Appendix A Coroners Reports (1)

(156)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Andrea Eileen Baldock

Find that:

a) The identity of the deceased is Andrea Eileen Baldock.
b) Ms Baldock was born in Launceston on 9 June 1970 and was aged 46 years.
c) Ms Baldock died on 7 September 2016 at the Launceston General Hospital (LGH) in Launceston.
d) The cause of Ms Baldock's death was the combined effects of aspiration pneumonia due to gastroparesis following sleeve gastrectomy and ischaemic heart disease with cardiomegaly.

Background

Ms Baldock was single and resided with her mother in Latrobe. She was her mother's carer. Her medical history included obesity (on 7 April 2016 she weighed 143.8kg), Type 2 diabetes mellitus and hypertension. She was on a waiting list to undergo a sleeve gastrectomy to treat her weight loss.

Circumstances Surrounding the Death

On 22 April 2016 Ms Baldock underwent a laparoscopic sleeve gastrectomy at the LGH. The surgeon was Mr Girish Pande. The post-operative course was relatively uncomplicated. A gastrograffin swallow showed delayed emptying of the stomach which Mr Pande attributed to swelling around the surgical site. It was predicted that once the swelling went down Ms Baldock's ability to tolerate food would drastically improve. She was discharged home on 29 April with instructions to take a fluid diet for 4 weeks.

A review of Ms Baldock's medical records during the months following her surgery is instructive. A summary follows.

On 10 May 2016 Ms Baldock presented at the Mersey Community Hospital (MCH) after she collapsed and lacerated her forehead. She gave a history of decreased fluid intake over 3 weeks with vomiting. She was tired and reported decreased urine output. She was hypotensive with a significant postural fall in her blood pressure. She was treated with 1.5 litres of fluid and her blood pressure slowly rose. She was discharged before the results of

blood tests were known. Later they revealed acute renal failure and Ms Baldock was requested to return to the MCH.

On 12 May Ms Baldock re-presented at the MCH and her blood tests were repeated which showed improved renal function. However, Ms Baldock reported that her on-off vomiting persisted. On the same day she was reviewed by Mr Pande in the LGH's surgical clinic where she reported that she was able to drink 1.5 litres of fluids a day. (Her weight was recorded at 127kg indicating a fall of 16 kg at 20 days post-surgery) She was advised to return to the clinic in 3 weeks to undergo an endoscopy and dilation of the stomach tube if her intake had not improved.

On 13 May Ms Baldock visited general practitioner, Dr N Fernando. Further blood tests showed worsening renal function. Persistent vomiting continued.

On 17 May Ms Baldock saw general practitioner, Dr V Chawtur. She reported feeling dry, sipping liquids but also vomiting.

Ms Baldock saw Dr Chawtur again on 20 May. The sutures in her forehead were removed. It was considered that the gastric sleeve was too tight.

On 24 May Ms Baldock re-visited her general practice and saw Dr M Baig. Her diabetic and blood pressure medications were ceased.

On 27 May Ms Baldock again saw Dr Chawtur. He took a history of constant vomiting with a fluid intake of less than 1000 ml/day. Dr Chawtur discussed the situation with Mr Pande's registrar. It was agreed that Ms Baldock would be referred to the MCH for re-hydration.

In the afternoon of 27 May Ms Baldock presented at the MCH. Blood tests showed metabolic alkalosis, significantly abnormal renal function and an abnormal urea creatinine ratio. Further blood tests later that evening showed significant hypokalaemia (low potassium level). It was planned for Ms Baldock to be transferred to the LGH.

In the early morning of 28 May Ms Baldock was transported by ambulance to the LGH. A barium meal follow-through on 30 May showed a trace of contrast only in the duodenum and noted that fluid had been refluxing into the dilated mid and lower third of the oesophagus throughout the screening time. The following day gastroenterologist, Dr B Mitchell performed a gastroscopy. There was mild oesophageal dilation with fluid refluxing from the stomach. The stomach showed no peristaltic waves and some minor food residual. There was a muscular contraction ring in the distal stomach with functional gastric outlet obstruction. The area was dilated and a naso-duodenal feeding tube inserted. It was concluded that Ms Baldock had gastroparesis due to her sleeve gastroscopy. The plan was for tube feeding and naso-duodenal drug administration with review in 4 weeks. The fluid balance chart on 4 June showed an oral intake of 1260 ml and a vomiting volume of 1142 ml suggesting little improvement in the level of vomiting post-dilation. Nevertheless, Ms Baldock was discharged home on 5 June. It seems that at the time of discharge those medications recommended by Dr Mitchell had not been commenced.

Ms Baldock was seen again by Dr Chawtur on 16 June. It was noted that she was still dehydrated with only a slight improvement in her gastric obstruction. Four days later he saw

Ms Baldock again and referred her back to the MCH because of abnormal blood tests. At this time Dr Chawtur noted: *"Despite 1 dilation already the sleeve is so tight it is almost obstructed. She cannot hold down fluid, which tends to sit in her oesophagus and stomach sleeve + then she vomits it up. She can hold down a little custard and yoghurt. As at present she is dehydrated, and has been seriously deficient in potassium (2.3 [mmol/l]) and magnesium, and has had non-sustained ventricular tachycardia".*

At the MCH electrolyte replacement was provided with intravenous fluid. Three episodes of ventricular tachycardia were reported. It was recommended that she be transferred to the LGH but she refused notwithstanding risk of cardiac arrest. The following day Ms Baldock discharged herself against medical advice.

On 23 June Ms Baldock re-presented to the LGH to be reviewed by the surgical team. She reported an inability to eat or drink for the previous 24 hours. Blood tests showed the same abnormalities as previously. She was reviewed in the afternoon when her heart rhythm was noted to be rapid atrial fibrillation. There was a MET call an hour later when her heart rate increased to 152 bpm. She was admitted to a ward in the early evening. There were further MET calls on 27 and 28 June because of an elevated heart rate. Also on 28 June she had a gastroscopy with re-dilation of the distal stomach. The findings again indicated no movement in the stomach and no mechanical obstruction. Gastroparesis was again diagnosed. She vomited before breakfast on 29 June but was able to keep her breakfast down later that morning and was then discharged. The plan was to repeat the dilation in 2 weeks.

Ms Baldock had a follow-up visit to Dr Baig on 1 July. He noted that she was still magnesium deficient. On 7 July she attended the general surgery clinic. Her weight was noted to be 106.2 kg. She reported tolerating a soft diet but still vomited once a day. Two days later she had a further review with Dr Chawtur.

On 28 July Ms Baldock was returned to the MCH by ambulance following a fall at her home. She had severe electrolyte disturbance and renal impairment. Her c-reactive protein (CRP) was elevated as well as the white cell count. A chest x-ray showed patchy consolidation consistent with bronchopneumonia. It was noted that Ms Baldock had lost 40 kg since her surgery. In the morning of 29 July Ms Baldock discharged herself despite medical advice urging her to remain in hospital.

On 1 August Ms Baldock again attended Dr Baig complaining of feeling unwell. A diagnosis of bronchopneumonia was made and later confirmed by x-ray. She was given oral antibiotics but refused a hospital review. Three days later she was reviewed in the general surgery clinic. She reported that she was eating better with no vomiting. However, her weight was 94 kg indicating a loss of 12 kg in one month. She was referred for review by a dietician.

On 15 August Dr Baig learned of a report that Ms Baldock was not eating or drinking. He called her and she reported that she was well.

Ms Baldock re-presented to the MCH on 22 August via ambulance. She complained of weakness and drowsiness. She was unable to stand unaided. She gave a history of daily vomiting since her surgery. It was noted that she weighed 96 kg, a reduction of 47 kg post-surgery. She was admitted. The following day she was seen by a dietician who noted her to be *"severely malnourished."* It was recorded that Ms Baldock had been offered a wide range

of nutritional supplements but had declined them. In the early hours of the following morning vomit was found in her bed. She was unaware that she had vomited. Later she had an endoscopy and probably severe gastroparesis and decompression of gastric contents were then reported as diagnoses. On 25 August Ms Baldock was transferred to the LGH for continuation of her care.

Ms Baldock had a further dietician review on 26 August. It was noted that she had lost around 53 kg in less than five months with 20 kg lost since June. It was concluded that she was suffering from malnutrition and chronic dehydration. The following day a petechial rash was present. On 28 August Ms Baldock experienced oxygen desaturation. A chest x-ray showed changes in the right lung. A CT pulmonary angiogram excluded pulmonary embolism but showed evidence of innumerable pulmonary nodules.

On 30 August Ms Baldock's situation was reviewed by Mr Pande and the surgical team. It was determined that because the gastroparesis was not improving that it would be best to offer a surgical gastric bypass. This involved joining the proximal part of the stomach directly to the jejunum thereby passing that part of the stomach that was moving slowly. The following day Mr Pande met with Ms Baldock and explained to her the need for the proposed surgery. She agreed to the procedure but was keen to go home and return the day beforehand. However, Mr Pande persuaded her to remain in hospital explaining that it was desirable to improve her nutrition and hydration and to treat her chest infection. The surgery was planned for 6 September.

On 3 September a MET call was made at 6.45am when Ms Baldock's oxygen saturation had fallen to 83% and her respiratory rate was 28 bpm with associated tachycardia. A chest x-ray showed a new left lower lobe pneumonia. A naso-gastric tube was inserted to empty her stomach.

On 5 September Ms Baldock was reviewed by the anaesthetic registrar. He was concerned by her active chest infection and suggested that the surgery be postponed. Ms Baldock was unhappy with this advice and wanted to go home. Later a family meeting was organised after which Ms Baldock was persuaded to remain in hospital until after the surgery.

The following day Ms Baldock was reviewed by consultant anaesthetist, Dr Greg Best. He advised that Ms Baldock was suitable for surgery that day but only if a High Dependancy Unit bed was available for her post-operative recovery. However, such a bed was not available causing the surgery to be cancelled and re-scheduled for 9 September. Nursing notes show that later in the day Ms Baldock was emotionally labile. She was unwilling to mobilise, refused to shower and also refused to participate in physiotherapy.

In the early morning of 7 September a Code Blue call was made when nursing staff observed Ms Baldock to be visibly short of breath. CPR was commenced. However, Ms Baldock could not be revived and she was declared deceased at 3.22am.

Post-Mortem Report

This was carried out by State forensic pathologist, Dr Christopher Lawrence. In his opinion the cause of Ms Baldock's death was the combined effects of aspiration pneumonia due to gastroparesis following sleeve gastrectomy and ischaemic heart disease with cardiomegaly.

In plain English she inhaled food after her stomach stopped emptying properly following gastric surgery for weight loss. She also had heart disease.

Dr Lawrence includes this helpful comment in his report:

> *"The autopsy reveals no apparent significant mechanical problem with the sleeve procedure but appearances consistent with gastric aspiration due to a functional gastro-paresis. This led to accumulation of food in the oesophagus and aspiration of food into the lungs. Gastroparesis is associated with Type II diabetes but is not a common complication of laparoscopic sleeve gastrectomy although it can be associated with gastric surgery. Histology of the lungs shows acute on chronic aspiration pneumonia. The vomiting had also been associated with low potassium."*

Investigation

This has been informed by:

1. An affidavit provided by Michael Baldock, a brother of Ms Baldock.
2. Reports provided by Dr Baig and by Mr Pande.
3. A review of Ms Baldock's hospital records undertaken by research nurse, Ms L K Newman.
4. A report upon Ms Baldock's medical care and treatment compiled by Dr A J Bell as medical adviser to the coroner.

In his report Dr Bell included these observations:

- That sleeve gastrectomy is becoming a common form of weight loss surgery. It involves reducing the size of the stomach by converting it into a long tube. This requires stapling the stomach along its length and then removing the excess. Its advantages include low complication and mortality rates, the ease of performing the procedure, preservation of the pylorus (distal end of the stomach), maintenance of the food passage and the avoidance of foreign material. Its most common complications include bleeding, narrowing or stenosis of the stoma and leaks. Common side effects are nausea and delayed gastric emptying, each of which contribute to less fluid intake and dehydration.
- Gastroparesis involves partial paralysis of the stomach. It is also described as delayed gastric emptying. It causes food to remain in the stomach for an abnormally long time. Cardinal symptoms are nausea, vomiting, early satiety, bloating and/or upper abdominal pain. Multiple conditions have been associated with gastroparesis but the majority are idiopathic, diabetic or post-surgical.

Dr Bell also offered these opinions:

- That Ms Baldock was a suitable candidate for anti-obesity surgery.
- Gastroparesis is a recognised complication of surgery. It is difficult to treat in its severe form. There is a natural reluctance to treat it with gastric bypass surgery and the preferred course is to give non-invasive therapies time to be effective.
- In this case gastroparesis was almost certainly a consequence of Ms Baldock's surgery.

- That the prevention of Ms Baldock's aspiration was virtually impossible. Best management required a nil-by-mouth regime and use of a naso gastric drain tube and a naso-jejunal feeding tube. However, entries in the LGH notes suggest that Ms Baldock resisted their use.

Findings, Comments and Recommendations

I accept Dr Lawrence's opinion upon the cause of death.

The evidence clearly shows that from the outset Ms Baldock's post-operative recovery was beset with difficulties, largely attributable to her gastroparesis, which were unpleasant and must have caused considerable distress. Her experience confirms Dr Bell's advice that gastroparesis is a condition which is difficult to treat.

It is my understanding that the surgical remedy for gastroparesis is gastric bypass surgery which is a major procedure which has complications of its own. It is therefore prudent to treat this as a last resort and instead to employ non-invasive therapies and give them ample time to work. Quite properly this course was taken in this instance. However, the history in the 3 months following the diagnosis of the gastroparesis suggests to me that Ms Baldock's management was ad hoc, unstructured, largely unmonitored and without any direct supervision. This leads me to **recommend** that the LGH conduct a review of its practices surrounding the post-operative care of weight reduction patients with a view to having in place a structured and closely monitored plan designed to maximise the prospects of patient recovery, particularly in those instances where post-surgical complications arise.

Finally, I make the observation that this tragic case very clearly illustrates that weight reduction surgery is not risk free and should not be lightly embarked upon, particularly without having first exhausted all suitable non-invasive options.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how her death occurred and the particulars needed to register her death under the *Births, Deaths and Marriages Registration Act 1999*. I do not consider that the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me. The circumstances of the death do not require me to make any further comment or to make any recommendations.

I convey my sincere condolences to Ms Baldock's family and loved ones.

Dated: 16th day of June 2017 at Hobart in the State of Tasmania.

Rod Chandler
Coroner

APPENDIX A (CORONERS REPORTS (11)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Margaret Evalina Bugg

Find that:

a) The identity of the deceased is Margaret Evalina Bugg;
b) Mrs Bugg was born on 14 February 1926 and was aged 89 years;
c) Mrs Bugg died on 30 December 2015 at the North West Regional Hospital (NWRH) in Burnie;
d) The cause of Mrs Bugg's death was pneumonia and acute renal failure following a fractured right femur due to a fall while being transported by a stand-up lifter.

Background

Mrs Bugg was a widow, her husband having died in 2000. In around August 2009 she had right knee replacement surgery. It was not completely successful and thereafter Mrs Bugg had difficulty mobilising. As a result she was unable to safely live at home. In November 2009 she became a resident of Yaraandoo Nursing Home at Somerset ("Yaraandoo"). Her medical history also included high blood pressure, dementia with a high falls risk, Type 2 diabetes, and a stroke in 2012. She was wheelchair bound.

Circumstances Surrounding Death

In the morning of 17 December 2015 Mrs Bugg was being attended by two carers at Yaraandoo, namely Jamie Hooper and Alison Bluett. They were in the course of transferring Mrs Bugg from her room to her ensuite using a hoist or lifter when a strap became detached causing her to fall towards the floor. She suffered a laceration to her right lower leg. A registered nurse at Yaraandoo inspected the injury and it was decided that Mrs Bugg should be taken to the NWRH for assessment and treatment. At the hospital's Emergency Department Mrs Bugg's lower leg laceration was dressed. She had an x-ray of her left hip which was reported as showing no fracture. She was then returned to Yaraandoo. Over the following few days Mrs Bugg was reluctant to have food and complained of pain. On 23 December 2015 she was taken back to the NWRH. An x-ray of her right leg revealed a spiral fracture of the distal right femur. It was decided with family involvement that the best course of treatment was comfort care with palliation. Mrs Bugg died on 30 December 2015.

Post-Mortem Examination

This was carried out by State Forensic Pathologist, Dr Christopher Lawrence. In his opinion the cause of Mrs Bugg's death was pneumonia and acute renal failure following a fractured right femur due to a fall while being transported by a stand-up lifter.

I accept Dr Lawrence's opinion upon the cause of death.

Investigation

This has been informed by:

- An affidavit provided by Mrs Rosemary Milne, a daughter of Mrs Bugg.
- Affidavits provided by Mr Hooper and Ms Bluett.
- An affidavit provided by Senior Constable Dale Wylie of Tasmania Police.
- A review of Mrs Bugg's records at NWRH and at Yaraandoo carried out by research nurse, Ms L K Newman.
- A report upon Mrs Bugg's medical care made by Dr A J Bell as medical adviser to the coroner.

The investigation has focussed upon two issues. The first concerns the circumstances of Mrs Bugg's fall from the stand-up hoist on 17 December 2015 and how this came to pass. The second concerns the failure to diagnose Mrs Bugg's right leg fracture when she presented at the NWRH on that same day. I will deal with each in turn.

