, instructed by Ms A Quinlivan (HWL Ebsworth Lawyers) Ms L Boyd instructed by Ms J Power (Crown Solicitor's Office) for Sydney Children's Hospital Network Ms H Cooper (Legal Aid Commission of New South Wales) for Mr S Sawle

Findings:

I find that OS died on 26 March 2015 at Colyton NSW 2760. The cause of OS's death was due to the combined effects of prescribed morphine, valproic acid and midazolam leading to sedation and eventually terminal respiratory depression. OS died as a result of the synergistic effects of his existing medication regime and medication prescribed to him on 26 March 2015 leading to an unintended higher bioavailability of morphine.

Non-publication orders:

Pursuant to section 75(2) of the *Coroners Act 2009* publication of any matter (including the publication of any photograph or other pictorial representation) which identifies any of the following persons is prohibited:

1. OS
2. JM also known as JS
3. TS
4. JS

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1. Introduction

1.1 OS was five years old at the time of his death. On 17 March 2015 OS suffered a burn whilst in the bath at home. After having his burn wound treated at hospital on 19 March 2015, OS returned to hospital a week later to have his wound dressing changed, and was prescribed oral sedative medication as part of the procedure. After returning home from hospital, and only a matter of hours after the procedure, OS was found to be unresponsive with no signs of life. Following unsuccessful resuscitation attempts, OS was tragically pronounced deceased.

2. Why was an inquest held?

2.1 Under the *Coroners Act 2009* (**the Act**) a Coroner has the responsibility to investigate all reportable deaths. This investigation is conducted primarily so that a Coroner can answer questions that they are required to answer pursuant to the Act, namely: the identity of the person who died, when and where they died, and what was the cause and the manner of that person's death. All reportable deaths must be reported to a Coroner or to a police officer.

2.2 OS's death raised a number of questions about the circumstances surrounding it, in particular the events at hospital on 21 March 2015 which immediately preceded his death. Furthermore the postmortem examination performed after OS's death was unable to ascertain a cause of death. As a result an inquest was required to be held in an attempt to determine the cause and manner of OS's death, and so that these issues relevant to these questions could be explored in detail.

2.3 In this context it should be recognised at the outset that the operation of the Act, and the coronial process in general, represents an intrusion by the State into what is usually one of the most traumatic events in the lives of family members who have lost a loved one. At such times, it is reasonably expected that families will want to grieve and attempt to cope with their enormous loss in private. That grieving and loss does not diminish significantly over time. Therefore, it should be acknowledged that the coronial process and an inquest by their very nature unfortunately compels a family to re-live distressing memories several years after the trauma experienced as a result of a death, and to do so in a public forum.

2.4 Inquests have a forward-thinking, preventative focus. At the end of many inquests Coroners often exercise a power, provided for by section 82 of the Act, to make recommendations. These recommendations are made, usually, to government and non-government organisations, in order to seek to address systemic issues that are highlighted and examined during the course of an inquest. Recommendations in relation to any matter connected with a person's death may be made if a Coroner considers them to be necessary or desirable.

3. Family history and background

3.1 OS was born to his parents, Narelle Sawle and Geoffrey Simpson on 28 January 2010. Ms Sawle is from the Darug mob of the Eora nation and Mr Simpson is an Aboriginal elder from the Gunagarra mob near Bulli. OS's name means "[redacted]".

3.2 OS was the sixth child of Ms Sawle. He had an older sister (TS) and four older brothers (Clinton, Daniel, JS and JS). Shortly after his birth OS was removed from the care of his parents and placed into foster care with David and Jodi Agius on 8 February 2010. OS's brother JS had earlier been placed into foster care with David and Jodi on 7 December 2009. In May 2010 final orders were made for OS with respect to all aspects of parental responsibility to the Minister until OS turned 18.

3.3 OS experienced a gastro-oesophageal reflux as a baby which later resolved at six months of age. In December 2012 OS developed seizures. Despite extensive workup and investigations no definitive diagnosis was made as to the cause of his seizures.

3.4 Following an assessment at the Children's Hospital at Westmead (**the Hospital**) in August 2014 OS was diagnosed with global developmental delay, mild intellectual disability and attention deficit hyperactivity disorder (**ADHD**). OS was prescribed multiple medications including valproic acid (to treat epilepsy), clobazam (a benzodiazepine to treat seizures), lamotrigine (anticonvulsant medication), topiramate (to treat seizures), clonidine (to treat ADHD), and melatonin (to treat sleep disorder).

3.5 OS had been referred for early intervention services which included physiotherapy, speech therapy and a review with a psychologist once a week. He also had ongoing follow-up with a paediatrician from the Child Development Unit at the Hospital for his developmental issues.

4. What happened on 19 March 2015?

4.1 On the afternoon of 19 March 2015 a childcare worker from the day care centre which OS attended brought OS to the Emergency Department (**ED**) at the Hospital. The childcare worker reported that OS had arrived at the day care centre that morning and was noticed to be teary with a burn to his lower back which had a loosely applied dressing. The childcare worker subsequently spoke to Jodi about the burn. Jodi explained that OS had been in the bath with his brother, who had turned on the hot water tap causing the burn to OS's back.

4.2 OS was subsequently admitted under the surgical team who reviewed his burn in the ED. On examination OS was found to have a 5cm by 5cm superficial to partial thickness burn on his back. The burn was noted to be one percent of total body surface area on the lower back and was suggestive of a contact burn.

4.3 Jodi later attended the ED and provided a history regarding the burn. She indicated that OS most likely sustained the burn on 17 March 2015 when he was having a bath. The incident was not witnessed but Jodi said that she believed OS may have fallen against the hot water faucet in the bath. Afterwards OS's foster parents noticed a small blister on his back, but that OS was otherwise well. David applied dressings to OS's back and his foster parents kept an eye on him. Jodi reported that the blister later burst and then the redness surrounding the burn increased in size.