Upon the first issue the investigation reveals the following:

1. That Yaraandoo employs two types of hoists to transfer residents with reduced mobility. One is known as a 'stand-up lifter' which can be utilised for residents who have some capacity to weight bear. The other is known as a 'full hoist' which is needed in the transfer of residents who are unable to weight bear.

2. That on 30 October 2015 Mrs Bugg underwent a mobility assessment at Yaraandoo. The report upon the assessment comments that she: *"now requires full hoist for all transfers, unable to WB (weight bear) safely enough to use stand up lifter."* It then expressly stipulates that Mrs Bugg required a full hoist for transfers. In accord with the assessment Yaraandoo management had affixed a notice in Mrs Bugg's room reminding staff that her transfer required the use of a full hoist.

3. That both Ms Bluett and Mr Hooper were experienced extended care assistants who were familiar with Mrs Bugg and had previously assisted in her transfer within Yaraandoo.

4. That on 17 December Ms Bluett and Mr Hooper were utilising a stand-up lifter and not a full hoist to transfer Mrs Bugg to her ensuite.

5. Ms Bluett says that initially a stand-up lifter was used to assist with Mrs Bugg's mobility but over time a full hoist was introduced. It was her understanding that at the time of the incident on 17 December the practice was for a stand-up hoist to be used

during the day and a full hoist at night. She explains: *"Basically, I thought the standing hoist was still being used for Peg during the day and only the full hoist at night. I had seen other staff using the standing hoist on Peg during the day right up until the day of the incident."*

6. Mr Hooper also confirms that the stand-up lifter was used. He provided this explanation: *"On this day, Alison brought the stand-up lifter in and I didn't check what was supposed to be used. There is normally a card up on the back of the toilet door of what you're supposed to use. I just went with it, so we used that lifter. We wasn't supposed (sic) to, no. I checked the card on the back of the toilet door afterwards and it was there. The card said we were supposed to use a full hoist for Peg. At this time I wasn't sure what lift was supposed to be used. I normally check, but for some reason on this occasion I just went with it. I guess I just trusted Alison. I mean, there's people there that were using the stand-up lifter with Peg, and some just used it by themselves."*

7. The stand-up hoist is fitted with straps that are used to hold the patient in an upright position during the transfer. In this instance the evidence shows that one end of a strap became detached from the hoist on the right side. Mrs Bugg was unable to hold herself upright and lost her grip with her right hand. She fell to one side but remained suspended by the strap which remained attached on the left hand side. Mr Hooper then used the controls of the hoist to then lower Mrs Bugg to the floor. It was then that a laceration on Mrs Bugg's right lower leg was noticed and the registered nurse called.

8. There is no evidence to suggest that the strap became detached from the lifter because of a mechanical fault. This makes it probable that the detachment occurred because of operator error.

I now turn to consider the circumstances surrounding the non-diagnosis of Mrs Bugg's right leg fracture. The evidence shows:

- That in the early morning of 26 November 2015 Mrs Bugg was found on a fall-out mat besides her bed. It was evident that she had fallen from her bed. The only injury noted was bruising to both hands.

- That it was recorded on 16 December that Mrs Bugg was suffering a stage 2 pressure injury to her left hip.

- That the paramedics from Ambulance Tasmania who transported Mrs Bugg to the NWRH on 17 December noted in their Patient Care Report that they did not observe any obvious deformity or new bruising of the right leg. They further noted that Mrs Bugg had had a fall three weeks previously and had significant bruising to her left thigh and arms. (This presumably is a reference to Mrs Bugg's fall from her bed on 26 November although the Incident Report concerning that event makes no reference to injuries affecting the left thigh and arms).

- That the paramedics also recorded that Mrs Bugg complained of pain to her head, abdomen, left arm and lower right leg.

- That a discharge letter written by the NWRH states:

    - *"Thank you for referring Margaret.*
    - *She has had a fall to day.*
    - *She has a skin tear to the right shin. This has been cleaned, steri-stripped and dressed.*
    - *She has been given ADT.*
    - *She has extensive bruising about the left hip from a fall a few weeks ago.*
    - *X-ray of the hip is normal.*
    - *She will need ongoing care of this leg skin tear....."*

- That Mrs Bugg's right leg was not x-rayed.

Findings, Comments and Recommendations

It is apparent, and I find, that because of her general state of health, but particularly because of her mobility difficulties, Mrs Bugg was unsuited for transfer by a stand-up lifter and instead required a full hoist. This state of affairs was recognised by Yaraandoo by late October 2015 and as a result it had caused a card to be placed in Mrs Bugg's room reminding staff of the lifting device to be used. Notwithstanding this notice Ms Bluett and Mr Hooper employed a stand-up lifter when attempting to transfer Mrs Bugg in her room on 17 December 2015. The evidence clearly shows that when the transfer went awry, seemingly because a strap had not been properly attached, Mrs Bugg was unable to support her own weight and fell.

I am satisfied that as a result of the lifting incident on 17 December Mrs Bugg sustained the spiral fracture of her right femur which led to her re-presentation to the NWRH six days later. I am further satisfied that the leg fracture precipitated a decline in her health which led to her demise.

Both Ms Bluett and Mr Hooper assert in their respective affidavits that they were unaware that a full hoist was required at all times to transfer Mrs Bugg. Further, they contend that other staff members were utilising a stand-up lifter for Mrs Bugg and that this practice was ongoing subsequent to the mobility assessment almost two months prior to 17 December 2015. These assertions raise serious concerns relating to the adequacy of those systems which Yaraandoo presumably has in place to ensure that its staff is aware of and complies with all the requirements of all patients care plans. In these circumstances it is my **recommendation** that Yaraandoo carry out a comprehensive review of its practices with a view to reducing the risk of its staff members failing to comply with patient care plans. Mrs Bugg's unfortunate death illustrates the urgent need for this to be done and for any recommended system changes to be implemented.

It is of course regrettable that Mrs Bugg's right leg was not x-rayed and its fracture diagnosed when she presented at the NWRH on 17 December. However, I make no criticism of the

hospital for this missed diagnosis, bearing in mind that Mrs Bugg did not make any complaint of upper leg pain and also given the history of another fall three weeks previously with evident bruising of the left thigh. In these circumstances it was understandable that the clinicians focussed their attention upon the left and not the right lower limb.

In his report Dr Bell has advised me that the surgical repair of Mrs Bugg's fracture was *"high risk"* in the light of her general state of health *"with minimal chance of success."* In these circumstances he advises that the decision to implement comfort care was reasonable. I accept this advice. It follows that in my view it is unlikely that Mrs Bugg's death would have been avoided if her right leg fracture had been diagnosed at the first opportunity.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how her death occurred and the particulars needed to register her death under the *Births, Deaths and Marriages Registration Act* 1999. I do not consider that the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me.

I convey my sincere condolences to Mrs Bugg's family and loved ones.

Dated: 12th June 2017 at Hobart in the State of Tasmania.

Rod Chandler
Coroner

Appendix A CORONER'S REPORTS (III)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Simon Cooper, Coroner, having investigated the death of Wendy Crosswell,

Find, pursuant to section 28 (1) of the *Coroners Act* 1995, that:

(a) The identity of the deceased is Wendy Crosswell;

(b) Ms Crosswell died as a result of complications caused by a dislodged percutaneous endoscopic gastrostomy (PEG) tube;

(c) The cause of Ms Crosswell's death was peritonitis; and

(d) Ms Crosswell died on 12 December 2015 at the Royal Hobart Hospital, Hobart, Tasmania.

In making these findings I have had regard to the material obtained as a result of investigation into Ms Crosswell's death carried out pursuant to the provisions of the *Coroners Act* 1995. That information included affidavits dealing with formal identification of the body and life being extinct, a report of Dr Christopher Hamilton Lawrence the Tasmanian State Forensic Pathologist who performed an autopsy on Ms Crosswell's body on 15 December 2015; an affidavit from her older sister Cheryl Harris and medical records.

In addition I was provided with a report from Dr Anthony J Bell MD FRACP FCICM who reviewed the circumstances surrounding Ms Crosswell's death. Reports were also sought, and obtained, from Dr Michael Wilkinson MB Ch B FRANZCR, visiting specialist radiologist at the Royal Hobart Hospital, who reviewed, along with Dr Anning, a registrar in radiology, the results of a fluoroscopic procedure performed by Dr Anning on Ms Crosswell between 3.00pm and 3:15pm on Friday 11 December 2015.

I find, on the basis of the evidence as follows. Ms Crosswell developed pyelonephritis whilst holidaying with her family on a cruise ship in the South Pacific. She was transferred from the ship by helicopter to hospital in Brisbane on 5 October 2015.

Although gravely ill, Ms Crosswell's condition stabilised sufficiently such that she was able to be airlifted from Brisbane to the Royal Hobart Hospital on 20 November 2015.

At the Royal Hobart Hospital Ms Crosswell was assessed and appropriate therapy started. She was having significant difficulty swallowing and as a result a nasogastric (NG) feeding tube was inserted. However, because of continuing difficulties with her continuing to pull the

NG tube out, a decision was made to insert a PEG tube. This procedure was done without apparent complication on 1 December 2015.

In the early hours of 11 December 2015 Ms Crosswell was thought to have pulled at, and dislodged, the PEG tube. Notes recorded at the time indicate she was seen to be agitated and scratching at her skin. Accordingly an x-ray of the tube was ordered and carried out. The results of the x-ray were interpreted wrongly, as the tube being in the correct position.

Dr Bell's opinion, supported by the appearance of the images themselves and the opinion of radiologist Dr Stephen Broadhurst, is that the radiological imagery was wrongly interpreted and the radiological report produced as a consequence of the procedure also wrong. It is noted that Dr Wilkinson and Dr Anning both acknowledged the error that had been made in interpreting the radiological results.

The result of the PEG tube being dislodged and the fact that it was dislodged not being detected was that Ms Crosswell quickly developed peritonitis which rapidly proved fatal.

Dr Bell expressed the opinion that the handling of the situation with respect to the PEG tube was poor. He said that "the suspicion of displacement should have led to... rapid investigation of the tube position". He noted no antibiotic coverage was given and that the radiological examination was not carried out expeditiously and when it was performed was incorrectly reported.

I observe that Dr Bell's written opinion was sent to the appropriate officer at the Royal Hobart Hospital with an invitation for the hospital or the Tasmanian Health Service to make any comment. No reply was received.

I accept Dr Bell's opinion. I am satisfied that the care afforded to Ms Crosswell was not of an acceptable standard. I do note however that Ms Crosswell was very ill indeed with little or no realistic chance of recovering to any functional degree from the pyelonephritis which was the cause of her being in hospital in the first place. It follows that I cannot conclude that the substandard care she received made any change to her probable outcome.

Recommendations and Comments

The circumstances of the death of Ms Crosswell require me to **recommend** pursuant to Section 28 (2) of the *Coroners Act* 1995 that more care must be taken in interpreting and reporting upon the results of radiological examination in circumstances where PEG tubes are suspected of being dislodged or wrongly positioned.

I convey my sincere condolences to the family and loved ones of Wendy Crosswell.

Dated 20 June 2017 at Hobart in the state of Tasmania

Simon Cooper
Coroner

APPENDIX A (CORONER'S REPORTS (IV)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Maurice Cecil Nancarrow

Find, that:

a) The identity of the deceased is Maurice Cecil Nancarrow;
b) Mr Nancarrow was born at Winchester in the United Kingdom on 30 August 1938 and was aged 76 years;
c) Mr Nancarrow died on 15 August 2015 at the Royal Hobart Hospital (RHH) in Hobart;
d) The cause of Mr Nancarrow's death was multiple organ failure due to a large volume aspiration of gastric contents that occurred during anaesthetic induction for rigid cystoscopy.

Background

Mr Nancarrow was a retired salesman and manager. He resided with his wife Joan at 59 Blair Street in New Norfolk. They were married for 58 years and had five children, now all adults. In February 2013 Mr Nancarrow had a Hartmann's procedure with stoma formation to treat his recurrent diverticulitis. Relevantly, Mr Nancarrow was referred to the RHH's urology service on 19 May 2015, with symptoms of bladder outlet obstruction.

Circumstances Surrounding the Death

On 30 July 2015 Mr Nancarrow was admitted to the RHH for surgery to reverse the Hartmann's procedure. The surgeon was Mr Srini Yellapu. The surgery was not straightforward because of adhesions. The initial laparoscopic procedure was converted to open surgery. There was an inadvertent injury to the spleen which required repair. There was also an accidental serosal tear that required repair.

Post-surgery Mr Nancarrow was progressing well when reviewed in the morning of 31 July. However, that afternoon he complained of being dry and thirsty. His pulse rate rose to 115 bpm in a regular rhythm. His blood pressure was stable. Urine output was reduced. He was treated with an intravenous fluid bolus and an increased rate of intravenous maintenance fluids. He was reviewed at 10.30pm. The tachycardia had persisted. His temperature was slightly elevated. Urine volume had increased.

In the morning of 1 August Mr Nancarrow was found to have a distended abdomen. There had been minimal draining of intra-abdominal fluid. Blood tests continued to show an increased urea compared to creatinine. The albumin level had fallen to 22 mg/L and the CRP had risen to 341 mg/L. In the early afternoon Mr Nancarrow was reviewed by a surgical registrar who increased the intravenous fluid input. Nursing staff reported Mr Nancarrow to be intermittently drowsy during the day. His oxygen saturations fell to 87% despite an increased concentration of supplementary inspired oxygen. When his adult deterioration detection score (ADDS) was recorded at four and oxygen saturations continued to fall, a review by a surgical intern was requested. Crackles were heard in the lung fields. The abdomen remained distended. A chest x-ray was reported to show no pulmonary oedema. Mr Nancarrow was treated with a diuretic for fluid overload. However, he continued to deteriorate and a medical emergency team (MET) call was made at 7.45pm. Oxygen saturations had fallen to 82%. A further chest x-ray was reported to show bibasal atelectasis. A large gastric bubble was noted. The stomach was drained. At this point Mr Nancarrow was intubated as a precaution against aspiration of gastric contents and he was transferred to the intensive care unit (ICU). Standard ICU support was commenced with mechanical ventilation, inotropes, antibiotics and the insertion of a naso-gastric tube.

In the morning of 2 August a clinical examination identified four issues, namely: sepsis, over-sedation, possible adrenocortical insufficiency and respiratory failure due to bibasal atelectasis. Significant doses of corticosteroids were commenced.

Over the following two days there was some improvement in Mr Nancarrow's condition and he was transferred to the surgical ward. However, problems persisted. A sputum culture grew three different organisms consistent with aspiration pneumonia. Mr Nancarrow complained of swallowing difficulties associated with hiccups which caused abdominal pain. An ECG on 6 August showed acute changes in the septal leads consistent with myocardial ischaemia. Renal function was normal with a creatinine level of 61 micromoles/l. On 7 August Mr Nancarrow was afrebile. Inflammatory markers continued to indicate progressive sepsis. There was renal failure with a rise in the creatinine level to 172 micromoles/l. The fluid balance chart showed multiple small urine volumes.

A CT scan on 10 August was reported as: *"Conclusion: Small amount of free fluid in the left perisplenic space. No evidence of acute diverticulitis. Left sided hydronephrosis and hydroureter with possible 7.8mm left vesicoureteric junction calculus. Assessment is difficult due to significant beam hardening effect in the pelvis."*

On 12 August Mr Nancarrow was reviewed by Mr Yellapu. Left lower abdominal pain was considered to be due to renal calculi. The nursing notes made on 12 August state that Mr Nancarrow was able to mobilise with assistance, he was tolerating diet and fluids and his loose bowel actions had slowed in frequency. It was noted that his blood pressure had dropped to 90 (systolic) after his shower that day but this returned to within normal limits once he returned to bed. His white cell count was high.

During the evening Mr Nancarrow's condition deteriorated. He had become hypoxic and tachypnoeic. He had a medical review at 9.30pm. It was noted that his breathing was laboured and the jugular venous pressure was elevated. The abdomen was distended and hyper-resonant. He was treated with a diuretic. At midnight blood measurements showed

creatinine of 194 micromoles/l with other markers of acute renal failure. The haemoglobin level had fallen, the neutrophil count was high as was the platelet count. A diagnosis of fluid overload was made and a diuretic administered.

The following day a chest x-ray showed further deterioration with extensive consolidation on the right side with sub-segmental collapse and volume loss. Less extensive changes were on the left-hand side. Because of his apparent progressive sepsis (thought to be attributable to the blocked left ureter), and hydronephrosis Mr Nancarrow was booked for the placement of a double J catheter, a tube device which is placed in the ureter and is intended to permit the drainage of urine from the kidney into the bladder. However, upon induction of the anaesthetic Mr Nancarrow had a large volume aspiration that led to the procedure being aborted and Mr Nancarrow's return to ICU. Over the following days his condition slowly deteriorated and he died at 10.40am on 15 August 2015. Post-operative findings recorded by Dr Fadi Nuwayhid, the Director of Urology at the RHH, confirmed the presence of a chronic bladder neck obstruction. It had not been possible during the procedure to determine whether the left ureter was blocked.

Post-Mortem Examination

This was undertaken by forensic pathologist, Dr Donald Ritchey. In his opinion the cause of Mr Nancarrow's death was multiple organ failure due to a large volume aspiration of gastric contents that occurred during anaesthetic induction for rigid cystoscopy.

I accept this opinion upon the cause of death.

Investigation

This has been informed by:

1. An affidavit provided by Mrs Nancarrow.

2. Reports provided by Mr Yellapu, Dr Fadi Nuwayhid, and Clinical Associate Professor Andrew Turner, Director of Critical Care Medicine at the RHH.

3. A report from Dr Helen Harris, Deputy Executive Director of Medical Services at the RHH.

4. A review of Mr Nancarrow's records at the RHH carried out by research nurse, Ms L K Newman.

5. A radiology review undertaken by Dr Pip Taplin of Radiology Tasmania.

6. A report upon Mr Nancarrow's medical care and management compiled by Dr A J Bell as medical adviser to the coroner.

7. Discussion at meetings attended by myself, Dr Bell, Ms Newman, Dr Ritchey and State Forensic Pathologist, Dr Christopher Lawrence.

At this point it is necessary for me to record that Dr Taplin's report upon the CT scan of 10 August 2015 differs from the RHH report in several respects. Firstly, it describes the bladder as *"large in volume."* This contrasts with the RHH report which made no reference to the bladder size. Secondly, it takes issue with the suggestion that calculus was present in the left ureter. Instead Dr Taplin says the *"calcification described on the RHH report is unlikely to lie within the distal left ureter"* but acknowledges the difficulty in being certain *"due to the severe beam hardening artefact in the pelvis."* Finally, it records: *"In my opinion, the degree of prominence of the left renal collecting system and the left ureter is not significantly different from that on the right side."* It is my understanding that this finding is inconsistent with a diagnosis of unilateral hydronephrosis.

In his report Dr Bell expresses these opinions:

- The diagnosis of fluid overload made on 1 August was incorrect. Rather, by this date Mr Nancarrow had developed an ileus; that is a non-working bowel which had become distended with air and fluid.

- Sometime during 1 August Mr Nancarrow aspirated (inhaled foreign matter into the lungs) and then developed bilateral lung collapse which was confirmed by CT scan.

- Mr Nancarrow was appropriately treated in ICU up to his discharge on 4 August. By this time his ileus had resolved. Although not recorded at discharge by the ICU Mr Nancarrow's working diagnosis for his future management at this point should have been aspiration pneumonia.

- From 7 August 2015 Mr Nancarrow developed progressive renal failure as evidenced by an increasing creatinine level (a reading on 10 August showed a 2/3rd loss of renal function) and his need to pass multiple small volumes of urine as recorded on the fluid balance chart. However, there was a delay in attempting to determine its cause. In particular there was no consideration of bladder neck obstruction, a common cause of renal failure in elderly males.

- The CT scan of 10 August was misreported in the two respects identified by Dr Taplin. In Dr Bell's opinion the scan showed the bladder to be abnormally large.

- Had the large volume bladder been reported it should have led to consideration of a diagnosis of bladder neck obstruction. This in turn should have led to Mr Nancarrow's bladder being drained via a catheter.

- The report of the CT scan of 10 August caused the clinical teams to wrongly believe that the cause of Mr Nancarrow's renal difficulties was a blockage of the left ureter caused by renal calculus (kidney stone).

- By 13 August Mr Nancarrow was seriously ill. Chest x-ray showed significantly increased consolidation changes, especially in the right lung. This clearly evidenced aspiration pneumonia but this diagnosis was not considered. Instead a

wrong diagnosis of fluid overload was maintained and treatment with diuretics continued.

- By 13 August Mr Nancarrow required re-admission to ICU for urgent treatment of his aspiration pneumonia.

- The diagnosis of bladder neck obstruction should have been made, at least by 10 August 2013, particularly in light of the escalating creatinine levels, large bladder and the equal sized renal collecting systems.

- The surgery undertaken on 13 August would probably not have been required if Mr Nancarrow's bladder neck obstruction had been diagnosed and treated.

Findings and Comments

I accept the opinion of Dr Ritchey upon the cause of death.

I am mindful that Dr Bell has been aided by hindsight in his consideration of this case. Nevertheless, I am satisfied and accept that Dr Bell has identified a number of shortcomings in Mr Nancarrow's management at the RHH which should not have occurred and which represent a substandard level of care. These are:

1. The failure to investigate the cause of Mr Nancarrow's progressive renal failure.

2. The failure to report upon the abnormally large bladder shown on the CT scan of 10 August 2015.

3. Reporting the possible presence of calculus in the left ureter supposedly shown on the CT scan of 10 August, when the calcification more likely lay outside the ureter.

4. Misdiagnosing Mr Nancarrow to have a left ureter blocked by calculus when he in fact suffered from a bladder neck obstruction.

5. Failing to diagnose Mr Nancarrow with aspiration pneumonia, evident by 13 August, and initiating appropriate treatment.

It is my understanding that once diagnosed bladder neck obstruction can be simply and successfully treated. If this had been achieved in this instance it is likely that the surgery embarked upon on 13 August would have been unnecessary and the aspiration that eventually led to Mr Nancarrow's death would have been avoided. It therefore follows that in all likelihood Mr Nancarrow's unfortunate death would have been prevented if his bladder neck obstruction had been promptly diagnosed and treated.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how his death occurred and the particulars needed to register his death under the *Births, Deaths and Marriages Registration Act 1999*. I do not consider that

the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me.

I convey my sincere condolences to Mr Nancarrow's family and loved ones.

Dated: 19 June 2017 at Hobart in the State of Tasmania.

Rod Chandler
Coroner

APPENDIX A (CORONER'S REPORTS (V)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

IN THE MATTER OF THE *CORONERS ACT* 1995

AND

IN THE MATTER OF AN INQUEST TOUCHING THE DEATH OF ANNE MAREE WOULLEMAN-JARVIS

FINDINGS, RECOMMENDATIONS AND COMMENTS of Coroner Rod Chandler following an inquest held in Hobart on 1,2,14 and 20 February 2017.

Introduction

On 9 July 2015 Mrs Anne Maree Woulleman-Jarvis had a fall at a building site on Hobart's Eastern Shore. Eight days later she died at her home from an undiagnosed subdural haematoma. An inquest into her death has focussed upon her medical management following the fall and in particular upon the failure to diagnose and treat her fatal head injury. These are my findings arising from that inquest.

Background

Mrs Woulleman-Jarvis was aged 62 years. She resided with her husband Robert Jarvis at Fentonbury, a small township sitting adjacent to the Mount Field National Park. She had three sons from a previous relationship, all now adults. The youngest, Matthew, still resided with her and her husband.

Mrs Woulleman-Jarvis had a reasonably extensive medical history including asthma, depression and psoriasis. She also suffered from chronic pain attributable to severe arthritis which was managed with a medication regime put in place by the Chronic Pain Unit at the Royal Hobart Hospital (RHH). She used a walking stick or 4-wheeled walker for stability. Most relevantly she had had heart surgery and was fitted with a metallic aortic valve. As a result Mrs Woulleman-Jarvis took warfarin, a blood thinning medication or anti-coagulant designed to avoid blood clots. Its purpose was to help prevent the formation of blood clots on the metallic valve and to thereby reduce the risk of embolism. Bleeding is a side effect of warfarin and its use requires regular monitoring to ensure that a balance is maintained between preventing clots and causing excessive bleeding. The relevant test is known as International Normalized Ratio (INR) which is a laboratory measurement of the length of time it takes for blood to form a clot. In Mrs Woulleman-Jarvis' case the target INR was within the range of 2.5 to 3.5 seconds.

Events Leading to Death

The circumstances surrounding Mrs Woulleman-Jarvis' death are straightforward and non-controversial. They can be simply stated.

Around midday on 9 July 2015 Mrs Woulleman-Jarvis and her husband attended at a building site in Bellerive intending to collect some timber which had become available following the demolition of some sheds. After opening the gate Mr Jarvis drove their utility vehicle onto the property with his wife following behind on foot. Mr Jarvis parked the vehicle and then heard his wife shout out. He saw her lying on the ground. She had clearly fallen and struck the right side of her head on a bitumen surface. Almost immediately *"a lump and bruising as big as an egg"* appeared above her right eye. It was agreed that Mrs Woulleman-Jarvis should see a doctor. She rang the Salamanca Medical Centre where Dr Juliet Lavers worked. Dr Lavers had been Mrs Woulleman-Jarvis' general practitioner since 2008. Mrs Woulleman-Jarvis was advised that she could be seen immediately.

Dr Lavers saw Mrs Woulleman-Jarvis at 1.50pm. She gave a history of having tripped and fallen. She had abrasions to her right knee, right forehead, right elbow and right little finger.

She stated that there had not been any loss of consciousness. She appeared alert and fully orientated. She did not complain of nausea or headaches. In Dr Lavers' view Mrs Woulleman-Jarvis' presentation did not suggest that she had suffered major head trauma and she therefore did not arrange a CT scan of her brain. Mrs Woulleman-Jarvis was advised to return to the surgery if there was any deterioration in her condition or if she had any concerns. She was in particular reminded of the increased risk of bleeding because of her warfarin therapy. Her most recent INR result was 2.9.

I need to record here that it was the evidence of Mr Jarvis that at the consultation on 9 July Dr Lavers arranged for his wife to have an immediate CT scan of her brain at Calvary Hospital and that a Dr Yong advised them of the results of the scan later that afternoon. I am satisfied that this evidence is incorrect and that the true position is that the scan and the attendance upon Dr Yong occurred on a later date as will become evident as I continue this narrative.

Mr Jarvis reports that in the days following his wife experienced headaches which she self-managed with medication including, at times Endone. In the morning of 14 July the headaches were particularly discomforting. Mrs Woulleman-Jarvis contacted Dr Lavers' rooms and she was advised to attend immediately. She was seen by Dr Lavers at 11.06am. On this occasion Dr Lavers noted Mrs Woulleman-Jarvis to be alert and orientated with no obvious neurological signs or symptoms. She noted a haematoma on her right forehead but recorded a normal cranial examination. However, she was concerned by the complaint of ongoing headaches. She therefore 'phoned Regional Imaging and arranged for an urgent CT scan of the brain. The written request form completed by Dr Lavers describes the reason for the scan in these terms: *"fall few days ago trauma right forehead and zygoma....on warfarin.....headache.....exclude subdural."* At the same time Dr Lavers organised an INR test to check the coagulation status.

Dr Lavers was not working in the afternoon of 14 July as she was leaving the State for a pre-arranged holiday. She therefore organised for the results of the scan and the INR to be forwarded to her colleague, Dr Kim Yong and for him to meet with Mrs Woulleman-Jarvis to report the results and to act upon them as required.

The CT scan of the brain was carried out by a radiographer at Calvary Hospital and was then digitally reviewed by radiologist, Dr Catherine Jones. She did not see Mrs Woulleman-Jarvis in person. After her review Dr Jones completed her medical imaging report which was then dispatched to the Salamanca Medical Centre. In that document she concluded: *"Scalp haematoma overlying the right frontal bone. No skull fracture or intracranial abnormality."* Dr Jones has since acknowledged that this report was incorrect and that she had failed to observe on the imaging a *"7mm small acute extradural hematoma over the convexity of the right superior temporal gyrus, distant to the site of impact."*

Dr Yong saw Mrs Woulleman-Jarvis at 2.09 that afternoon. He advised her that the CT scan was reported to be normal apart from evidence of a sinus infection. Most particularly, there was no reported evidence of bone fractures or intracranial bleed. Despite its urgent request the INR result had not been received. (It was in fact received by the Medical Centre at 4.30pm on 14 July. The reading was 3.6. It seems Mrs Woulleman-Jarvis was not advised of this result and no steps were taken with regard to it. Dr Yong did not consider any

treatment to be necessary. He re-assured Mrs Woulleman-Jarvis and advised her to seek further help if there was any deterioration in her condition.

Mr Jarvis reports that in the evening of 15 July *"Anne was really bad."* Records show that at 9.20pm Health Direct Australia (HDA) was contacted by 'phone. This is an after-hours nurse triage service. A Joanne Williams was informed that Mrs Woulleman-Jarvis had struck her head in a fall a week previously, that she was suffering nausea and a headache and that she had taken panadeine with nil effect. In accord with its protocols HDA transferred the matter to GP Assist Tasmania (GPA) for medical triage. Dr Jeff Ayton was consulting as a triage doctor with GPA at this time. At 9.30pm he spoke to Mrs Woulleman-Jarvis by 'phone. He was informed that she had had a CT scan the previous day but she was not entirely sure of the results. It was his impression that Mrs Woulleman-Jarvis may have been suffering from a serious condition with a potential diagnosis of intracranial bleed secondary to her recent head injury whilst on an anticoagulant. Given her symptoms of nausea and headache along with her location he advised that Mrs Woulleman-Jarvis immediately call an ambulance and go to hospital for re-assessment.

I interpolate at this point to observe that the service provided to Mrs Woulleman-Jarvis, initially by HDA and then by Dr Ayton was, in my opinion, timely, professional and appropriate. It does not warrant any criticism.

Mrs Woulleman-Jarvis acted on Dr Ayton's advice and immediately 'phoned Ambulance Tasmania (AT). It dispatched a vehicle which arrived at the Fentonbury property at 10.10pm. Mrs Woulleman-Jarvis was then conveyed to the RHH arriving at 11.58pm. A paramedic made a written case description, a copy of which was left with the RHH's Emergency Department (ED). It says:

> *'Called to 63-year-old female PT who has developed a headache this evening with associated nausea post a fall onto her head and face 6/7 ago. PT has had a CT scan 24 hours ago at Calvary with nil bleeds noted but PT unsure of results exactly. PT is alert and oriented pink and perfused talkative and interactive with AT. PT has bruising to the right side of face and temporal and frontal and has periorbital bruising. PT is on warfarin. Pt has headaches in frontal region and behind her eyes that is rated at 8/10 but not relieved with IV fentanyl. PT has nausea enroute but nil vomiting noted. It does not appear that pt has improved with management. PT interactive with AT enroute. ALL VSS within normal route limits enroute to RHH for assessment. All neuro obs NAD. PEARL. Pt has taken her own endone this afternoon and panadol forte as well but these did not help headache."*

Mrs Woulleman-Jarvis was administered 10×10mcg doses of Fentanyl en route to the ED according to the AT records.

In the ED Mrs Woulleman-Jarvis was initially attended by a nurse and triaged as a category three patient requiring her to be seen within 30 minutes. The nurse noted: *"feels dizzy, frontal headache, very agitated, nauseous, last INR 2.7 6/7 ago."* That night the registrar-in-charge was Dr Rudesh Prasad. He authorised nursing staff to provide analgesia to Mrs Woulleman-Jarvis and the hospital records show that at 0.20am on 16 July Panadeine Forte

and Nurofen were administered orally followed 20 minutes later by Fentanyl delivered intravenously.

At the time of Mrs Woulleman-Jarvis' presentation Dr Bhavana Mirpuri had been working at the RHH as a medical intern since January 2015. She had had around 5 weeks' experience in the ED. Dr Prasad requested her to take a full history from Mrs Woulleman-Jarvis, to examine her and to then report back to him. Before doing so she read the case description provided by AT along with the notes made by the triage nurse.

Dr Mirpuri saw Mrs Woulleman-Jarvis at 1.10am. She learned of the fall, the use of warfarin and complaints of headaches (rated at 8/10), nausea, vomiting and light-headedness. She learned too that Mrs Woulleman-Jarvis had had a brain scan and that she had been self-medicating with fentanyl, ondansetron, endone and panadeine forte. In the history *"multiple falls"* were noted. On examination she observed Mrs Woulleman-Jarvis to be drowsy but responsive to voice and oriented. Extensive bruising was seen on the right side of the face. Blood tests were ordered (the INR was later reported as 2.7). An online check was made of the CT scan reported upon by Dr Jones and it was noted that there was not any *"skull fracture or intracranial abnormality."* She also noted that nursing staff had recorded that Mrs Woulleman-Jarvis had reported feeling more comfortable after receiving analgesia.

It was Dr Mirpuri's evidence that she then reported to Dr Prasad in these terms:

> *"I would have presented it exactly as I have documented it; as a 62-year-old lady who has presented after a fall; on Warfarin, six days ago, and now presenting with headache. I would have presented the past medical history of her having atrial fibrillation and having a metallic heart valve and I would have informed him that she is on Warfarin refer therapeutic INR and would have given him her social history about where she lived and that she was living with her husband and had some falls and was mobilising with a walker. I would have given him my examination findings and informed him that there was no positive neurological findings and he was aware that there was a CT brain that had already been performed at the private hospital. So, we discussed the report of the CT brain. He then gave me the instructions which I then documented in the plan."*

It was Dr Mirpuri's further evidence that she suggested to Dr Prasad, in light of the history of multiple falls, that Mrs Woulleman-Jarvis be admitted to the short-stay Emergency Medical Unit (EMU) for observation and for assessment later that morning. However, as Mrs Woulleman-Jarvis had reported that her headache and nausea had improved, Dr Prasad proposed that she be discharged home provided that nursing staff was satisfied that she could mobilise independently. According to Dr Mirpuri, Dr Prasad did not consider a repeat CT scan of the brain was required given the report on the scan done previously at Calvary hospital. The discharge was then proceeded with. Dr Mirpuri provided Mrs Woulleman-Jarvis with scripts for Panadeine Forte and ondansetron and advised a follow-up review by Dr Lavers.

It needs to be noted that it was Dr Prasad's evidence that he had no recollection of this conversation with Dr Mirpuri and hence was unable to verify or contradict Dr Mirpuri's account.