5. What happened on 20 March 2015?

5.1 OS was subsequently admitted overnight. Dr Alan Pham, a registrar attached to the Hospital's Burns Unit, reviewed OS on the morning of 20 March 2015. At the time it was noted that OS's pain was controlled, itch was not a problem for him, and that his dressings were dry and intact.

5.2 Arrangements were made for the Hospital's Child Protection Unit (**CPU**) medical team to review OS later that day. As part of the review it was noted that OS was observed to be drowsy, unbalanced and demonstrated an unsteady gait on examination in the ward. It was also noted that in the days and weeks prior to OS's admission childcare workers at OS's day care centre reported that he was drowsy and would sleep all day if permitted to, sometimes having to be woken for lunch. As a result the CPU team raised concerns that the symptoms were a result of an underlying neurological cause or the result of side-effects from OS's medications. Following this review neurology and haematology consults were requested, and a request was also made that OS not be discharged until the CPU team had liaised with the Burns Unit.

5.3 Dr Lok Chi Denise Chan, neurology fellow, reviewed OS at about 5:00pm on 20 March 2015 with Jodi present. Jodi reported that since OS had his dose of clonidine changed to a morning dose, she had noticed OS to be drowsy for a few hours in the mornings. However, Jodi reported that by the afternoon OS was his usual hyperactive self and difficult to put to bed at night. Dr Chan noted that OS's platelets on admission were low, and that a repeat platelet count performed that afternoon was also low, consistent with a previous count that had occurred in August 2014.

5.4 Dr Chan discussed OS's condition with Dr Simone Ardern-Holmes, a paediatric neurologist. Dr Ardern-Holmes had seen OS on a number of earlier occasions since OS was referred to her in May 2013 following an increase in the frequency of his seizure activity from December 2012. Following this discussion it was felt that OS's morning drowsiness was likely the result of the change in the timing his dosage. Accordingly, Dr Ardern-Holmes recommended that OS's clonidine dose be changed back to a night dose to help him sleep at night. Dr Ardern-Holmes also recommended a haematology review and that blood tests be repeated before OS's discharge to ensure that OS's platelet level was not dropping further.

5.5 Dr Pham reviewed OS again that afternoon. He confirmed with the CPU team that they were happy for OS to be discharged, and that the neurology team had requested another repeat platelet count the next morning before OS was discharged home. Dr Pham confirmed with the haematology on call doctor that the repeat platelet count should be performed prior to discharge. A follow-up clinic appointment was then scheduled for OS in the Burns and Plastics Treatment Centre on 26 March 2015. A general case conference and a video electroencephalogram (to record electrical patterns in the brain as part of treatment for epilepsy) were also planned for 30 March 2015.

6. What happened on 26 March 2015?

6.1 On the morning of 26 March 2015 David drove Jodi and OS from their home in Colyton to the Hospital so that OS could attend the Burns Unit to have his dressing changed. Upon arrival in the Burns Unit OS was taken to a waiting room area by Nurse Practitioner (**NP**) Kelly Waddell. In accordance with standard procedure within the Burns Unit OS was prescribed oral sedation prior to having his dressing changed.

Oral sedation and change of dressing in the Burns Unit

6.2 Dr Tristan Bennett was the Burns Unit Anaesthetic Fellow on 26 March 2015. After reviewing OS's clinical progress notes and the medication chart from OS's most recent admission, Dr Bennett prescribed paracetamol, morphine and midazolam to be administered to OS as oral sedation. The Hospital's *Oral Sedation Guidelines for Burn Dressing Changes with Nitric Oxide* (**the Sedation Guidelines**) sets out the amount of oral sedation to be administered to a patient according to their weight. In accordance with the Sedation Guidelines, after NP Waddell performed the appropriate calculations, the following oral sedation was administered to OS:

(a) 250 milligrams of paracetamol based on a calculation of 15 milligrams per kilogram (rounded up from 214.5 milligrams);

(b) 7 milligrams of morphine based on a calculation of 0.5 milligrams per kilogram (rounded down from 7.15 milligrams); and

(c) 4.5 milligrams of midazolam based on a calculation of 0.3 milligrams per kilogram (rounded up from 4.29 milligrams).

6.3 According to the Ward Register for drugs of addiction the quantities of midazolam and morphine were given to OS at 9:35am. Observations and vital signs were taken at the time of administration and OS remained in the waiting room to allow the medications to take effect. Following this OS was taken to the procedure room where his dressing was removed. At this time NP Waddell noted that OS was mildly sedated, he was orientated to place, he could hold his head up and could follow demands, and he was able to sit up without being held.

6.4 After removing the dressing NP Waddell observed that OS's wound had healed and whilst it remained pink in colour, this was normal for its stage of healing. Sorbolene cream was massaged into the wound area and its surrounds, and the burn site was left uncovered. NP Waddell noted that OS showed nil distress during the procedure, and that observations taken at the time when the oral sedation was initially administered, and at the time when the procedure concluded at 10:10am, were both within normal limits.

6.5 NP Waddell provided Jodi with a copy of a *Healed Burns Fact Sheet*, and discussed with her the steps required to manage the burn area over the following months. NP Waddell noted that following the procedure OS was sitting up on the dressing table, and was alert and eating a biscuit. She said that OS remained under observation until he and Jodi left the procedure room a short time later. Following this NP Waddell commenced an entry into the electronic medical record at 10:56am, completing it at 11:01am. NP Waddell said: *"At no time during the course of the morning was OS*

drowsy or unrouseable. He remained alert but relaxed once the medication had taken effect. He remained in that state throughout the course of the procedure. There was no indication of any ongoing sedation at the time OS left the Burns Unit".[1]

6.6 In evidence given during the inquest Jodi said that she was told that OS was being given midazolam to relax him in case he became active during the procedure. She recalled being told that OS's burn appeared well and was healing. Jodi said that she recalled asking if she and OS could leave following the administration of midazolam. She said they she was told that OS would be fine, just a *"little bit wobbly"*.