Sometime between 3.00am and 4.00am on 16 July Mr Jarvis was phoned by an unknown person at the RHH and advised that his wife was to be discharged and that he should attend to collect her. He resisted saying that he had an appointment in Hobart later that morning and proposed that he collect his wife then. Mr Jarvis was then told that *"they needed the bed and that she had to leave."* He then drove the 1.5 hour trip to the RHH, collected his wife and then took the return trip home. The RHH records show the actual time of Mrs Woulleman-Jarvis' discharge to be 5.35am. They also show, via her Medication Chart that 15 minutes beforehand Mrs Woulleman-Jarvis was provided with a dose of ondansetron, presumably because of ongoing nausea and/or vomiting.

At home Mrs Woulleman-Jarvis spent the day seated in a lounge chair. She had a small amount of soup for her evening meal but nothing more. Her husband had to help her to the toilet. She went to bed at around 10.00pm. Mr Jarvis checked her at around midnight and *"she appeared fine, she was still asleep."* He checked her again at around 4.00am *"and touched her, I did not feel any movement, and I knew she was gone. I checked her breathing and felt nothing."* AT records show that at 4.47am it received a request for an ambulance to attend Mrs Woulleman-Jarvis. It arrived at 5.19am but it was apparent to the attending paramedics that Mrs Woulleman-Jarvis was deceased.

Post Mortem Examination

This was carried out by forensic pathologist, Dr Donald Ritchey. His report includes this description of Mrs Woulleman-Jarvis' head and brain injuries:

> *"The autopsy revealed.....extensive contusion on the right side of the face and forehead where there was a 3cm haematoma. There was extensive bruising of the scalp confined to the right side of the head. There was no calvarian or basal skull fractures however there was a large volume partially clotted soft subdural haematoma overlying the right parietal and temporal cerebral hemispheres producing significant mass effect and leftward deviation of the cranial contents."*

In Dr Ritchey's opinion the cause of Mrs Woulleman-Jarvis' death was a subacute subdural haematoma due to a closed head injury sustained in a fall from standing height. In his further opinion significant contributing factors were a mechanical aortic valve replacement and warfarin anticoagulation.

I accept Dr Ritchey's opinion upon the cause of death.

Issues for Consideration

The circumstances surrounding Mrs Woulleman-Jarvis' death have given rise to a number of issues requiring my consideration. They are:

1. Whether Mrs Woulleman-Jarvis required a CT scan of her brain on the day of her fall?

2. Whether a brain CT carried out on 9 July 2015 would have disclosed any intracranial abnormality?

3. The interpretation of the CT scan taken on 14 July 2015.

4. The adequacy of the treatment and care provided by the RHH to Mrs Woulleman-Jarvis upon her presentation to the ED.

5. Whether the resources of the ED were sufficient to permit it to provide Mrs Woulleman-Jarvis with a proper and adequate standard of medical care?

I will deal with each of these matters in turn.

Need for a CT Scan of the Brain on 9 July 2015.

Mrs Woulleman-Jarvis was aged 62 years. She was taking warfarin because of her metallic aortic valve. She had had a fall and suffered abrasions to her right forehead but without loss of consciousness. These were matters all known to Dr Lavers when she was consulted on 9 July. Nevertheless she did not request a CT scan of the brain. This was because, following her examination, she did not consider that Mrs Woulleman-Jarvis had sustained major head trauma. Dr Mary-Anne Lancaster has been working as a general medical practitioner in Victoria since 1991. She gave opinion evidence upon Dr Lavers' management of Mrs Woulleman-Jarvis. Specific to the care provided on 9 July it was her view that it was; *"All absolutely appropriate. With no loss of consciousness there was unlikely to be any acute issue such as an acute bleed. I would have done exactly the same."*

Dr A J Bell is a specialist intensivist and nephrologist and a former Chief Medical Officer at the RHH. He advises the coroners' office upon medical issues. His opinion on this issue differs from that of Drs Lavers and Lancaster. Instead he expressed the view that it's appropriate for all anticoagulated patients who have suffered minor head trauma to have a CT scan of the brain and this applies, even in those instances where the patient has not experienced a loss of consciousness. This view, he said was based upon the results of separate studies published in Canada and New Orleans. The other evidence pertinent to this issue was a set of Guidelines published by NSW Government Health and entitled, 'Initial Management of Closed Head Injury in Adults', 2nd Ed. That document includes these references:

> At p7 in a section headed 'Indications for CT scan for Mild Head Injury;'
>
> *"Significant head injuries can occur without loss of consciousness or amnesia and that the absence of these features should not be used to determine the need for CT scanning."*
>
> And;

> *"Known coagulopathy and particularly supra-therapeutic anti-coagulation are significant risk factors for intracranial injury and that these patients should have early CT scans and be considered for reversal of anticoagulation."*
>
> At p27 under the title, 'Which Patients with Mild Head Injury Require a CT scan?' the Guidelines identify *"known coagulopathy or bleeding disorder"* as a high risk factor for patients with a mild head injury and recommends that such patients *"should have early CT scanning if available..."*

It was the express evidence of Dr Lancaster that she was unaware of any guidelines recommending the prompt CT scanning of any anti-coagulated patient who suffers a head injury. Although not expressly stated I infer from Dr Lavers' conduct on 9 July and her evidence generally that she was similarly unaware.

I am satisfied upon the evidence that Dr Lavers is an experienced, competent and caring general practitioner who was fully cognisant of the risk posed to Mrs Woulleman-Jarvis by her head injury in the context of her warfarin use. This was reflected by her preparedeness to immediately see Mrs Woulleman-Jarvis and her thoroughness at the consultation including the appropriateness of the advice to her patient. However, was she remiss in not requesting a CT scan contrary to those guidelines to which I have referred?

In my view it would be unreasonable to expect a general practitioner such as Dr Lavers, to be aware of the detail of either those guidelines referred to by Dr Bell or the NSW Guidelines, most particularly their conclusion that an anti-coagulated patient suffering a head injury requries an early scanning even if a loss of consciousness has not occurred. I therefore make no criticism of Dr Lavers for not arranging for Mrs Woulleman-Jarvis to have a CT scan of her brain on 9 July, notwithstanding her anti-coagulation status. Nevertheless, I am mindful of this comment made by Dr Bell in his evidence related to the several guidelines; *"I believe when you put neurological societies and neurosurgeons together in two different countries- invite people worldwide and come up with these opinions based on the evidence then you need to respect those opinions."* I strongly agree with this comment. This leads me to conclude that prudent practice does require compliance by general practitioners with that advice contained in the referenced guidelines, notably that all anti-coagulated persons who suffer a head injury, even without a loss of consciousness, should undergo scanning at the earliest opportunity. For this reason a copy of these findings will be provided to General Practice Tasmania and to the Royal Australian College of General Practitioners with a **recommendation** that they inform their members of the contents of the several guidelines as they relate to the prompt scanning of anti-coagulated patients with a head injury.

Likely Result of a CT Scan of the Brain on 9 July 2015

The question whether a scan carried out on the day of Mrs Woulleman-Jarvis' fall would or would not have revealed an intracranial abnormality was the subject of comment by multiple witnesses. They follow:

1. Dr Philippa Taplin is a radiologist employed in private practice. She described the haematoma identified on the 14 July scan as being *"very small, without significant mass effect."* In her opinion the haematoma would not have been visible from a scan carried out on the day of Mrs Woulleman-Jarvis' fall. She explained; *"Subdural haematomas classically bleed 'slowly,' and are often not apparent on trauma CT brains when these CTs are performed early."*

2. There was evidence in the form of a report from neurosurgeon, Mr Peter Gan. He states that the haemotoma probably would not have been detectable in a scan taken on 9 July as the amount of blood identified on 14 July was *"tiny and small"* and appeared to be relatively fresh suggesting that it had only been there *"for, at the most, three days...."* and was consistent with bleeding having started around 14 July when Mrs Woulleman-Jarvis' headaches became more severe.

3. Professor Dr. Jens Froelich is a professor of interventional and neurointerventional radiology attached to the RHH. In his view the lesion or haematoma evident from the scan on 14 July was small and difficult to see. He was unable to say whether it would have been visible on a scan performed 5 days previously.

4. Forensic pathologist, Dr Ritchey expressed the view that it was unlikely that the haematoma would have been seen on a scan taken on 9 July because they *"tend to grow so slowly."* He further explained; *"The other thing about subdural haematomas is that they start off very small and can be very difficult to see in radiology scans, and that's a problem for patients and it's a problem for doctors as well."*

In my opinion the foregoing makes it clear that in all likelihood a CT scan of Mrs Woulleman-Jarvis' brain carried out on the day of her fall would not have revealed the subdural haematoma which later became evident by scanning carried out 5 days later. This finding inevitably leads to the conclusion that the failure on Dr Lavers' part to request a CT scan when she was consulted by Mrs Woulleman-Jarvis on 9 July was not a factor causative of her death.

The CT Scan of 14 July 2015

I have already recorded Dr Jones' acceptance that her report upon this scan was incorrect in that it did not identify an intracranial haematoma. This concession is compatible with the evidence of Professor Dr Froelich and Dr Taplin, both of whom reviewed the imaging and confirmed that it identified an intracranial haematoma. In their respective reports it is described by Professor Dr Froelich as an *"Acute ~ 6×12 mm diameter right sided fronto-temporal intracranial convex shaped type 1 epidural ('extradural') hematoma"* and by Dr Taplin as a *"Small, acute extra-axial haematoma right temporoparietal region. No significant mass effect."*

I am satisfied upon the evidence that there were factors which militated against the seriousness of Dr Jones' misreporting and provide some explanation for its occurrence. These are:

- The small size of the haematoma making it difficult to see. It's pertinent to observe that Dr Taplin acknowledged that because she was requested to review the scan for the purpose of this inquest that she was alert to the likelihood that she was looking for *"something probably subtle"* and that she may not have detected it if she had viewed the scan in the course of her day-to-day practice.

- Of the approximately 500 images created by the scan only three identified the haematoma.

- The presence of a right frontal scalp haematoma, the underlying area of which Dr Jones may have overly focussed upon relative to other regions of the brain. In her written report Dr Jones advanced this factor as the possible explanation for her failure to observe the intracranial haematoma.

- The work demands upon Dr Jones on 14 July. She described that day as *"extraordinarily busy"* because she and a colleague were required to manage a workload ordinarily undertaken by four radiologists.

Dr Jones' failure to identify the intracranial haematoma on 14 July leads to the question whether this error played a role in bringing about Mrs Woulleman-Jarvis' death. Counsel for Dr Jones has urged me to answer this question in the negative. I am unable to do so.

When Dr Yong was assigned the task of seeing Mrs Woulleman-Jarvis in the afternoon of 14 July he was aware of her referral for a CT scan to exclude an intracranial bleed upon the background of a fall causing head trauma 5 days previously and her use of warfarin made necessary because of her aortic valve replacement. Although he gave no direct evidence upon the matter I have no doubt that Dr Yong, as a qualified medical practitioner, would have appreciated the serious implications for Mrs Woulleman-Jarvis if the CT scan indicated an intracranial bleed and he would have either sought the further advice of Dr Lavers, if she was contactable, or more probably would have acted upon his own initiative. In either event it is in my view certain that Mrs Woulleman-Jarvis would not have been sent home to Fentonbury as did occur but rather would have either been directed to attend the ED at the RHH or alternatively would have been directly referred to a neurosurgeon. If the former option was taken it is also in my view certain that the medical staff in the ED, when made aware of a CT scan showing an intracranial bleed, would have ensured that Mrs Woulleman-Jarvis was referred to the neurosurgical unit. All of this leads me to be satisfied that if the CT scan of 14 July had been accurately interpreted by Dr Jones, Mrs Woulleman-Jarvis would have been placed under the care of a neurosurgeon by the late afternoon or early evening of 14 July 2015. What would then have transpired?

It was the evidence of Professor Dr Froelich that Mrs Woulleman-Jarvis' injury was treatable on 14 July and he would have arranged for her instant neurosurgical review once an intracranial bleed was identified. Similarly, Mr Gan considered the injury to be treatable. The first step was to reverse the anticoagulation. After this surgery to evacuate the blood could have been undertaken, either in the form of a craniotomy or by burr holes. Her likely chance of survival was in the range of 80 to 90%.

In my view the evidence makes it clear that if Mrs Woulleman-Jarvis' intracranial bleed had been identified by the scan on 14 July she would have been promptly referred for neurosurgical management with her injury, most probably, being successfully treated and her death prevented. In other words the first opportunity for Mrs Woulleman-Jarvis' death to have been avoided was lost because of the misreported scan and it is in this context that I consider Dr Jones' conduct to have been a factor which enabled or contributed to death.

The Adequacy of the Treatment and Care Provided by the RHH

It was the unequivocal evidence of Dr Bell that Mrs Woulleman-Jarvis' presentation at the ED mandated a repeat CT scan of the brain, notwithstanding the apparent negative scan reported on by Dr Jones 1.5 days previously. His report states; *"A patient on warfarin is vulnerable to cerebral haemorrhages including subdural haematoma. The exclusion of a subdural haematoma the day before does not exclude that a bleed has occurred today. Changing signs means changing pathology."* In a similar vein it was the evidence of Mr Gan that *"her condition with such severe headaches, she should really have been re-scanned even though she had the scan done two days prior. She certainly should have been kept in hospital for observation and she should never have been sent home."*

Dr Emma Huckerby is a specialist in emergency medicine and the Director of the ED at the RHH, a position which she held at the time of Mrs Woulleman-Jarvis' death. It was her express evidence that Mrs Woulleman-Jarvis was not appropriately treated and that she required a repeat CT scan of the brain which should have been done at around 2.00am after Dr Mirpuri's assessment or later that morning. In the latter instance she should not have been sent home but rather kept in the hospital, ideally in the EMU.

Further, it was the view of Dr Prasad, expressed retrospectively, that: *"If I had known all the information I would have (ordered a CT scan). If it was presented as a – this lady is Warfarinised, has a headache, has had a negative CT scan but is just not settling with industrial strength opiates, that's a kind of a red flag, and I would have scanned."*

In my view the evidence clearly establishes that the level of care provided by the ED was sub-standard. Her complaints of worsening headaches unresponsive to self-administered pain relief, nausea and lightheadedness upon a background of a fall causing head trauma for an anticoagulated patient mandated a repeat CT scan, notwithstanding the previous scan. It also mandated Mrs Woulleman-Jarvis being kept under observation in hospital until the scan was undertaken and the results known.

I am further satisfied that if Mrs Woulleman-Jarvis had a repeat CT scan of her brain on 16 July it would have clearly demonstrated that same haematoma, previously overlooked by Dr Jones. This discovery would have led to Mrs Woulleman-Jarvis being admitted and urgently referred for neurosurgical assessment. In turn it is likely that she would have required that same surgical intervention that Mr Gan indicated would have been appropriate if her haematoma had been detected earlier. Even at this later stage I am satisfied, accepting Mr Gan's further evidence, that Mrs Woulleman-Jarvis' prospects of surviving her injury would have been *"high"* if the correct diagnosis had been made on 16 July 2015.

The foregoing leads to the obvious question; ie. how did it come about that Mrs Woulleman-Jarvis was administered a sub-standard level of treatment that directly facilitated her death? In my opinion there are several factors which provide an explanation. They are:

- Dr Mirpuri's inexperience. She had graduated in 2014 and on 15/16 July was the most junior medical officer in the ED having worked there for just 5 weeks as part of her training on rotation. No criticism is made of the history she obtained or of her examination. However, she made, as Dr Huckerby observed, the *"cognitive error"* of being falsely re-assured by the normal head scan reported by Dr Jones. I am satisfied that this, along with a failure to appreciate that the apparent improvement in Mrs Woulleman-Jarvis' symptoms was attributable to the medication masking the seriousness of her underlying condition, caused her to wrongly focus on the fall history as the most serious aspect of her presentation. I note that it was the fall history which was the basis for Dr Mirpuri's recommendation, made to Dr Prasad, that Mrs Woulleman-Jarvis be kept for observation and not because of a possible brain injury.

- The failure on Dr Prasad's part to direct that Mrs Woulleman-Jarvis have a repeat CT scan. I have set out above Dr Mirpuri's evidence upon the information she provided to Dr Prasad following her examination of Mrs Woulleman-Jarvis including advice of the recent head trauma and her use of warfarin. I accept that this information was in fact relayed to Dr Prasad and it raises the question why it did not cause him to order a repeat scan particularly in light of his experience and seniority within the ED. In my view 'pressure of work' is the very obvious explanation. Later in these findings I comment in more detail upon the staffing structure in place for the night shift on 15 July 2015 and its effect upon the ED's capacity to properly care for its patients. At this point I make the finding that because of the staffing structure in place Dr Prasad was working under significant work pressure and this, in my view, led to him failing to fully digest or process all the information presented to him by Dr Mirpuri. Instead it caused him to adopt, without close analysis, the conclusion of his most junior colleague that the real problem related to Mrs Woulleman-Jarvis' fall history. It was in this context that he authorised her discharge once nursing staff was satisfied that she was able to safely mobilise.
  Before leaving this subject it's relevant for me to observe that Dr Huckerby, when asked to comment on those factors which she considered pertinent to the poor level of care provided by the ED made this comment; *"I think that there is also an error from the perspective that the intern should have been able to be more closely supervised but I believe that it was unreasonable for one registrar to have to supervise so many staff and provide care in that particular environment, especially considering there were up to 15 admitted patients in the emergency department at the time who they would also be responsible for, so I think the workload definitely comes into it."*

The Adequacy of the RHH Resources

A consideration of the capacity of the RHH to provide proper care to Mrs Woulleman-Jarvis requires me first to set out the relevant evidence. Much of it was provided by Dr Huckerby. The evidence shows:

- In accord with the arrangements in place at the time of Mrs Woulleman-Jarvis' death the ED medical staff comprised an Emergency Registrar-in-Advanced Training, namely Dr Prasad, an Emergency Registrar-Junior, a Resident Medical Officer and an intern, namely Dr Mirpuri. Separately there was a consultant in emergency medicine available on-call.

- A night shift at the ED begins at 11.00pm and continues to 8.00am the next day. When the shift began on 15 July the incoming medical staff took over responsibility for 21 patients who were receiving care in the ED and a further 7 patients who were accommodated in the EMU. During the course of the shift a further 35 patients required assessment and treatment.

- For the 12 months to February 2017 the average number of presentations to the ED night shift was 25 patients. This indicates that the 35 presentations on 15 July 2015 represented a 40% increase on the nightly average and was described as a *"a busy night."*

- Documents presented at the inquest and explained by Dr Huckerby show that in the months prior to Mrs Woulleman-Jarvis' death she had very real concerns related to the capacity of her Department to cope with its workload. On 4 March 2015 she had a meeting with Mr Matthew Daly, the RHH's Acting Chief Executive Officer. At that meeting she raised her concerns re, inter alia, ED patient flow and medical staff numbers. On 17 March Dr Huckerby forwarded to Mr Daly and other senior hospital personnel a briefing paper entitled 'Royal Hobart Hospital Emergency Department Medical Staffing.' The expressed aim of that document was to record a comparison of medical staff numbers at the ED of the RHH with staff numbers at four other comparative hospitals in Australia with a view to assessing the adequacy of the staffing levels at the RHH's ED. A summary in the document sets out its conclusions, namely;

    a) *"Current levels of medical staffing at the RHH ED are inadequate with the gap being that of 10 to 15 FTE non-specialist doctors and 3 specialist shifts a week.*

    b) *"This gap in staffing is significantly impacting on the capacity of the ED to provide safe and timely emergency care to patients attending the RHH.*

    c) *"The gap needs to be addressed urgently as current trends in patient attendances and complexity at the RHH (and at all major referral hospitals in Australia) show that ED workload is going to continue to increase."*

- On 21 April Dr Huckerby was requested to provide a formal business case including her proposed strategies. This was done and distributed on 26 August 2015. It

recommended replacing the existing Model of Care with a Team Based Care Model. To facilitate this it recommended; *"....to increase the number of non-specialist medical staff in the RHH ED by the appointment of an additional five FTE registrars, five FTE residents and five FTE interns."* It asserted that such an increase in medical staff would enable a Team Leader to assess and supervise every patient's care, a task that was not possible under the existing Model because it required the senior practitioner to assume a substantial patient load. In her business case Dr Huckerby asserted the existing Model to be *"unsafe"* as it meant *"many patients leave the ED without being reviewed by a senior doctor"* and that it enabled patients suffering a serious illness but *"presenting with features suggestive of a more minor condition (to be) misdiagnosed by the junior medical staff with insufficient experience to recognise the more serious condition."*

- Prior to completion of her business case Dr Huckerby was moved to write to the RHH's Chief Executive Officer (by this time Mr Craig Watson held this position). Her letter is dated 4 June 2015 and she reports; *"The current state of the ED....overcrowding, increasing attendances and staffing that does not comply with national benchmarking.....is causing both potential and actual patient harm. This is unacceptable."* The letter then provides a detailed account of the difficulties faced by the ED and includes this concluding remark; *"The current situation is untenable in terms of the level of risk to our patients, our community, our staff and the organisation."*

- In late 2015 Dr Huckerby learned that approval had been given for the appointment of an additional five registrars and three to five extra interns to the ED. For the ED's night shift this meant that it could be manned by three registrars and a junior doctor, whether it be a resident or an intern. At that time Dr Huckerby considered this a satisfactory outcome.

The foregoing evidence, which I accept, leads me to positively find that in July 2015 the ED's staffing levels did not comply with national bench-marking and exposed patients to the risk of *"actual patient harm."* This is a matter of grave concern, firstly because the RHH's senior management permitted this state of affairs to materialise without an appropriate response and secondly, because it took almost nine months for the situation to be addressed after Dr Huckerby brought it to management's notice in the starkest of terms. Regrettably and tragically Mrs Woulleman-Jarvis was in that nine month period a victim of the *"actual patient harm"* forewarned by Dr Huckerby.

Dr Prasad was still employed as a registrar in the ED in late 2015 when the new staffing structure was put in place. It was his evidence that this enabled him to personally see each of the patients who had been assigned to an intern. He said; *".....I see every intern's patient, because with more seniors, there's less demand for myself to have my own patient load, which means I can actually float and provide more consultation."* I accept this evidence. It, along with the evidence of Dr Huckerby concerning workload as a factor impacting on Dr Prasad's work performance on 16 July 2015, leads me to conclude that if the new staffing structure had been in place at that date it is likely that Dr Prasad would have had the time to personally examine Mrs Woulleman-Jarvis and to have more closely considered her full

clinical picture. This, I am sure would have led to a decision for a re-scanning to be done which, as previously explained would have led to the intracranial haematoma being detected and life-saving treatment initiated. It thus follows that the staffing insufficiencies in place in the ED on 15/16 July 2015 was a factor causative of death.

Summary

In my opinion Mrs Woulleman-Jarvis was the victim of a cruel trifecta. First, was the failure on Dr Jones' part to identify the intracranial haematoma on the CT scan of 14 July 2015. Second, was the sub-standard level of care provided by the ED. Third, was the medical staff insufficiencies in the ED which adversely impacted upon the Department's capacity to provide its patients with a proper level of care. In my further opinion, Mrs Woulleman-Jarvis' death would, in all likelihood, have been prevented had any one of these factors not arisen.

In accord with s28(1) of the Coroners Act 1995 I make these findings:

a) The identity of the deceased is Anne Maree Woulleman-Jarvis.

b) Death occurred in the circumstances set out in these findings.

c) The cause of Mrs Woulleman-Jarvis' death was a subacute subdural haematoma due to a closed head injury sustained in a fall from standing height.

d) Death occurred on 17 July 2015 at 59 Hall Road in Fentonbury.

Concluding Comments

I extend to Mr Jarvis and to his family my sincere condolences for his wife's death and I trust that this inquest has been of some benefit to all of them in coping with it.

Finally, I wish to record my thanks to counsel-assisting, Ms Allison Shand and to coroner's associate, Ms Katie Luck for their excellent work, both before and during the course of the inquest.

Dated at Hobart this: 17th day of May 2017.

Rod Chandler
Coroner

APPENDIX A (CORONERS REPORTS (VI)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Robert Neil Handasyde

Find that:

(a) The identity of the deceased is Robert Neil Handasyde;

(b) Mr Handasyde was born on 16 January 1929 and was aged 86 years;

(c) Mr Handasyde died at the North West Regional Hospital (NWRH) in Burnie on 27 June 2015;

(d) The cause of Mr Handasyde's death was sepsis with septic shock, due to urinary tract infection, pyelonephritis (infection in the kidney) and focal areas of infection in the prostate gland.

Background

Mr Handasyde and his wife Noreen resided in Penguin. He was a retired farmer. His medical history included coronary artery bypass grafting in 1987 and an old right frontal lobe infarct revealed by CT scan in 2009. In about September 2014 he ceased all medications preferring to only take natural health products.

Mr Handasyde first attended general practitioner, Dr Elvis Igbinovia, on 26 March 2015. He reported having had ongoing dizzy spells for about one month. They usually lasted for about five minutes and resolved spontaneously. Mrs Handasyde, who also attended with her husband, expressed her concerns about her husband's urinary incontinence and nocturia. She reported that he had been under the care of an urologist some years previously for *"folds in his bladder."* An electrocardiogram was interpreted by Dr Igbinovia to show atrial fibrillation. Mr Handasyde was referred to a cardiologist and for a Holter monitor investigation. He was also referred to an incontinence clinic.

On 31 March Mr Handasyde had two further dizzy spells which he reported to Dr Igbinovia. He was prescribed clopidogrel and indapamide. However, on 14 April Mr Handasyde reported feeling better and that he had not had any dizzy spells during the preceding week.

On 24 April the Holter monitor recording was carried out. It was analysed three days later. However, the report upon the monitoring was not forwarded to Dr Igbinovia until 19 May. He considered the report to be *"unremarkable."*

From 30 April to 3 May Mr Handasyde was a patient of the NWRH having presented at the Emergency Department (ED) with multiple episodes of bright red rectal bleeding. A colonoscopy showed pan-colonic diverticulitis. He continued to receive clopidogrel and indapamide. During his admission he suffered an episode of urinary retention. A bladder scan showed 700ml of urine in the bladder. A urinary catheter was inserted and 450ml of urine was drained. Over 4 hours a total of 1150ml was drained. On 30 April an ECG was performed but was not interpreted.

On 11 May physician, Dr R A Watts, performed an echocardiogram. It seems likely that at this time he was unaware of the results of the Holter monitoring.

Mr and Mrs Handasyde attended Dr Igbinovia on 20 May and 11 June. On each occasion they expressed concern about his persistent use of incontinence pads at night. On the latter occasion Mrs Handasyde reported having measured her husband's urine output for one night and that he had passed 400mls.

On 15 June Mr Handasyde again presented at the ED of NWRH having had a fall at home several days previously. Respite arrangements were then organised and on 16 June 2015 Mr Handasyde was admitted to the Eliza Purton Nursing Home (EPNH) in Ulverstone.

Circumstances Surrounding the Death

At EPNH Mr Handasyde complained from the outset of low back pain and dysuria (difficulty passing urine and pain when doing so). He was seen by Dr Igbinovia on 17 June and started on topical Voltaren. The next day he had a urinalysis which indicated a likely urinary tract infection and Dr Igbinovia was advised the details. Dr Igbinovia then directed that Mr Handasyde be administered oral Trimethoprim 300mg daily.

On 19 June it was recorded that Mr Handasyde had been groaning in pain overnight. He was given paracetamol. During the day his temperature was recorded at 40.4°C, blood pressure of 145/55 mmHg, respiratory rate of 19bpm and heart rate of 78bpm. It was noted that Dr Igbinovia had been expected to attend to review Mr Handasyde but did not do so. At 6.00pm the Trimethoprim was commenced.

Mr Handasyde's condition remained relatively unchanged on 20 June. However, the following day it was noted that the simple analgesia was not managing his pain. He was reviewed by Dr Ian Emmett who prescribed Endone 5mg. It was recorded that later that day he seemed more settled. The administration of Endone continued and by about midday on 23 June Mr Handasyde had become unresponsive and opiate toxicity was suspected by EPNH's nursing staff. Ambulance Tasmania was called and paramedics administered IV naloxone. Mr Handasyde was then transported to NWRH. In the ED was observed until 3.00pm. He remained alert and comfortable. His Endone dose was reduced and he was discharged back to EPNH arriving at about 4.00pm. The following day he was reviewed by

Dr Igbinovia who directed that the Endone be withheld for 24 hours with the EPNH nurse recording that Dr Igbinovia did not believe Mr Handasyde to be in pain and that he was *"delirious and confused."*

In the morning of 25 June staff at EPNH reported to Dr Igbinovia that Mr Handasyde was again unresponsive. He then spoke to Mrs Handasyde after which he directed that Mr Handasyde be transferred back to NWRH. This occurred via ambulance at about 1.00pm. At the hospital Mr Handasyde's condition was assessed to be terminal. Medical staff discussed his situation with family members and it was agreed to implement comfort care. Mr Handasyde's condition continued to deteriorate and he died at about 8.25am on 27 June 2015.

Post-Mortem Examination

This was carried out by pathologist, Dr Terry Brain. In his opinion the cause of Mr Handasyde's death was sepsis, prostatic and renal severe inflammation.

Investigation

This has included consideration of:

- An affidavit along with a handwritten chronology provided by Mrs Handasyde.
- Medical reports provided by Dr Igbinovia and Dr R A Watts.
- A review of Mr Handasyde's records at NWRH and at EPNH carried out by Research Nurse, Ms L K Newman.
- A report made by Dr A J Bell as medical adviser to the coroner.

In his report Dr Bell has made these observations:

- Mr Handasyde's history made evident on 26 March 2015 clearly suggested that he had a bladder neck obstruction with over-flow incontinence. He required urgent referral to an urologist for assessment. It would not have been inappropriate to have made a contemporaneous referral to an incontinence clinic.
- The symptoms of bladder neck obstruction with over-flow incontinence can be readily and simply relieved by insertion of a urinary catheter or supra-pubic catheter, either of which would have provided immediate relief. A catheter can be left in situ to provide ongoing symptom relief.
- In addition bladder neck obstruction can be treated with urological surgery. This was an option for Mr Handasyde.

- By inserting a catheter and/or undergoing urological surgery Mr Handasyde would have been relieved of having a full bladder and hence would not have been exposed to the risk of serious infection.

- From 21 to 23 June 2015 Mr Handasyde received 20mg of endone within 48 hours per 5mg doses. The usual starting dose for endone is 2.5 to 5mg orally. For patients with renal failure the blood levels after a single dose are doubled. This occurred in Mr Handasyde's case because of his renal failure. It caused his opiate toxicity and coma which required his visit to hospital on 23 June 2015.

- That in his opinion Mr Handasyde had septic shock by 19 June 2015 as evidenced by his temperature of 40.4°C and a blood pressure systolic falling by 40mmHg. He required emergency transfer to hospital and commencement of intravenous antibiotics. The use of an oral antibiotic was incorrect practice.

- When Dr Igbinovia did not attend to review Mr Handasyde on 19 June EPNH should have, on its own initiative, arranged for his hospitalisation. The seriousness of his condition required this to occur.

- That in his opinion the medical staff at NWRH overly focussed upon Mr Handasyde's rectal bleeding when he presented on 30 April 2015. Instead focus should have been on the patient as a whole. This should have led to a full history being obtained which should have alerted the medical staff to Mr Handasyde's urinary obstruction and his immediate need to see an urologist.

- That in his opinion the failure to make a timely diagnosis of Mr Handasyde's urinary obstruction and to implement appropriate treatment led to Mr Handasyde developing an infection of the retained urine leading to pyelonephritis septicaemia and leading in turn to death.

- Had Mr Handasyde's urinary condition been promptly and appropriately treated it is likely that his death would have been avoided.

- That the cause of death as stated by Dr Brain, could be expansively described as sepsis with septic shock, due to urinary tract infection, pyelonephritis (infection in the kidney) and focal areas of infection in the prostate gland.

Findings, Comments and Recommendations

It is apparent, and I so find, accepting the description provided by Dr Bell, that the cause of Mr Handasyde's death was sepsis with septic shock, due to urinary tract infection, pyelonephritis (infection in the kidney) and focal areas of infection in the prostate gland.

I am satisfied, accepting the opinion of Dr Bell, that Mr Handasyde was suffering from a bladder neck obstruction when he first attended Dr Igbinovia on 26 March 2015. Regrettably the diagnosis was not made at that time or on later occasions when Mr Handasyde

consulted his general practitioner or indeed following his admission to NWRH on 30 April 2015. In the result Mr Handasyde's urinary condition went untreated leading to infection and death. This was a tragic outcome which, in all likelihood would have been avoided if Mr Handasyde had received proper medical care.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how his death occurred and the particulars needed to register his death under the *Births, Deaths and Marriages Registration Act 1999*. I do not consider that the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me.

I convey my sincere condolences to Mr Handasyde's family and loved ones.

Dated: 8 May 2017 at Hobart in the State of Tasmania.

Rod Chandler
Coroner

APPENDIX A (CORONER'S REPORTS (VII)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Marlene Jean Harper

Find, pursuant to Section 28(1) of the Coroners Act 1995, that:

(a) The identity of the deceased is Marlene Jean Harper;

(b) Mrs Harper was born in Hobart on 4 September 1942 and was aged 72 years;

(c) Mrs Harper died at the Royal Hobart Hospital ('RHH') in Hobart on 21 June 2015;

(d) The cause of Mrs Harper's death was multiple bilateral pulmonary infarcts caused by multiple pulmonary thromboemboli due to deep vein thrombosis. Significant contributing factors were chronic lymphocytic leukaemia (CLL) and atherosclerotic vascular disease.

In making the above findings I have had regard to the evidence gained in the investigation into death. The evidence comprises the police report of death; an opinion of the forensic pathologist as to cause of death; relevant police and witness affidavits and medical records.

Background:

Mrs Harper resided alone at 50 Bowden Street in Glenorchy. She was retired but previously had worked for many years as a waitress. She had one child, namely Leanna Helen Payne. Her known medical history included CLL.

Circumstances surrounding the death:

On 16 March 2015 Mrs Harper consulted General Practitioner Dr Stefan Visagie complaining of a sore right leg. She was diagnosed with a superficial thrombophlebitis and a bandage was applied to her right thigh. One week later she was reviewed by her regular General Practitioner, Dr Don McLeod. He confirmed the diagnosis and advised that Mrs Harper take aspirin for several days. Dr McLeod saw Mrs Harper again on 24 April and noted that her leg was better. He next saw her on Thursday 18 June. The Patient Health Summary completed by Dr McLeod at the time of this consultation states; "*Pain in the leg thrombophlebitis.*" In a report provided by Dr McLeod following Mrs Harper's death he has provided this account of that attendance:

> *"On examination (Mrs Harper) was tender over the medial aspect of her thigh just above the knee but although there was tenderness there was no redness on this occasion but I thought it might be very early on. She did not have any pain below the knee or calf tenderness."*

He also noted that she "*looked off colour but denied cough or any chest symptoms.*" He ordered a full blood count and some inflammatory markers and planned to review Mrs Harper the following Monday.