Departure from the Burns Unit

6.7 After leaving the clinic Jodi took OS downstairs to a coffee shop in the Hospital to get him something to eat. At about 10:21am Jodi sent David a text message advising him that the procedure had finished and that OS was eating a sausage roll. OS took a bite from a sausage roll and then went to sleep in Jodi's arms. Following this Jodi sent a text message to David and they arranged for him to pick them up. Jodi left the Hospital to meet David, whilst carrying OS in her arms as he was still asleep. CCTV footage from the Hospital shows Jodi and OS exiting the Hospital at about 10:27am.

6.8 After leaving the Hospital Jodi and David drove to a McDonald's in Wentworthville, about 15 minutes away, to get some lunch. OS was in the back seat of the car and still asleep at this time. The family stayed at the McDonald's for about five minutes and then drove home, another 10 to 15 minutes away.

Arrival at home

6.9 Upon arriving home, OS was still asleep and so Jodi laid him down on the lounge. She then attended to some housework. Shortly before 3:00pm David left home to pick up JS from school. At this time OS was still asleep on the lounge. Jodi explained that this was not unusual as OS used to frequently get tired from his medication. David said that before leaving the house to pick up JS he did not see OS move or rouse.

6.10 After David and JS returned home, JS did some homework and then he and David played in the backyard for a short time. At about 4:15pm they went to the shops in St Mary's, about 10 minutes away, and then drove to another location 15 minutes away to get dinner.

6.11 At about 5:00pm Jodi went to check on OS who was still lying on the lounge and believed to be asleep. Jodi found that OS was instead unresponsive. Jodi picked up OS and carried him next door to a neighbour's house seeking assistance. Emergency services were called at about 5:05pm and paramedics arrive at the scene at about 5:12pm. Cardiopulmonary resuscitation (**CPR**) was initiated but OS could not be revived. He was later pronounced deceased at 5:24pm.

[1] Exhibit 1, page 544 at [36].

7. What was the result of the postmortem examination?

7.1 OS was later taken to the Department of Forensic Medicine located (at the time) in Glebe. A postmortem examination was performed by Dr Jennifer Pokorny on27 March 2015. Dr Pokorny subsequent prepared an autopsy report dated 6 July 2015.

7.2 The toxicological analysis at autopsy provided the following results:

(a) Clobazam, 0.14 mg/L
(b) Lamotrigine 1.7 mg/L
(c) Midazolam <0.005 mg/L
(d) Morphine (free) 0.33 mg/L
(e) Morphine-3-glucuronide 0.16 mg/L
(f) Morphine-6-glucuronide 0.03 mg/L
(g) Paracetamol 5.8 mg/L
(h) Valproic acid 130 mg/L

7.3 Dr Pokorny noted that the clobazam and paracetamol were at therapeutic levels, the midazolam was at a low level, the lamotrigine was at a level slightly below therapeutic use, and the valproic acid was at a supratherapeutic level and within the range in which toxic effects including sedation have been reported. Dr Pokorny went on to note that the level of morphine was *"higher than what is typically seen with morphine used in an outpatient setting for short term pain relief"*[2] and that at this concentration it may cause sedation and respiratory depression. She went on to explain that *"these effects would likely have been exacerbated by the concurrent supratherapeutic valproic acid, and the midazolam"*.[3]

7.4 Ultimately Dr Pokorny concluded that the cause of OS's death remained unascertained following postmortem examination, but noted that the clinical findings raised the following considerations:

(a) The effects of multiple medications, some of which were at supratherapeutic levels and potentially sedating, when given in combination may be unpredictable;

(b) OS had a history of drowsiness which predated his burn injury and was noted to have quite frequent seizures which may have led to drowsiness in the post-ictal state;

(c) Widespread inflammatory changes in the brain of uncertain pathogenesis and unclear significance raised the possibility that these changes may have contributed to OS's drowsiness or to the cause of death; and

(d) Due to his history of epilepsy OS was at increased risk of Sudden and Unexpected Death in Epilepsy (**SUDEP**). Dr Pokorny went on to note that *"there are typically no morphological abnormalities that can be seen at autopsy and the diagnosis is largely one of exclusion, which given the toxicological and neuropathological findings in this case is not possible"*.[4]

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[2] Exhibit 1, page 10.
[3] Exhibit 1, page 10.
[4] Exhibit 1, page 11.

8. What issues did the inquest examine?

8.1 Prior to the commencement of the inquest a list of issues was circulated amongst the sufficiently interested parties, identifying the scope of the inquest and the matters to be considered. That list identified the following issues:

(a) In relation to the medical treatment provided to OS on 26 March 2015:

(i) Was it appropriate for the medication to be administered?

(ii) Was there a need for consultation with OS's treating specialists about his pre-existing medications to discuss the risk of potential complications with additional opioid medication?

(iii) Was OS adequately observed post administration of medication given his existing medication regime and side effects?

(iv) Was OS safe for discharge?

(b) What was the cause of OS's death?

8.2 Each of the above issues is discussed in detail below. In order to assist with consideration of some of these issues, opinion was sought from the following experts below as part of the coronial investigation. Each of the experts provided reports which were included in the brief of evidence, and also gave evidence during the inquest.

(a) Professor MacDonald Christie, consultant pharmacologist;

(b) Dr Scott Dunlop, consultant paediatrician; and

(c) Dr Michael Kennedy, consultant physician and clinical pharmacologist.