In the afternoon of Saturday 20 June Ms Payne visited her mother intending to do her house work. Her mother told her that "*I feel absolutely lousy.*" Ms Payne offered to take her to the doctor or to hospital but she declined.

At about 5.00am the next day Ms Payne received a telephone call from her mother who said that she could not breathe. Ms Payne and her partner immediately drove to Mrs Harper's home. They found her lying across her bed. She appeared not to be breathing. CPR was commenced on instructions over the phone from Ambulance Tasmania. Shortly afterwards an ambulance arrived and Mrs Harper was then conveyed to the RHH. CPR was continued but Mrs Harper could not be revived. Her time of death was recorded at 7:30am.

Post Mortem Examination:

This was carried out by forensic pathologist, Dr Donald Ritchey. In his opinion the cause of Mrs Harper's death was multiple bilateral pulmonary infarcts caused by multiple pulmonary thromboemboli due to deep vein thrombosis. Significant contributing factors were CLL and atherosclerotic vascular disease.

In his report Dr Ritchey provides this helpful explanation:

> *"Pulmonary thromboemboli (PTE) begin as blood clots (thrombi) in the deep veins usually of the legs (deep vein thrombi, DVT). When these dislodge they travel through the venous vascular system (embolise) and right side of the heart and become impacted within the arteries supplying blood to the lungs (PTE). When small and medium sized, these pulmonary thromboemboli are often experienced as shortness of breath and chest pain. The lung within the distribution of these blocked arteries dies (pulmonary infarcts). When large clots embolise, the impacted thromboemboli occlude blood flow to the lungs resulting in respiratory failure and cardiac arrest.*
>
> *Individuals with CLL are at increased risk of developing deep vein thrombi and their complications including PTE."*

Investigation:

This has included the following:

1) Consideration of an affidavit provided by Ms Payne.

2) Consideration of a report provided by Ambulance Tasmania.

3) Consideration of a report provided by Dr McLeod with accompanying records.

4) A review of Mrs Harper's records undertaken by research nurse Ms Libby Newman.

5) Consideration of a report compiled by Dr A J Bell as medical adviser to the Coroner.

6) A meeting to review the investigation attended by myself, Ms Newman, Dr Bell, Dr Ritchey and State Forensic Patholgist, Dr Christopher Lawrence.

The focus of the investigation has been upon the adequacy of Mrs Harper's medical management over the months prior to her death and in particular whether her presentation should have alerted her treaters to the possible diagnosis of deep vein thrombosis, which required further investigation. Upon this issue Dr Bell advises that in his view warning signs were present which should have led to the investigation of possible deep vein thrombosis. These factors include:

- Mrs Harper's diagnosed CLL. He says that studies suggest the rate of deep vein thrombosis is 3 to 10 times greater in patients with CLL, although many patients have additional risk factors such as obesity. Obesity is not relevant here.

- The location of Mrs Harper's superficial thrombophlebitis diagnosed by Dr McLeod on 18 June. Dr McLeod's description suggests this thrombophlebitis to involve the superficial saphenous vein and to be sited in proximity to the deep vein system (the saphenopopliteal junction). Dr Bell reports that studies show that there is an incidence of co-existant deep vein thrombosis in patients with superficial phlebitis ranging from 6 to 53% with the highest risk occurring when the proximal great saphenous vein is involved.

- Mrs Harper's recent past history of a superficial venous thrombosis indicating a propensity to clot.

In Dr Bell's opinion Mrs Harper required a duplex ultrasound. This is a readily available investigative tool that provides immediate results. It is likely, in Dr Bell's opinion, that it would have revealed deep vein thrombosis. Treatment is with anticoagulant medication which is almost immediately effective. He says that within 24 hours of anticoagulation being commenced the rate of pulmonary embolism decreases to 1%.

Findings, Comments and Recommendations:

I accept the opinion of Dr Ritchey upon the cause of death. I accept too the opinions of Dr Bell as set out above. This leads me to the conclusion that Mrs Harper's death may have been avoided if she had been investigated for deep vein thrombosis.

This tragic case should serve as a reminder to the medical fraternity that patients who present with pain and tenderness along the known course of a superficial vein, most particularly the great saphenous vein, are likely to have not only superficial phlebitis but also the potential for a deep vein thrombosis which mandates investigation. This is especially so when other risk factors exist such as CLL.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how her death occurred and the particulars needed to register her death under the *Births, Deaths and Marriages Registration Act* 1999. I do not consider that the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me. The circumstances of the death do not require me to make any further comment or to make any recommendations.

I extend my sincere condolences to Mrs Harper's family and loved ones.

Dated: the 20th day of April 2016 at Hobart in the State of Tasmania.

Rod Chandler
Coroner

APPENDIX A (CORONERS REPORTS (VIII)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of James Maurice Smith

Find that:

(a) The identity of the deceased is James Maurice Smith.

(b) Mr Smith was born on 30 October 1976 and was aged 38 years.

(c) Mr Smith died on 13 June 2015 at the Royal Hobart Hospital (RHH) in Hobart.

(d) The cause of Mr Smith's death was haemopericardium (pericardial tamponade) due to an aortic dissection.

Background

Mr Smith was an unmarried Information Technology manager and the father of two daughters. Prior to June 2015 he had enjoyed good health.

Circumstances Surrounding the Death

In the evening of 9 June 2015 Mr Smith presented at the Emergency Department (ED) of the RHH via ambulance. The Patient Care Report with Ambulance Tasmania indicates that Mr Smith had been well during the day but in the evening, when having a bath, he experienced a *"rolling sensation"* down his neck into his back and then radiating to the lower right side of his abdomen. When he got out of the bath he was unable to control his right leg which also had 'pins and needles.' He was described by the paramedics when they arrived as diaphoretic (perspiring), anxious, hyperventilating and nauseous.

Mr Smith was seen in the ED at 11:41pm by junior resident medical officer, Dr Alice Mulcahy. In her notes she has recorded that Mr Smith, during his bath that evening, *"had (an) unusual sensation of pulsation from mouth to stomach as if 'I'd swallowed something huge'."* She also noted that in the ambulance he developed low abdominal and right low back pain. On arrival at the ED Mr Smith had an episode of vomiting and diarrhoea after which he reported his right lower limb symptoms had resolved. The impression was recorded as abdominal pain and flank pain with resolved neurology right lower limb. A differential diagnosis of appendicitis versus renal colic versus vascular was made. Venous blood gases showed a lactate level of 5 mmol/l; double the upper limit of normal.

Mr Smith was monitored over the following hours by Dr Mulcahy. She consulted with the medical officer in charge, Dr Richard Austin. He considered the diagnosis was unlikely to be vascular and concluded that Mr Smith had musculoskeletal low back pain and viral gastroenteritis. He was discharged home with ondansetron and a script for panadeine forte along with advice to follow up with his general practitioner. The actual time of discharge is not clear. The last entry made in the nursing notes was at 3.25am.

Mrs Penelope Smith is Mr Smith's mother. She says that her son visited her in the early afternoon of 11 June intending to stay for the evening meal. However, he was unwell, looked *"shocking"* and *"couldn't eat or even hold down a coffee."* The following day Mr Smith re-presented at the ED arriving at 2.55pm. At an unrecorded time he was seen by an ED registrar, Dr O'Donnell. The impression recorded was *"?appendicitis? other."* A surgical review was advised. That did not take place until 11.59pm when Mr Smith was seen by General Surgical Registrar, Dr Nicholas Davies. He was unsure of the diagnosis and listed a number of possible causes including colitis, gastroenteritis, pyelonephritis, renal colic or an unspecified viral illness. He did not consider Mr Smith's undiagnosed back and abdominal pain to be consistent with appendicitis. In his view it was difficult to explain Mr Smith's presentation with a single common diagnosis. He believed the predominant serious diagnosis that needed to be excluded was an epidural abscess. It was decided to proceed with a CT scan of the abdomen. At that time of night an MRI scan was not an available option.

At 4:25am on 13 June consultant radiologist, Dr John Vedelago reported verbally on the CT scan to Dr Davies. He advised that the scan showed aortitis with some aneurysmal dilation and an abdominal aortic dissection. The aortic dissection was said to be 'focal.' There was a thrombus in the superior mesenteric artery with good distal flow and some enlarged lymph nodes in the retroperitoneum. The radiologist recommended a formal CT aortogram, a heart echocardiogram and a vascular review. Dr Davies then reviewed Mr Smith again following which he telephoned vascular surgeon Mr David Cottier to inform him of the patient, the CT findings and to seek advice on appropriate management. It was Mr Cottier's opinion that the aortic dissection was likely incidental to aortitis. He suggested full anticoagulation for treatment of the thrombus, commencement of antibiotics for a presumed infective cause of the aortitis and institution of steroids. Mr Cottier did not accept Mr Smith as a vascular admission but instead suggested that he be referred to rheumatology for admission. In his report to the coroner Mr Cottier includes these statements:

> *"(Dr Davies) informed me of the presence of dissection in the aorta though my recollection is that this was thought to be focal and I do not recollect being informed that the superior extent of the dissection had not been visualised."*
>
> *"I have no recollection of being informed about his admission 48 to 72 hours prior to his index presentation to the Emergency Department."*
>
> *"If I had been informed of this prior attendance to the emergency department and the fact that the upper limit of Mr Smith's aortic dissection was not seen on the CT*

scan, I may have been more likely to have considered the possibility that he was suffering from an acute dissection of his thoracic area."

At 5.32am Mr Smith was being attended by a nurse when he suddenly appeared to stop breathing. CPR was promptly commenced but Mr Smith could not be revived. He was declared deceased at 6.17am on 13 June 2015.

Post-Mortem Examination

This was carried out by forensic pathologist, Dr Donald Ritchey. In his opinion the cause of Mr Smith's death was haemopericardium (pericardial tamponade) due to an aortic dissection.

Investigation

This has been informed by:

- A review of Mr Smith's records at the RHH carried out by research nurse, Ms L K Newman.
- An affidavit provided by Mrs Smith.
- Medical reports provided by Dr Austin, Dr Davies, Dr Vedelago (2), Mr Cottier and Dr Brian Doyle.
- A report upon Mr Smith's medical care and management prepared by Dr A J Bell, as medical adviser to the coroner.
- Meetings attended by myself, Dr Bell, Ms Newman, Dr Ritchey and State Forensic Pathologist, Dr Christopher Lawrence.

In his report Dr Bell advises:

1. That it was an error on Dr Austin's part to conclude that Mr Smith was suffering from musculosketal back pain and viral gastroenteritis when he presented on 9 June 2015. Such conclusion was at odds with the clinical history.
2. It was poor medical practice for Mr Smith's elevated blood lactate to have been ignored. The usual rule is that a patient with abdominal pain and an elevated blood lactate has ischaemia and requires investigation. Ischaemia is a clue that the issue may be vascular in nature.
3. Aortic dissection can be a difficult diagnosis to make. Nevertheless there were symptoms and signs evident on 9 June which required the condition to be considered as a possible diagnosis and for it to be positively excluded. In this context an article

published in The Medical Journal of Australia and available at https://www.mja.com.au/journal/2014/201/10/collaboration-between-coroner-and-emergency-physicians-efforts-improve-outcomes is particularly relevant. It was created following a roundtable meeting between members of the Coroners Prevention Unit (Victoria) and emergency physicians following the death of a woman from an undiagnosed aortic dissection. It sets out multiple clinical, system and cultural features of aortic dissection presentation which are likely to assist in the detection and management of the condition. Had the course recommended in this article been followed it is likely that the investigations for Mr Smith would have included a CT aortogram which would have confirmed the diagnosis.

4. The assessment carried out when Mr Smith represented to the ED on 12 June was superficial. Notably, it appears that no consideration was given to the information gained at the earlier presentation. An interval of 8 hours before a surgical review took place was unreasonable and poor practice.

5. That it was misleading for Dr Vedelago to describe the aortic dissection seen on the CT scan of the abdomen as being 'focal' when its superior or upper extent had not been determined by the scan of the abdomen. 'Focal aortic dissection' is a standard medical term which is commonly understood to indicate that urgent surgery is not required.

6. That the opportunity to appropriately treat Mr Smith's aortic dissection was effectively lost when the diagnosis was not made at the time of his first presentation to the RHH.

7. An aortic dissection is a medical emergency. It requires urgent surgery. In Mr Smith's case his prospects of surviving such surgery and resuming a normal life were approximately 75% if that surgery had been undertaken at the time of the first presentation.

Findings, Comments and Recommendations

I accept Dr Ritchey's opinion upon the cause of Mr Smith's death.

Aortic dissection is a serious and life threatening condition. Its prompt diagnosis is necessary to maximise the prospects of survival. I accept that it is often a difficult diagnosis to make and it is in this context that the publication produced upon the initiative of the Victorian Coroners Prevention Unit is particularly helpful. I commend it to the medical profession, most particularly those employed in emergency facilities.

The evidence shows that despite his two presentations to the ED the correct diagnosis of Mr Smith's illness was not made. This was a most regrettable outcome. I am satisfied, accepting the advice of Dr Bell, that at the time of Mr Smith's first presentation aortic dissection should have been included as a differential diagnosis. Appropriate steps taken then to investigate that condition would almost certainly have led to the diagnosis being made. Such an outcome would have given Mr Smith his best chance of survival. After Mr Smith presented at the ED on the second occasion the prospects of his aortic dissection being promptly diagnosed and treated were compromised by the apparent failure on the part

of the attending clinicians to inform themselves of the detail of Mr Smith's earlier presentation and by the 8 hour delay before a surgical review took place. That surgical review led to investigations being commenced which would have eventually enabled the diagnosis of an aortic dissection to be made. However, there was insufficient time for those investigations to be completed and for remedial surgery to be undertaken before Mr Smith's cardiac arrest and regrettable death.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how his death occurred and the particulars needed to register his death under the *Births, Deaths and Marriages Registration Act 1999*. I do not consider that the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me.

I convey my sincere condolences to Mr Smith's family and loved ones.

Dated: 19 May 2017 at Hobart in the State of Tasmania.

Rod Chandler
Coroner

Explanatory Addendum.

On 3 June 2016 Coroner Olivia McTaggart as Delegate of the Chief Magistrate pursuant to s58(1) of the Coroners Act 1995 directed that the investigation into the death of Mr Smith be re-opened pursuant to s58(1)(b) and (d) and the findings be re-examined by Coroner Rod Chandler. The findings set out in this document are made following that re-examination.

APPENDIX A (CORONER'S REPORTS (IX)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Mark Alexander Gordon

Find That:

(a) The identity of the deceased is Mark Alexander Gordon;

(b) Mr Gordon was born in Italy on 2 March 1964 and was aged 51 years at the time of his death;

(c) Mr Gordon died on 22 March 2015 at the Royal Hobart Hospital (RHH) in Hobart;

(d) Mr Gordon died in the circumstances described in this finding; and

(e) The cause of Mr Gordon's death was a pericardial tamponade (bleeding into the pericardial sac) complicating a Type I aortic dissection in the setting of long-standing hypertensive cardiovascular disease. Significant contributing factors were essential hypertension, Type II diabetes mellitus, morbid obesity (BMI 48), hepatic steatosis and chronic thyroiditis.

Background

Mr Gordon resided at 1/20 Susan Parade in Lenah Valley. He was single and employed as an accountant. His past medical history included hypothyroidism and hypertension.

Circumstances Surrounding the Death

At about 8:30 pm on Friday, 20 March 2015 Mr Gordon was playing table tennis at the Showgrounds Table Tennis Club in Glenorchy when he experienced the sudden onset of chest pain. An ambulance was called. Mr Gordon complained to the ambulance officers of chest pain radiating to the left side of his jaw. He said the pain was associated with light headedness. His temperature was recorded at 37.8°C, his blood pressure was 153/119 mmHg and his heart rate was 111 bpm. Mr Gordon was treated with glycerol trinitrate and aspirin. He was then conveyed to the Emergency Department (ED) at the RHH.

In the ED Mr Gordon was reviewed by Dr D Archer. He gave a history of the sudden onset of central chest ache, rating his pain at 7/10. He reported that the pain radiated to the jaw

bilaterally but not to his arms. It was noted that Mr Gordon felt faint but the feeling passed when he sat down. There was no associated nausea, no shortness of breath, no loss of consciousness, no calf pain, no fever and no sweating. His vital signs were recorded by nursing staff. He was afebrile, blood pressure was 150/85 mmHg, heart rate was 97 bpm, respiratory rate was 18 bpm and oxygen saturation was 94%. A blood glucose level was noted to be 28 mmol/L. Apart from obesity no abnormal clinical signs were noted. At this point the impression recorded was *"chest pain risk factors for IHD (ischaemic heart disease)."* Dr Archer discussed the situation with the medical officer-in-charge in the ED and the plan was settled to carry out blood tests and for a discussion to be had with the general medical team. The medical officer gave a diagnosis of a non-ST segment elevation myocardial infarction. It was noted that the blood troponin was elevated.

In the early hours of 21 March Mr Gordon had an unwitnessed collapse in the ED. He was found lying on the floor. He was pale and sweating. He was reviewed by Dr K Blackman, an emergency physician consultant. His blood pressure was noted at 128/91 mmHg and he was afebrile. An ECG was repeated.

At 3.00 am Mr Gordon was attended by Dr E Mountain who obtained a formal hospital admission history and carried out a further examination. The information obtained included a typical history indicating the onset of diabetes. Mr Gordon was then formally admitted to the Acute Planning Unit (APU).

At 9.00 am on 21 March Mr Gordon was seen as part of her morning ward round by consultant physician, Dr Nicole Hancock. His chest pain was noted to be worse when breathing in, a sign of pleuritic pain. Blood pressure was 140/110 mmHg and heart rate was 97 bpm. No diagnosis was recorded and no plan was noted to establish a diagnosis.

At 11.30 am that morning Mr Gordon was seen by Associate Professor Tim Greenaway. He reviewed Mr Gordon and ordered a management plan for his newly diagnosed diabetes.

The next day at 1.30 am Dr Mountain again reviewed Mr Gordon because of his continuing pain. Mr Gordon complained that the pain had radiated into his back. He said that he could bring on the pain with movement. A repeat ECG taken at this time was described as unchanged. Mr Gordon was given analgesia.

At 9:30 am Dr R Valsalam, an APU registrar reviewed Mr Gordon. At this time he complained of a dull ache that was worse with inspiration. His blood pressure was 130/100 mmHg, heart rate was 100 bpm and respiratory rate was 22 bpm. His chest was clear and the heart was normal to examination. An ECG showed no new changes. The blood troponin was noted not to have risen. No therapeutic changes were made and no diagnosis was given.

At 9.46 am Mr Gordon suffered a cardiac arrest. He could not be resuscitated and life was declared extinct at 10.22 am on 22 March 2015.

Post-mortem Examination

This was carried out by forensic pathologist, Dr Donald Ritchey. In his report Dr Ritchey includes this explanatory paragraph:

"The autopsy revealed a well-developed, morbidly obese (morbid obesity defined as a body mass index of greater than or equal to 40 kg/m2) adult Caucasian man with a Type I aortic dissection. Aortic dissections occur when a small tear forms in the endothelial lining of the ascending aorta and allows blood to escape into the wall of the aorta (aortic dissection). The blood dissected distally to the level of the diaphragm. Additionally, blood dissected back towards the base of the heart where it perforated into the pericardial sac causing acute accumulation of blood within the pericardium (pericardial tamponade). The major risk factor for developing aortic dissection is long-standing high blood pressure (essential hypertension). Obese individuals are at increased risk of high blood pressure and Type II diabetes."

In Dr Ritchey's opinion the cause of Mr Gordon's' death was pericardial tamponade (bleeding into the pericardial sac) complicating a Type I aortic dissection in the setting of long-standing hypertensive cardiovascular disease. Significant contributing factors were essential hypertension, Type II diabetes mellitus, morbid obesity (BMI 48), hepatic steatosis and chronic thyroiditis.

I accept Dr Ritchey's opinion upon the cause of death.

Investigation

This has focussed upon the failure of the RHH medical staff to diagnose and treat Mr Gordon's aortic dissection during the 1.5 days that he was in their care. It has included:

1. Consideration of an affidavit provided by Mr Atef Shabaneh, a brother-in-law of Mr Gordon's.

2. Consideration of reports provided by Dr Hancock, Dr Blackman and Professor Greenaway.

3. A review of Mr Gordon's records at the RHH.

4. Consideration of a report compiled by Dr A J Bell as medical adviser to the coroner.

5. Meetings attended by me, research nurse Ms L K Newman, Dr Bell, Dr Ritchey and State Forensic Pathologist, Dr Christopher Lawrence to review the investigation.

In his report Dr Bell includes these opinions which I accept:

- That aortic dissection is a differential diagnosis for patients suspected to be suffering an acute coronary syndrome.

- The varying nature of pain is a marker of an aortic dissection. It is contraindicative of ischaemic heart disease. Radiation of pain to the back is a classic sign of an aortic dissection.
- Mr Gordon's clinical examination should have included a recording of his blood pressure in each arm. Different readings, if obtained, would have been a strong indicator of an aortic dissection.
- The onset of pleuritic pain is suggestive of either mediastinal blood or pericarditis.
- Insufficient consideration was given to the cause of Mr Gordon's feeling of faintness and syncope. When associated with chest pain syncope can be a sign of aortic dissection and carries a higher mortality rate than exists for patients without syncope.
- That there were sufficient clues to suggest Mr Gordon may have had an aortic aneurism. He required an urgent CT scan of the chest with contrast.
- It is likely that a CT scan of the chest would have enabled a diagnosis of aortic aneurism. This condition has a high mortality rate if untreated and is a medical emergency mandating an immediate response. It required Mr Gordon's prompt admission to the Intensive Care Unit, medication with beta blockers or calcium channel blockers to reduce his blood pressure and blood flow velocity and preparation for immediate surgery.
- In all likelihood Mr Gordon required aortic surgery and perhaps a Bentall procedure. A significant risk attaches to this procedure. In Mr Gordon's case he had an approximate 65% chance of the procedure being successful and he enjoying a long term survival.

Findings, Comments and Recommendations

It is clear from Dr Ritchey's post-mortem results that Mr Gordon's death was precipitated by an aortic dissection. It is also clear that at no time was the diagnosis of this condition considered by the medical staff caring for Mr Gordon. This is a matter of real concern.

When Mr Gordon was seen by Dr Hancock in the morning of 21 March he had been complaining of variable pain for over 12 hours, he had been light headed and had suffered an unwitnessed syncope. Her own examination indicated pleuritic pain. These matters together should, in my view, have alerted Dr Hancock to the possibility that his chest pain was not attributable to acute coronary syndrome but may have another cause including an aortic dissection. Even more certainly, the possibility of an aortic dissection should have been evident when Mr Gordon reported pain radiating into his back in the early hours of the next morning, when seen by Dr Mountain. Regrettably, despite these circumstances, the investigation of an aortic dissection was not initiated. An early step in that investigation would have been a CT scan which almost certainly would have identified the aortic dissection and been a catalyst for an immediate response including surgery.

I am unable to positively find that Mr Gordon's death would have been avoided if his aortic dissection had been promptly diagnosed and appropriately treated. Nevertheless, the failure to make the diagnosis denied Mr Gordon the opportunity to undergo surgery, which although involving risk, had the prospect of enabling him to resume a normal life.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how his death occurred and the particulars needed to register his death under the *Births, Deaths and Marriages Registration Act* 1999. I do not consider that the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me. The circumstances of the death do not require me to make any further comment or to make any recommendations.

I convey my sincere condolences to Mr Gordon's family and loved ones.

Dated: 20 June 2016 at Hobart in the State of Tasmania.

Rod Chandler
Coroner

APPENDIX A (CORONER'S REPORTS (X).

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Jason Keith Harrison

Find that:

(a) The identity of the deceased is Jason Keith Harrison;

(b) Mr Harrison was born on 7 January 1981 and was aged 34 years;

(c) Mr Harrison died at the North West Regional Hospital (NWRH) in Burnie on 7 February 2015;

(d) The cause of Mr Harrison's death was sepsis due to an extensive small bowel infarct caused by bowel incarceration within a congenital mesenteric defect (mesenteric hernia).

Background

Mr Harrison was single and lived alone in Queenstown. His mother, Margaret Rose Harrison, resided nearby and he visited her daily. He had a learning disability but otherwise enjoyed good health.

Circumstances Surrounding the Death

Mrs Harrison advises that her son visited her in the morning of 6 February 2015. He asked for something to eat and she made him some toast and gave him some cordial. Afterwards Mr Harrison vomited. He then complained that his stomach was sore and getting worse. He said that he had been unable to open his bowels for a few days. He said he was hot and felt weak. He requested his mother call an ambulance, which she did. Mr Harrison continued to vomit. An ambulance arrived after a short delay. Mr Harrison was then transported to the West Coast District Hospital (WCDH). The Ambulance Tasmania (AT) notes show that Mr Harrison reported having woken at 6.00am with *"epigastric crampy pain"*, that he had vomited, that he was agitated, that he denied consumption of alcohol or use of illicit drugs, and that he claimed to be unable to walk but was able to do so with encouragement. The initial assessment by AT was *"gastrointestinal problem."*

The hospital records show that Mr Harrison was triaged at 8.37am. He was noted to be complaining of epigastric pain and vomiting. His clinical observations were blood pressure 127/59, pulse 50, respirations 16, oxygen saturation 100% and temperature 34.8°C. Shortly afterwards he was reviewed by Dr Dennis Pashen. It was noted that Mr Harrison was alert but agitated. It was recorded that Mr Harrison said that he had been to Strahan the previous afternoon and had had some chips and soft drink. He denied any illicit drug use. However, others at the hospital informed Dr Pashen that Mr Harrison had been seen in the company of known drug users. Examination by Dr Pashen showed generalised tenderness over the abdomen with normal bowel sounds, no rebound or guarding. The decision was taken to admit Mr Harrison for further observation. A definite diagnosis was not made at this time. Intravenous fluids were commenced, a drug screen was ordered, analgesics and medication for gastritis were given and appeared to be beneficial. A review was planned in two hours.

Mr Harrison was reviewed at 2.00pm. It remained difficult to obtain a clear history from him because of his agitation and general demeanour. He refused an ECG. He still complained of upper abdominal pain but the pain seemed not to stress him. It was noted that he denied using medication or illicit drugs. At 2.40pm, nursing staff noted that he was *"verbally abusive and threatening to pull out the IV cannula. Client is constantly asking for sedatives stating, 'I want something to sleep.'"*

During the afternoon Dr Pashen telephoned the NWRH and enquired about possible illicit drugs that Mr Harrison may have obtained. The NWRH was unable to help. Mr Harrison's vital signs remained normal. It was decided to continue his observation.

At 8.30pm it was recorded that ongoing management was to continue and that a fluid balance sheet was to be maintained. However, Mr Harrison was not *"co-operating with measurement of output."* At 11.30pm it was noted that Mr Harrison had had a shower and was sleeping.

The following morning at 6.30, Mr Harrison informed nursing staff that he was experiencing an urgency to urinate but was unable to void. Assessment found a distended bladder and abdomen. He was tender to touch. Dr Pashen was advised and he directed that Mr Harrison be catheterised. It was then recorded: *"Collected 50mL of urine. Not draining. Informed GP for review of patient."*

At 7.15am Dr Pashen reviewed Mr Harrison. His abdomen remained distended and tender. It was noted that he had passed *"minimal urine"* and that his observations had been stable until this review, when his respiration rate was elevated. He continued to have generalised pain and tenderness and his analgesia was changed to morphine. The decision was taken to transfer Mr Harrison to the NWRH. In his letter of referral Dr Pashen indicated a diagnosis of *"probable bowel obstruction ? cause....."*

The ambulance attended at WCDH at 8.36am and departed with Mr Harrison at 9.00am. At Tullah he was transferred to a second ambulance. It arrived at the NWRH at 11.27am. The triage notes state that Mr Harrison was pale and looking unwell on arrival. He was also noted to be tachycardic and tachypnoeic. Shortly after his arrival he became unresponsive. Resuscitation was commenced. It was overseen by Emergency Medicine consultant, Dr

Brian Doyle, and was maintained for about 30 minutes. However, Mr Harrison could not be revived and he was declared deceased at 12.28pm on 7 February 2015.

Post-Mortem Examination

This was carried out by forensic pathologist, Dr Donald Ritchey. In his opinion the cause of Mr Harrison's death was sepsis due to an extensive small bowel infarct caused by bowel incarceration within a congenital mesenteric defect (mesenteric hernia).

Investigation

This has included:

1. Consideration of an affidavit provided by Mrs Harrison.
2. Obtaining Ambulance Tasmania reports.
3. Consideration of a report provided by Dr Pashen.
4. A review of Mr Harrison's records at the WCDH and the NWRH, carried out by research nurse, Ms L K Newman.
5. Consideration of a report provided by Dr A J Bell as medical adviser to the coroner.
6. Meetings attended by myself, Ms Newman, Dr Bell, Dr Ritchey and State Forensic Pathologist, Dr Christopher Lawrence, to monitor the investigation.

In his report Dr Bell makes these comments:

- Abdominal pain can be a challenging complaint for both a primary care and specialist physician because it is frequently benign but it can also herald serious acute pathology.

- The first diagnoses that must be considered in patients with acute abdominal pain are those that may require urgent surgical intervention. The 'surgical abdomen' can be usefully defined as a condition with a rapidly worsening prognosis in the absence of surgical intervention. Two syndromes that fit into this category are obstruction and peritonitis.

- The notes of Dr Pashen made at the time of his initial examination represent an inadequate assessment of Mr Harrison. He did not record a diagnosis or a differential diagnosis. The impression given is that drug abuse was suspected and that it was the cause of Mr Harrison's abdominal pain.

- Mr Harrison's history, as described by his mother, coupled with the information recorded by AT, should have alerted Dr Pashen to suspected bowel obstruction as a possible diagnosis for Mr Harrison.

- For most patients plain x-rays will quickly confirm a diagnosis of bowel obstruction. In this instance it seems that Dr Pashen failed to obtain x-rays.

- By 2.00pm on 6 February, the decision should have been taken to transfer Mr Harrison to the NWRH. The reasons are threefold. First, was his unco-operative behaviour and agitation which made his assessment and treatment difficult. Second, was the diagnosis of suspected bowel obstruction. For the reasons stated this should have been in Dr Pashen's contemplation. Third, the suspicion was that Mr Harrison was suffering from drug abuse. This raised the potential need for managing drug withdrawal, a process which is difficult and can be best managed in a larger and better resourced facility.

- Mr Harrison's condition required urgent surgery involving a laparotomy with bowel resection. For a person of his age, the prospects of this surgery being successful and Mr Harrison making a full recovery were good.

Findings, Comments and Recommendations

I accept Dr Ritchey's opinion upon the cause of death.

It is apparent, with the benefit of hindsight, that Dr Pashen's decision to retain Mr Harrison at WCDH and monitor his condition was a misjudgement. The evidence suggests that this decision was overly influenced by the suspicion that Mr Harrison's presentation was attributable to illicit drug use. Rather, it is my view, accepting the opinion of Dr Bell, that there was sufficient evidence to raise suspected bowel obstruction as an explanation for Mr Harrison's presentation. This mandated his evacuation to NWRH as this facility was equipped to confirm the diagnosis by CT scan and was resourced to promptly respond with a laparotomy and resection if it became necessary. Such evacuation should have occurred, again accepting Dr Bell's opinion, during the afternoon of 6 February. I cannot positively find that an evacuation at that time would have guaranteed Mr Harrison's survival from his bowel obstruction. However, I am satisfied that in this eventuality his prospects of survival would have been greatly enhanced.

This case, along with a recent finding of mine upon the death of Mr Ian Summerfeldt, should serve as a reminder to all small-scale hospital facilities in the State of the need to take a particularly cautious approach when managing patients with potential 'surgical abdomens', given their limited capacity to respond in the event of a rapid deterioration in the patient.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how his death occurred, and the particulars needed to register his death under the *Births, Deaths and Marriages Registration Act 1999*. I do not consider that

the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me.

I convey my sincere condolences to Mr Harrison's family and loved ones.

Dated: 16th day of December 2016 at Hobart in the State of Tasmania.

Rod Chandler
Coroner

APPENDIX A C& CORONER'S REPORTS (X1)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Olivia McTaggart, Coroner, having investigated the death of Jason Mark Brook

Find, pursuant to Section 28(1) of the Coroners Act 1995, that:

a) The identity of the deceased is Jason Mark Brook;

b) Mr Brook died as a result of massive pulmonary embolism due to deep vein thrombosis of the left leg due to a left leg injury and immobilisation following a motor cycle accident;

c) The cause of Mr Brook's death was pulmonary embolism;

d) Mr Brook died on 7 November 2014 at 80 Summerhill Drive, Port Sorell, in Tasmania; and

e) Mr Brook was born in Devonport on 27 April 1969 and was aged 45 years; he was single and was unemployed at the date of death.

In making the above findings I have had regard to the evidence gained in the comprehensive investigation into Mr Brook's death. The evidence comprises an opinion of the forensic pathologist who conducted the autopsy; review by the coronial medical consultant; relevant police and witness affidavits; medical records and reports; and forensic evidence.

In October 2014 Mr Brook travelled to Thailand for a family wedding. Whilst he was there he was involved in a motorcycle accident and sustained injuries to his left foot, including a wound. He sought medical treatment which involved dressing the wound and antibiotics. In consultation with his family, he decided to return to Tasmania in the event that his injury became more serious and required further treatment.

On 21 October 2014, after landing at Devonport airport, he proceeded directly to the Mersey Community Hospital for treatment.

An x-ray was performed which showed that Mr Brook had a comminuted fracture of the first metatarsal bone. The wound was unpacked and cleaned. The wound was left open. He was also treated with intravenous antibiotics. In the morning Mr Brook was transferred to the North West Regional Hospital ("NWRH") and underwent surgical wound washout and debridement

on the 23 October 2014. The wound could not be closed and vacuum closure device applied. The fracture was immobilised in a plaster cast. Delayed primary closure of the wound was performed on the 25 October 2014. Mr Brook was discharged from the NWRH on 25 October 2014.