9. Was it appropriate for the medication to be administered to OS on 26 March 2015?

9.1 The first matter that arises for consideration is whether it was generally appropriate for oral sedation to be provided to OS at all in relation to his dressing change procedure. The *Procedural Sedation (Paediatric Ward, Clinic and Imaging Areas) Practice Guideline* (**the Practice Guideline**) was developed for use within the Sydney Children's Hospital Network (**the Network**). It provides guidelines for the safe administration of procedural sedation by non-anaesthetists within the Network. The Practice Guideline provides that the aims of procedural sedation are to minimise pain and physical discomfort for a patient, reduce movement by a patient (and therefore the need for physical restraint), and to reduce fear and anxiety in patient. Further, the Practice Guideline provides that these aims must never compromise patient safety.[5]

9.2 In evidence Dr Bennett explained that it was accepted as a starting point that some analgesia or sedation for a burns patient would be required for a change of wound dressing. He noted that

[5] Exhibit 1, page 562-23.

morphine was commonly used in the Burns Unit, and that it was prescribed in order to mitigate the pain associated with changing burns dressings. He explained that morphine was generally well tolerated by patients attending the Clinic and that in OS's case there was no evidence of previous adverse to reactions to morphine.[6]

9.3 On this issue Dr Dunlop expressed the following views:

(a) It was necessary and appropriate to administer medication for OS's dressing change as it was expected that the potential for pain could heighten a child's anxiety and complicate the procedure;[7]

(b) The dosages of oral sedation administered to OS were in keeping with the Sedation Guidelines, and appropriately administered just prior to the procedure;[8]

9.4 **Conclusion:** It can be accepted, as a general matter, that patients are likely to experience a degree of discomfort and pain associated with a dressing change for a burns wound. Having regard to the use of oral sedation to mitigate such discomfort and pain, and to alleviate any associated fear and anxiety (particularly in a child patient), it was appropriate for oral sedation to be administered to OS on 26 March 2015.

9.5 The second matter which arises for consideration is whether, accepting the general principles outlined above, appropriate consideration was given to OS's specific medical history and his medication regime. Dr Bennett said that at the time he performed his assessment of OS he was aware that OS suffered from a refractory form of epilepsy, and also aware of his medication regime. He agreed that OS's ataxia (a neurological disease resulting in lack of coordination and gait abnormalities) would be of concern but explained that the use of paracetamol was unrelated to this and that its indication would remain unchanged. Dr Bennett similarly explained that both morphine and midazolam also remained indicated, but that it was possible that their dosage may require some adjustment. However, Dr Bennett explained that awareness of ataxia would most likely be addressed with observations to ensure that, following administration, OS returned to his pre-sedation baseline level of consciousness.

9.6 Further, Dr Bennett explained that it was important to be aware of the fact that OS was administered both morphine and clobazam (a benzodiazepine) but that he was not aware of any synergistic effect between the two drugs. Dr Bennett also explained that OS's intellectual impairment and global development delay were not of significance in guiding the administration of medication and that it would depend on the individual manifestation of these conditions.

[6] Exhibit 1, page 562-8 at [7.4.4].
[7] Exhibit 1, page 106.
[8] Exhibit 1, page 106-107.

9.7 **Conclusion:** The evidence provided by Dr Bennett, and the opinions expressed by Dr Dunlop, established that appropriate consideration was given to OS's medical history and medication regime prior to the prescription of oral sedation on 26 March 2015. In particular, there is no evidence to suggest that there was any contraindication for the prescription of paracetamol, morphine and midazolam. The evidence established that OS's medical history and medication regime was taken into account in prescribing the appropriate dose for each medication, and that it was intended that aspects of OS's medical history (such as his ataxia) would be addressed with observations following administration of oral sedation. This issue is discussed further below.

9.8 The final matter which arises for consideration is whether it was appropriate to use oral medication in preference to nitrous oxide in order to achieve sedation. During a previous documented hospital admission OS had been given nitrous oxide in relation to a procedure on 19 March 2015 without incident. Dr Bennett explained that there was no impediment to the use of nitrous oxide in OS's case if it was required. When asked why nitrous oxide was not considered, Dr Bennett was unable to articulate a precise reason. He explained that it was usual practice to expect that a patient would experience some discomfort associated with a dressing change and that attempts would be made to prevent this with use of medication. He explained that if opioid analgesics were insufficient then use of nitrous oxide, which is rapid onset, could be considered. Dr Bennett went on to explain that a dressing change can be unpredictable and that there are limited options to manage a patient's pain. In this context if nitrous oxide was used, without oral medication having being administered, it could lead potentially to a situation where the procedure would need to be stopped.

9.9 **Conclusion:** Although there was no apparent barrier to the use of nitrous oxide to achieve sedation in OS's case, the evidence established that it was an ordinarily accepted procedure to primarily use oral sedation, with rapid onset nitrous oxide to be used if opioid analgesia was deemed to be insufficient.

10. Was there a need for consultation with OS's treating specialists?

10.1 Dr Ardern-Holmes was asked, in retrospect, given OS's underlying conditions and prescribed medication, whether it would have been useful for there to have been a discussion between herself (or Dr Chan) and Dr Bennett as to whether consideration needed to be given to altering the oral sedation prescribed to OS. Dr Ardern-Holmes said that she did not think that such a discussion would be mandatory. Instead she explained that it would be best practice to optimise multi-team management where possible, even though this practice would not necessarily occur for every child in OS's situation.

10.2 However Dr Ardern-Holmes agreed that because of the complexity of OS's case it would have been useful to have multidisciplinary shared care. Notwithstanding, Dr Ardern-Holmes indicated that such an approach would not have added to the information already available to Dr Bennett. Dr Ardern-Holmes went on to explain that there would be merit in a discussion taking place between the clinicians who prescribed the sedative medication, and those administering the medication.

10.3 Dr Dunlop expressed the view that "*in hindsight there may have been a missed opportunity for the treating surgical burns team to discuss OS's complex polypharmacy and developmental delay with the neurology team, in order to ascertain the appropriateness of minimal sedation*".[9] However, Dr Dunlop

[9] Exhibit 1, page 107.

noted that as the exact cause of death, based upon the autopsy report, was unclear it was not possible to say whether such a discussion would have materially altered the outcome.

10.4 Dr Ardern-Holmes agreed that from looking at the medical records and the Burns Unit patient booking form, Dr Bennett had a substantial amount of information available to him regarding OS. In this context Dr Ardern-Holmes was asked about the necessity for a further form which would allow a clinician to clearly identify medication which had been prescribed to a patient. Dr Ardern-Holmes agreed such a form would be of benefit and provide the potential to maximise positive outcomes for patients.

10.5 In this regard it should be noted that since OS's death the Network has issued the *Burns Plastic Treatment Centre: Initial Consult - Dressing Change with Sedation* form. Under the heading "*Past Medical History*" the form relevantly requires a clinician completing it to identify whether or not a patient has had any previous reactions to sedatives/opioids, and whether the patient is on two or more regular medications or benzodiazepines. If such enquiries are met with affirmative responses then the form provides that the patient must be escalated to the Anaesthetic team for review.

10.6 **Conclusions:** The evidence established that whilst not mandatory, best practice suggested that a discussion ought to have occurred between the relevant anaesthetic and neurology clinicians prior to the prescription and administration of oral sedation to OS. In this regard an opportunity was missed. That said, there is no evidence to suggest that further information would have been made available to Dr Bennett other than what was already known to him. In addition it should be noted, as referred to above, that Dr Bennett appropriately took into account aspects of OS's medical history and medication regime in his assessment of what oral sedation to prescribe to OS.

10.7 Given the development by the Network since OS's death of a form which specifically directs a clinician's attention as to whether or not oral sedation may be contraindicated because of a patient's previous medications, or current medication regime, it is neither necessary or desirable to make any recommendation in this regard.

11. Were adequate observations made of OS following the administration of oral sedation, and was OS safe for discharge?

11.1 The above two issues are related and it is convenient to deal with them together. Both the Practice Guideline and the Sedation Guidelines governed the timing of oral sedation administered to patients, and observations to be performed at the time of, and following, administration.

11.2 Section 5.4 of the Practice Guideline provides that "*sedated patients must be observed in 'line of sight/line of hearing' by the accountable staff member until the patient has returned to their pre-sedation state*".[10] It goes on to provide that sedation should be regularly monitored and documented using a tool which provides for a sedation score dependent upon patient response, ranging from awake and alert to deep sedation.

11.3 Section 5.4 also relevantly provides that in the post procedure sedation period:

[10] Exhibit 1, page 562-31.

(a) Documentation of the sedation score on the chosen observation chart should occur **regularly** until **sedation score < 2**; and

(b) For **sedation score of 2** (moderate sedation), observations should be recorded **every 15 minutes** until the patient sedation level **returns to 0 or 1**.[11] (original emphasis)

11.4 Section 5.6 of the Practice Guideline sets out the recommended discharge criteria for outpatients and relevantly includes a sedation score of zero (fully awake and alert), and that the patient can sit up unaided or has returned to baseline state of function. Section 5.6 also provides that verbal and written discharge information should be provided, including, relevantly, information on specific medications administered, management of side effects, contact numbers if problems persist, and follow-up instructions.[12]

11.5 The Sedation Guidelines provided that midazolam was to be administered to a patient 20 minutes prior to a procedure, and that morphine was to be administered at least one hour prior to a procedure.[13]

11.6 In evidence, NP Waddell accepted the provisions of the Sedation Guidelines in relation to the timing of medication administration prior to a procedure. However, she explained that it was often practice within the Burns Unit to administer morphine and midazolam concurrently. When asked to identify any factor that allowed for departure from the Sedation Guidelines in this regard, NP Waddell simply explained that it was often done in practice. NP Waddell indicated that as part of such practice morphine was administered between 30 and 60 minutes prior to a procedure. In this instance, patients would be asked to remain in the waiting room so that they could be observed for the effects of sedation (whether they started to become drowsy, how they were mobilising), although such observations were not documented. It should be noted that the layout of the Burns Unit did not allow nursing staff to maintain continuous line of sight with patients who were in the waiting room.

11.7 In OS's case, NP Waddell agreed that morphine was not administered 60 minutes prior to the procedure. She said that this was because she observed that it had already started to take effect when OS was in the waiting room. NP Waddell said that she took observations just prior to the medication being administered (at about 9:35am) and again at 10:10am when the procedure concluded. She estimated that the second observations were taken about five to 10 minutes before OS and Jodi left the Burns Unit. NP Waddell said that she was unaware prior to the procedure that OS was noted to be frequently drowsy prior to his admission on 19 March 2015. On this basis, NP Waddell conceded that it was possible that she was simply observing OS's normal state when he appeared to be relaxed and calm.

11.8 In evidence Dr Bennett explained that administration of morphine one hour prior to the procedure was to allow time for the morphine to take effect, although he considered that one hour would be more than expected for morphine to reach peak concentration. Notwithstanding, Dr Bennett agreed that it was fair to say that it was not possible to be reasonably certain that the morphine administered to OS had reached its peak concentration by 10:10am. NP Waddell agreed that she

[11] Exhibit 1, page 562-32.
[12] Exhibit 1, page 562-33.
[13] Exhibit 1, page 548.

heard Dr Bennett give this evidence, but sought to explain that the effects of sedation can be different for individual patients.

11.9 Dr Bennett considered that it would be good clinical practice to take observations immediately upon a patient leaving an area of care, and at 60 minutes or more following the administration of oral sedation. Dr Bennett agreed that the two-fold purpose of such observations in OS's case would be to observe for any signs of sedation once the morphine had reached its peak concentration, and to allow time for OS to be monitored until he returned to his pre-sedation baseline.

11.10 In contrast to the evidence given by NP Waddell, Registered Nurse (**RN**) Madeleine Jacques, who witnessed the administration of morphine and midazolam to OS on 26 March 2015, said that it was her practice to split the administration of such medication in accordance with the timeframes provided for by the Sedation Guidelines. However RN Jacques acknowledged that she was aware that medications were administered concurrently on occasion. She explained that ultimately it was the decision of the administering nurse as to the timing of administration.

11.11 Dr Bennett accepted that OS's drowsiness in the Hospital coffee shop and upon leaving Hospital could be explained by the morphine reaching peak concentration leading to OS's sedation. Further, Dr Dunlop expressed the view that a period of 35 minutes was insufficient time to be certain that morphine had reached its peak concentration and to allow for a proper assessment that OS had returned to his baseline level of functioning.

11.12 **Conclusions:** The evidence established that the morphine and midazolam were signed out of the Ward Register for drugs of addiction at 9:35am and administered to OS shortly afterwards. At this time observations were taken of OS's vital signs. A second and final set of observations was taken at about 10:10am when the procedure concluded. The evidence does not allow for a specific finding to be made as to when the procedure commenced. However it can be accepted that it commenced sometime between 9:35am and 10:10am. Therefore, a period of some 35 minutes elapsed between the administration of morphine and when the final observations were made of OS. Further, Jodi and OS exited the Hospital at about 10:27am, some 52 minutes after the morphine had been administered. The timing of this event is consistent with the evidence regarding a period of 60 minutes being required for morphine to reach peak concentration.

11.13 As a result of the above, the following conclusions can be reached: firstly, the timing of administration was not in accordance with the Sedation Guidelines; secondly, there was no accepted basis for NP Waddell to depart from the requirements of the Sedation Guidelines; thirdly, it is unlikely that the morphine reached its peak concentration prior to, or even during, the procedure; fourthly, the observations at 10:10am were inadequate in the sense that they did not allow for a proper assessment to be made that OS had returned to his baseline level of functioning prior to discharge; and finally, OS's presentation in the Hospital coffee shop and after leaving the hospital is consistent with the morphine reaching peak concentration and inducing sedation.

11.14 Since OS's death, a number of practical and policy improvements have been implemented by the Network. RN Jacques explained that the general practice within the Burns Unit at present is for observations to be completed post-procedure and at least 10 to 15 minutes apart. Section 4.1 of the Network's Burns Management Practice Guideline effective from 1 March 2019 (**the 2019 Practice**

Guideline) provides that: *"The Burns Anaesthetic Fellow will review each patient and prescribe an appropriate pre-medication. The drug/s of choice are determined on an individual basis and may include an opiate such as morphine or oxycodone, along with paracetamol. Midazolam may also be used for its dissociative, anxiolytic and sedative qualities. Inhaled nitric oxide may also be administered during the burns dressing change procedure".*[14]

11.15 Appendix 4 to the 2019 Practice Guideline sets out an oral sedation drug formulary for a burns dressing change. It nominates oxycodone as the opioid analgesia (to be administered 30 minutes pre-procedure) together with midazolam as an adjunct (also to be administered 30 minutes pre-procedure). These provisions are also repeated in the Hospital's *Burns & Plastics Treatment Centre (BPTC)- CHW Practice Guideline* effective from 1 March 2019.

11.16 Relevantly, the Network has since developed a form for the BPTC which applies to initial consults involving dressing change with sedation.[15] In order for a patient to be discharged following a procedure a clinician is required to identify that the following has occurred:

(a) Two sets of observations have been completed post sedation;

(b) There have been two consecutive observations within normal limits;

(c) The patient sedation score is <1 for both sets observations (with the Sedation Score table provided for on the form itself); and

(d) The patient meets all additional discharge criteria for sedation which includes:

(i) Cardiovascular function and airway are satisfactory and stable;

(ii) The patient is easily rousable, and protective reflexes are intact;

(iii) The patient's vital signs are stable;

(iv) The patient can talk and sit up unaided (if age-appropriate);

(v) The child has returned to his/her pre-sedation level of responsiveness.

11.17 Further, RN Jacques explained that since 2016 morphine has no longer been the most frequently used sedation agent at the Hospital. She explained that this change was not due to the circumstances of OS's case. Although RN Jacques could not say precisely why the change occurred, she explained that it was a result of the Hospital's transition to electronic documentation leading to increased uniformity in the type of sedation agent used.

11.18 Finally, although Jodi was given a fact sheet in relation to caring for OS's wound, she was given no written discharge information in accordance with Section 5.6 of the Practice Guideline as to how possible side effects from administered medication was to be managed, and what to do if such symptoms were observed. In her evidence RN Jacques referred to the fact that at the time of the

[14] Exhibit 1, page 1139.
[15] Exhibit 1, pages 1200-1204.

inquest a quality improvement project was underway at the Hospital to develop a written handout to be provided to parents/carers. The handout contains information regarding medication administered to a child, signs to be aware of, and contact details in the event of an emergency. Given the timing of this location by RN Jacques it was not possible to explore this issue in further detail during the inquest. However following closing submission an invitation was extended to the Network to provide further information in this regard. The Network subsequently provided copies of a current fact sheet in relation to procedural sedation, and a proposed revised fact sheet intended to be published by the end of March 2020.

11.19 **Conclusions:** Since OS's death the Network has taken a number of positive steps to ensure that appropriate observations of a patient are taken following administration of procedural sedation. Specifically, these observations seek to ensure that a patient has returned to their baseline level of responsiveness. Further, a fact sheet has been developed to provide parents/carers with appropriate information in relation to caring for children following discharge after procedural sedation. On this basis it is neither necessary nor desirable for any recommendation to be made.

12. What was the cause of OS's death?

12.1 The inquest focused on two aspects of the autopsy in relation to the possible cause of OS's death: whether it could be attributed SUDEP, or to the effects of the oral sedation that he was administered.

12.2 In evidence Dr Pokorny said that she considered the concentration of morphine to be unusual, and that it was within a range where it could be lethal on its own. However she explained that the other medication that OS was prescribed which had sedative effects likely potentiated the sedative effects of the morphine. In this regard, Dr Pokorny said that she considered that respiratory depression may be an indirect cause of death, or to have contributed to death. She said that the features of respiratory depression would manifest themselves in a person appearing to breathe shallowly or infrequently.

12.3 Dr Pokorny explained that because the toxicology results revealed a potential morphine concentration as a possible cause of death, OS's death could not be attributed to SUDEP. Firstly, Dr Pokorny noted that SUDEP is still an uncommon occurrence in persons with epilepsy. Secondly, she explained that SUDEP is a classification when no other cause of death can be found in a person with epilepsy. Dr Pokorny went on to explain that whilst the morphine concentration in OS's case was unusually high, his death could likely be attributed to SUDEP only if there was a reasonable explanation to suggest that the morphine concentration was an artefactual result.

12.4 Both Professor Christie and Dr Kennedy agreed that the concentration of morphine identified from the postmortem toxicology was at least within the reported toxic range, and may be within the reported lethal range. Further, both experts also agreed that the postmortem blood concentration of morphine was at a higher than expected level based on a 7 milligram dose administered to OS.

12.5 The question that therefore arises is whether the concentration of morphine was an artefactual result. As to this question, Dr Kennedy and Professor Christie disagreed. Dr Kennedy posited two explanations leading him to opine that the concentration of morphine was an artefactual result and that therefore OS's death was not a drug-related death. Firstly, Dr Kennedy considered that

postmortem concentration of morphine did not represent the antemortem concentration and was *"a result of redistribution from a site of high concentration in the myocardium by [cardiopulmonary resuscitation] and possibly some redistribution from peripheral muscle"*.[16] Secondly, Dr Kennedy noted that by the time of OS's autopsy *"there would also be adequate time for bacterial invasion and, as he was a small person, there would have been considerable mechanical action on the gut walls and other abdominal organs"*.[17]

12.6 In contrast, Professor Christie opined that *"postmortem redistribution of morphine was probably minimal because femoral blood was sampled and the postmortem to autopsy interval was brief"* and therefore unlikely to account for the postmortem concentration of morphine.[18] Instead, Professor Christie considered that the dose calculation for morphine prescribed to OS on 26 March 2015 likely did not anticipate the possibility that morphine metabolism may have been impaired due to OS's impaired liver function and his prescription of valproic acid (which broadly inhibits glucuronidation enzymes in the liver).[19]

12.7 As to the question of impairment of metabolism, Professor Christie and Dr Kennedy disagreed as to whether the ratio of free morphine to morphine-3-glucuronide (**M3G**, a metabolite of morphine) indicated an impairment of OS's ability to metabolise morphine. Professor Christie considered the concentration of M3G to be *"extraordinarily low"*, that the ratio was not consistent with normal metabolism, and that the likely explanation for such an occurrence was impairment of metabolism. Professor Christie explained that following oral dosing in normal individuals, M3G concentrations are consistently much higher than free morphine, being 10 times higher at the time of peak effect (approximately one hour after dosing) and 100 times higher four hours after dosing.[20] Professor Christie therefore considered it likely that OS *"was exposed to much higher blood morphine concentrations than expected because his hepatic metabolism of morphine was impaired"*.[21] On this basis Professor Christie considered that there were two plausible explanations for impairment of liver metabolism leading to unexpectedly high bioavailability of morphine: either liver function impairment would have impaired the formation of M3G; or valproic acid, which was noted to be at supratherapeutic levels, broadly inhibited glucuronidation enzymes in the liver.

12.8 In this regard it should be noted that Dr Michael Stormon, a paediatric hepatologist who saw OS on a number of occasions between 2012 and 2014 explained that OS's *"liver function tests indicated that the ability of his liver to metabolise drugs was highly likely to be normal"*[22] and his liver function should not have impacted on the ability of his liver to manage doses of prescribed drugs. Further, Dr Stormon noted that *"any possible interaction between, or atypical effects of, medication administered to OS cannot be ascribed to his liver condition"*.[23]

12.9 Dr Kennedy disagreed with the conclusions reached by Professor Kennedy but conceded that the case was *"very unusual"*. Dr Kennedy considered that the low level of M3G could be attributed to two factors: bacterial invasion (due to the ability of bacteria to remove glucuronides) and CPR in areas with high morphine concentrations pushing central blood peripherally. In response, Professor

[16] Exhibit 1, page 120.
[17] Exhibit 1, page 120.
[18] Exhibit 1, page 42.
[19] Exhibit 1, page 42.
[20] Exhibit 1, page 42.
[21] Exhibit 1, page 42.
[22] Exhibit 1, page 393 at [9].
[23] Exhibit 1, page 393 at [10].

Christie acknowledged that *"although central blood would have been moved peripherally by CPR, the concentration of morphine in the central blood would not be expected to be greater than in peripheral blood at very early post-mortem intervals"*.[24] Further, Professor Christie considered that the likelihood of bacterial action is speculative, not conclusive, and that no other evidence of bacterial action was noted in the autopsy report.[25]

12.10 Notwithstanding, Dr Kennedy agreed that there would be an expectation that the concentrations of M3G and morphine-6-glucuronide (**M6G**, another metabolite of morphine) would be well in excess of the level of free morphine after two hours of administration. When asked to posit an explanation for this ratio, Dr Kennedy indicated that the possibility of impairment would have to be assumed.

12.11 Professor Christie and Dr Kennedy were asked whether OS's dose of valproic acid inhibited glucuronidation enzymes in the liver, leading to the increased bioavailability of morphine. Professor Christie explained that it was a speculative possibility based on some evidence that high levels of valproic acid can inhibit enzymes in the body. However, he acknowledged that no clinical evidence exists to support this possibility occurring in humans. Notwithstanding, Professor Christie sought to make the point that the failure of glucuronidation begs an answer and that the only likely explanation is some form of liver failure or the level of valproic acid. In contrast Dr Kennedy did not consider inhibition of glucuronidation enzymes from valproic acid to be a possibility, noting that any interaction between morphine and valproic acid has not been demonstrated with clinical evidence or reported in literature, and that any suggested interaction remains, at best, a hypothesis.

12.12 Notwithstanding, both experts agreed that OS's regular dose of valproic acid (and other medications) exacerbated the drowsiness and sedation from the morphine administered on 26 March 2015. The experts further agreed that such interaction could lead to respiratory depression given that OS was already experiencing the effects of sedation. On this basis the experts agreed that one possible mechanism of death in OS's case was as a result of the synergistic effects of valproic acid and morphine leading to respiratory depression. Dr Kennedy opined that in such a scenario he would have expected respiratory depression to occur at the peak concentration of morphine around 60 to 90 minutes after administration. However, Dr Kennedy conceded that there was no direct evidence as to the time of OS's death, only that he was found to be unresponsive at 5:00pm, and that death may have occurred at an earlier time. Further, Dr Kennedy was unable to identify any specific feature which allows for respiratory depression to be excluded as the cause of OS's death.

12.13 One final, related issue which the experts were asked to address concerned a matter which David told the police about shortly after OS's death. In a record of interview conducted into the early hours of the morning on 27 March 2015, David said that he had a conversation with OS sometime after they returned home from the Hospital, likely after he returned from picking JS up from school. However in evidence during the inquest David said that he could not recall having any such conversation with OS. David explained that at the time of the interview he was feeling tired, angry and emotional, and that he had suffered a number of concussions (as a result of accidental falls) since 26 March 2015.

[24] Exhibit 1, page 93.
[25] Exhibit 1, page 93.

12.14 In contrast, Jodi explained in evidence that from the time she and OS left the Hospital until she went to check on OS she could not remember whether she heard OS speak. However she said that it appeared that OS was sleeping the whole time after arriving home. Jodi said that she did not give OS any medication after leaving the hospital, and that she did not notice any seizure activity during this period.

12.15 The experts were invited to consider the version of events which David initially related to police. Dr Kennedy explained that if OS had engaged in verbal communication following administration of oral sedation then this did not indicate the likelihood of respiratory depression leading to death. However, Professor Christie considered that reasoning that there is a linear trajectory of improvement and abatement of respiratory risk is flawed. In other words, the fact that a person rouses to consciousness at some point during the trajectory does exclude the possibility that they are at risk of opioid-induced terminal respiratory depression.

12.16 **Conclusions:** The clinical findings at autopsy raised two possibilities as to the cause of OS's death. At the outset it should be noted that if the high concentration of morphine was not an artefactual result then the likelihood of SUDEP can reasonably be excluded.

12.17 The evidence established that the elevated level of morphine could not be attributed to either postmortem redistribution or bacterial action. This is because whilst it can be accepted that central blood was moved peripherally by CPR, a femoral blood sample was taken and there was only a relatively brief postmortem interval. Further, no clinical finding of bacterial action was demonstrated at autopsy. This then leads to a conclusion that the elevated concentration of morphine was not an artefactual result.

12.18 There is no evidence of liver function impairment leading to a higher than expected bioavailability of morphine. Indeed, the evidence of Dr Stormon establishes that OS's liver function tests indicated that metabolism in the liver was highly likely to be normal. Therefore, the only reasonable explanation that remains is that the valproic acid prescribed to OS as part of his usual medication regime inhibited glucuronidation enzymes in the liver, leading to increased bioavailability of morphine. This conclusion is supported by the fact that on the available evidence there is no other likely explanation to account for the disproportionately low M3G level relative to free morphine.

12.19 It follows then that the prescription of morphine on 26 March 2015 resulted in a higher than expected bioavailability of morphine, due to the synergistic interaction with the valproic acid, leading to sedation (in combination with the sedative effects of OS's other prescribed medication) and ultimately respiratory depression resulting in death.

12.20 This conclusion is supported by a number of features, namely: the fact that OS was observed to be suffering the effects of sedation in the days leading up to 26 March 2015, oral sedation was administered on the same day that OS died, insufficient observations were performed following the administration of oral sedation, OS was observed to be drowsy following administration, and the absence of any specific feature (as acknowledged by Dr Kennedy) which allows for respiratory depression to be excluded as the cause of death.

12.21 It should be noted that the account provided by David to interviewing police about having a verbal interaction with OS after arriving home on 26 March 2015 is most likely unreliable. Although a contemporaneous account would ordinarily be considered to be accurate, in this case David's account is inconsistent with Jodi's and, by David's own acknowledgment, adversely affected by his emotional state at the time of the interview, and concussive events following the interview. It should also be noted that even if this is not the case, the evidence established that even if OS experienced a conscious interval on 26 March 2015 this does not preclude the possibility of subsequent terminal respiratory depression.

13. Acknowledgments

13.1 Before turning to the findings that I am required to make, I would like to acknowledge, and express my gratitude to Ms Maria Gerace, Counsel Assisting, and her instructing solicitor, Ms Bianca Holliday-O'Brien of the Office of the General Counsel, NSW Department of Communities and Justice. Their assistance during both the preparation for inquest, and the inquest itself, has been invaluable and of the highest standard. I also thank them for the sensitivity and empathy that they have shown in this particularly distressing matter.

14. Findings pursuant to section 81 of the Coroners Act 2009

14.1 The findings I make under section 81(1) of the Act are:

Identity
The person who died was OS.

Date of death
OS died on 26 March 2015.

Place of death
OS died at Colyton NSW 2760.

Cause of death
The cause of OS's death was due to the combined effects of prescribed morphine, valproic acid and midazolam leading to sedation and eventually terminal respiratory depression.

Manner of death
OS died as a result of the synergistic effects of his existing medication regime and medication prescribed to him on 26 March 2015 leading to an unintended higher bioavailability of morphine.

15. Epilogue

15.1 OS's death is particularly tragic because of the devastating effect that it has had on both his biological family members, and also on his foster carers. At the conclusion of the evidence in the inquest OS's maternal uncle, Steven Sawle, shared some heartfelt and moving words regarding the enormous pain that he and other members of OS's family have experienced as a result of OS's separation from his biological family and his untimely death.

15.2 On behalf of the Coroner's Court of New South Wales, and the Assisting Team, I offer my deepest sympathies, and most sincere and respectful condolences, to OS's family and carers for their most painful and overwhelming loss.

15.3 I close this inquest.

Magistrate Derek Lee
Deputy State Coroner
31 January 2020
Coroner's Court of NSW