On 30 October 2014 Mr Brook was seen in the outpatient clinic of the Mersey Hospital and it was noted that the wound was healing satisfactorily.

On 5 November 2014 Mr Brook was reviewed by Dr M Caudwell. There was pain on the top of the foot. The wound looked good, healing cleanly with no signs of infection. The sutures were removed. A below knee fibreglass cast was applied.

In the early hours of 7 November 2014 Mr Brook was at home. He called out from his room to his father, John Brook, stating "I am having a turn". When John entered his son's room he noticed that his breathing was laboured and he was gasping whilst laying on his back. John called 000 for assistance.

Mr Brook's condition deteriorated whilst awaiting the arrival of the ambulance and he became unresponsive. Upon arrival ambulance personnel determined that Mr Brook was deceased.

Upon autopsy, Dr Ruchira Fernando, pathologist, concluded that Mr Brook died as a result of a massive pulmonary embolism ("PE") obstructing blood flow to the lungs. The PE resulted from deep vein thrombosis ("DVT") of the left leg. I accept Dr Fernando's opinion.

Mr Brook was not given anti-coagulation medication in hospital, nor was it prescribed upon his discharge. Mr Brook was a person who was at risk of suffering DVT, being an adult with a lower limb fracture, infection, a prolonged period of immobilisation that was ongoing, and plaster cast immobilisation post discharge.

Dr Anthony Bell, coronial medical consultant, stated in his review that anti-coagulation should be given during the entire period of immobilisation in a cast, including post discharge. He noted that studies show that the rate of DVT is reduced by the use of prophylactic anti-coagulation. He noted that the studies do not show a significant corresponding reduction in the rate of PE. However, there were insufficient patient numbers studied to provide clear conclusions.

Nevertheless, Dr Bell stated that in the case of Mr Brook the PE resulted directly from the left leg DVT. He concluded that if Mr Brook had been receiving anti-coagulation medication, there would have been a significant chance that his DVT, and subsequent death from PE, would have been prevented. I accept Dr Bell's opinion in this regard.

I have received a report from Dr Tony Austin, Interim Director of Medical Services, Tasmania Health Services, North West. Dr Austin has reviewed the treatment provided to Mr Brook. He stated:

> "It is the opinion of the clinical leader and the organisation that this patient should have been administered anti-coagulation therapy as per recommended VTE (Venous Thromboembolism) protocol guidelines as the patient had the following risk factors for DVT.
>
> - Adult with lower limb fracture
> - Infection
> - Prolonged period of immobilisation

- Ongoing immobilisation
- Plaster of Paris immobilisation post discharge.

It is noted that the DVT management of Mr Brook is inconsistent. Upon review it is also noted that daily anti-coagulation was commenced on the day of discharge although not administered. This medication was not continued as discharge medication. It is not clearly known why anti-coagulation therapy was not continued but thought likely due to human error and heavy workload of the junior doctors over the weekend period.

Given that the clinical leader and the organisation are very concerned over the management of this patient, the organisation will undergo a root cause analysis and review into the use of VTE prophylaxis in the THS – North West Region. This will include a retrospective audit of all DVTs post op in the last 12 months and full review of management in these cases to identify trends in management and treatment. In addition medical staff education regarding optimal VTE management will be added into the orthopaedic education timetable. This case review will also be undertaken by the establishment and loaded into the SRLS system to be reviewed again through the usual Mortality and Morbidity committees for discussion.

All the doctors involved in treatment have been involved in the review process and are aware of the demise of Mr Brook. It is unclear without further analysis whether there is a systemic issue or this was a one off omission to align with current VTE protocols. Orthopaedics as a craft will be involved in the RCA to identify any further issues and to ensure future patients are managed according to guidelines.

The organisation acknowledges that Mr Brook died as a result of pulmonary embolus which has been identified by the pathologist as originating from a left calf DVT. Mr Brook should have been further anti-coagulated given his presentation and risk factors. The treating teams and the establishment are remorseful that Mr Brook died from his condition and will endeavour to extract learnings and better practice as a result."

I acknowledge that the hospital accepts that anti-coagulation treatment should have been administered to Mr Brook for the time that he was immobilised and that the failure to do so increased the risk of his death. This omission to supply Mr Brook with this standard treatment was most regrettable.

The Brook family raised the question of Dr Austin attributing responsibility to the junior medical staff for failure in anticoagulation. Dr Bell notes several issues in this regard. The initial post-operative instruction of the surgeon, Dr Nara (Narayanasamy), was for "clexane tonight". On this instruction, the junior doctor wrote up a single dose of clexane for that night. Usually the instruction would be interpreted to commence clexane prophylaxis that night and to continue it. Secondly this error meant that the drug chart did not have clexane recorded as a reminder to the discharging doctor that anticoagulation was required. Thirdly, there was no instruction for anticoagulation by Dr Caudwell after the surgery of 25 October 2014. Fourthly, the consultants and registrars on the orthopaedic ward round of 24 October 2014 did not notice the lack of prophylactic anticoagulation at that time. Thus, Dr Bell observes that errors occurred at multiple levels. I accept his opinion.

I endorse the efforts that have been made by the hospital, as set out in Dr Austin's report. I acknowledge the ongoing audit and education processes being undertaken to ensure appropriate anti-coagulation treatment to patients in the future.

Comments and Recommendations:

The death of Mr Brook highlights the need for vigilance and effective processes to ensure that patients receive appropriate post-surgery anti-coagulation therapy as recommended by relevant guidelines.

The circumstances of Mr Brook's death are not such as to require me to make any recommendations pursuant to Section 28 of the *Coroners Act* 1995.

I convey my sincere condolences to the family and loved ones of Mr Brook.

Dated: 26 August 2016 at Hobart in the State of Tasmania.

Olivia McTaggart
Coroner

This finding has been amended pursuant to an order under Section 58 of the Coroners Act made on 1 August 2016 and replaces the finding dated 27 June 2016.

APPENDIX A (CORONER'S REPORTS (XII)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Teressa Maree Beswick

Find That:

a) The identity of the deceased is Teressa Maree BESWICK;

b) Ms Beswick was born in Devonport on 2 January 1969 and was aged 45 years;

c) Ms Beswick died on 24 October 2014 at the Mersey Community Hospital ('MCH') in Latrobe; and

d) The cause of Ms Beswick's death was the combined effects of widely metastatic (end-stage) carcinoma of the cervix and bronchopneumonia.

Background

In about June 2013 Ms Beswick was diagnosed with cervical cancer. Her initial treatment included a course of chemotherapy. On 13 October 2014 Ms Beswick had a MRI scan and was then informed that her cancer had metastasised to her liver and spine. Her prognosis at this time was extremely poor.

Circumstances Surrounding the Death

On 23 October 2014 Ms Beswick was attended by a palliative care nurse at her home. She was suffering increased pain throughout her spine and lower abdomen. The nurse arranged for her to be taken by ambulance to the MCH.

Dr Amanda Felmingham was a General Practice Registrar who was attached to the North West Specialist Palliative Care Service for extended skills training in palliative care. Ms Beswick was known to Dr Felmingham as she had been treating her as part of her placement. Dr Felmingham was anticipating Ms Beswick's arrival in the ED as she had been forewarned by the palliative care nurse. She went to the ED and spoke to Dr Glenn Bennett, a consultant in emergency medicine. She informed Dr Bennett of Ms Beswick's imminent arrival and the need for her admission and for

treatment with palliative pain relief. She indicated that the best way to control Ms Beswick's pain may be to change from her use of fentanyl patches to morphine with a syringe driver. At the time Ms Beswick's prescription was for fentanyl patch 37mcg per hour.

When Ms Beswick arrived in the ED her management was assumed by Career Medical Officer, Dr Rajesh Menon. He was advised by Dr Bennett of the discussion with Dr Felmingham and the plan to admit Ms Beswick for pain relief.

Dr Menon spoke to Ms Beswick. He advised her of the plan for admission and to commence morhine using a syringe driver. She advised she had previously been sensitive to morphine. Dr Menon suggested that the morphine infusion be tried in the ED and for it to be changed back to fentanyl if she had an adverse reaction to the morphine. Ms Beswick agreed with this course.

It seems that Dr Menon had little experience in commencing palliative morphine with a syringe driver. He sought the advice of Dr Felmingham. However, she had left the hospital. He made contact with her by telephone. She was in her car. She explained to Dr Menon the dosage of morphine that Ms Beswick was receiving via her fentanyl patches and how this should be converted to subcutaneous morphine. Dr Menon then proceeded to do the conversion calculation which he set out in his notes. He calculated the dosage at 45 mg. Nursing staff were then instructed to prepare the syringe driver to deliver this dosage. The syringe driver order forms were completed to show the 24 hour dosage to be 1080mg, that is an hourly dose of 45mg. The calculations shown on the syringe driver records were co-signed by registered nurses Sarah Hill and Kendall Pearce. However, it is noted that in their statements both nurses indicated to Dr Menon that the morphine dosage seemed excesive but he assured them that it was correct.

The subcutaneous infusion of morphine was commenced at 5:20pm. A 50 mcg dose of intra-nasal fentanyl was also administered at the same time. Ms Beswick was then transferred to the surgical ward. She was admitted by Registrar, Dr Chau Ng. It seems that Dr Ng discussed the morphine dosage with Dr Menon who showed him his calculations as documented in the ED notes. Dr Ng accepted the dosage to be correct. Thereafter operational checks were made on the syringe driver. At 9.00pm it had infused 2.0 mL, i.e. 160 mg of morphine. At 12:20am the operational check showed that 3.6mL had been infused, i.e. 288mg of morphine. At this time Ms Beswick's respiratory rate was noted at 12 bpm. At 3:45am the next operational check showed 5.3mL had been infused, i.e. a total of 424mg of morphine. At this time Ms Beswick's respiratory rate was noted to be 10 bpm. At 5.00am it was recorded by nursing staff that Ms Beswick had been administered 493 mg of morphine since the infusion commenced. It was also recorded that she was *"difficult to wake, sweating++, pupils pinpoint, slurred speech, chest rattly."* At this point a Dr Brough requested that the syringe driver be turned off until Ms Beswick's condition improved or she complained of pain.

At an operational check at 5:30am on 24 October 496mg of morphine had been infused. The respiratory rate was 10 and Ms Beswick was described as having pin

point pupils, was *"sleepy"* and her oxygen saturation levels were noted at 88-91%. The infusion was stopped. It was restarted at 7:40am when it was noted that Ms Beswick was *"crying in pain"* and her respiratory rate was 24 bpm. However, it was ceased 20 minutes later when her sedation score was noted as "2." At this time nursing staff had concerns about the dosage of morphine and an urgent medical review was sought.

At 8:55am the MCH pharmacist recorded in the records: *"This dose needs urgent review as I don't believe the dose is correct."* At about 9.00am a Code Blue was called because Ms Beswick's conscious state was not improving. To this point a cumulative dose of 497mg had been administered. She was given oxygen. At 9:20am Ms Beswick was reviewed by Nephrologist and Consultant Physician, Dr Jay Sen Gan. It was clear by this point that Ms Beswick had been receiving an overdose of morphine. He noted that she was in respiratory distress and had mild hypotension and tachycardia. Clinically Ms Beswick appeared to Dr Gan to have narcotic withdrawal having been given naloxone shortly beforehand.

There was a further MET call at 11.45am because of the level of sedation, hypotension and increased respiratory rate. Thirty minutes later Ms Beswick was transferred to the High Dependancy Unit (HDU). She was treated with IV antibiotics and IV fluids. It was noted at this time that she was *"alert and asking for drinks of water."*

Dr Gan met with members of Ms Beswick's family at 1.45pm. He explained to them the morphine overdose. It was noted that the family did not want Ms Beswick to be treated with intubation or CPR. That afternoon Ms Beswick was closely monitored. Her condition further deteriorated and she died at 5.10pm.

Post-Mortem Examination

This was undertaken by Forensic Pathologist, Dr Donald Ritchey. He reports that in his opinion the cause of Ms Beswick's death was the combined effects of widely metastatic (end-stage) carcinoma of the cervix, bronchopneumonia and excessive morphine sedation.

In his report Dr Ritchey also makes these comments:

"Toxicology testing of peripheral femoral vein blood obtained at autopsy revealed a markedly elevated concentration of morphine that was, "within the reported fatal range." The interpretation of this result is complicated by several factors. First is that morphine is subject to post-mortem redistribution such that the concentration identified in post-mortem blood may not reflect the actual concentration in the blood at the time of death. Also individuals on long term opiate therapy develop tolerance to opiate medications of all types (cross tolerance) and blood concentrations that may be required for pain control in an opiate tolerant individual might be fatal to an opiate naïve individual.

"These findings are interpreted by me to suggest that Ms. Beswick was actively dying at the time she presented with intractable pain to the hospital. The extensive tumour burden seen at autopsy would be expected to cause imminent death. Analgesia with morphine at a high dose was clinically warranted, however the central nervous system and respiratory depressive effects of such a large dose, in the setting of acute bronchopneumonia and terminal tumour burden, likely accelerated the process."

Investigation

The focus of this investigation has been upon the excessive dosage of morphine administered to Ms Beswick. How did it come to pass that this overdose occurred? What role, if any, did it play in Ms Beswick's death?

To assist in the investigation, statements were obtained from the following personnel at MCH:

- Dr Rajesh Menon, Career Medical Officer
- Dr Amanda Felmingham, Palliative Care Consultant
- Dr Bruce Webb, Medical Officer
- Ms Kendall Pearce, ED Nursing Staff
- Ms Sarah Hill, ED Nursing Staff
- Rodney Rouse, After Hours Nursing Coordinator
- Ms Chantelle Graham, Nursing Staff
- Ms Cheryl Harrison, Nursing Staff
- Ms Simone Collins, Nursing Staff
- Ms Anne Jong, Nursing Staff
- Ms Sandra Graham, Nursing Staff
- Michael Grant, Nursing Staff
- Dr Faranak Dehghani, Medical Registrar
- Dr Thitipoom Aikphaibul, Medical staff
- Dr Jay Sen Gan, Consultant Physician
- Dr Chau Ng, Medical Registrar

- Ms Rachael Heng, Pharmacist

In addition, the investigation has been assisted by a Statutory Declaration provided by Ms Beswick's daughter, Ms Taneka Parker, along with a statement supplied by Ms Beswick's mother, Mrs Elaine Reid.

The investigation has also included:

1. A review of Ms Beswick's hospital records at the MCH carried out by research nurse, Ms L K Newman.

2. The compilation of a report upon the investigation made by Dr A J Bell as medical adviser to the Coroner.

3. Meetings attended by myself, Ms Newman, Dr Bell, Dr Ritchey and State Forensic Pathologist, Dr Christopher Lawrence, to periodically review the investigation.

In his report Dr Bell advises of the following:

- It is standard and appropriate practice to use the subcutaneous infusion of morphine via a syringe driver to control pain for a patient in Ms Beswick's situation.

- The maximum effective dose of morphine for an opiate tolerant patient such as Ms Beswick is 120mg per 24 hours (5mg per hour) when delivered by subcutaneous infusion.

- The morphine dose of 45mg per hour or 1080mg per 24 hours as calculated and prescribed by Dr Menon was nine times the maximum effective dose. Dosage of this magnitude was most inappropriate.

- The principal cause of Ms Beswick's death was her cancer which caused severe metabolic and immunological dysfunction. The evidence indicating that at 12.15pm on 24 October Ms Beswick was alert and requesting water suggests that her morphine overdose was a secondary cause of death.

Findings, Comments and Recommendations

When Ms Beswick presented at the ED on 23 October 2014 it is apparent that she required an alteration to her medication regime to better manage her pain. It was agreed to replace her fentanyl patches with the infusion of morphine utilising a syringe driver. I accept Dr Bell's opinion that this was an appropriate course of treatment. This change of opiate necessitated a calculation to be made of the amount of morphine to be infused which equated with Ms Beswick's fentanyl patch prescription. Dr Menon has acknowledged that in carrying out this calculation he

failed to appreciate or recognise that the 45mg which the conversion table showed to be the correct amount was for a 24 hour period and not for one hour. In the result, for an uninterrupted period of approximately 12 hours Ms Beswick's syringe driver was calibrated to deliver a dosage of morphine 24 times greater than was intended. Over this period the records show that she was infused with 496mg of morphine which represents in excess of 40mg per hour. This amount is grossly in excess of the 5mg per hour which, in the opinion of Dr Bell, represented the maximum appropriate dose for Ms Beswick. In these circumstances I find that Ms Beswick was grossly overdosed with morphine whilst a patient of the MCH.

In his report to the coroner Dr Menon has acknowledged his error and offered his apologies to Ms Beswick's family. I accept that the morphine overdose was the result of a genuine mistake on his part and may partly have been attributable to his unfamiliarity with palliating patients with the aid of a syringe driver. Nevertheless, it is of concern that a medical practitioner with his experience (he graduated in 2002) did not instinctively recognise that his calculation of the morphine dose could not be correct and required reconsideration and/or consultation with others.

Dr Ng was the registrar in charge of the surgical ward and the person responsible for Ms Beswick's care upon her admission to that ward. The evidence shows that he had reservations concerning the correctness of the morphine dosage but accepted it to be correct after being shown Dr Menon's calculation. This is another matter of concern. As with Dr Menon it is my view that he should, as a registrar, have instinctively recognised that the amount of morphine being delivered to Ms Beswick could not be correct and taken steps to right the situation.

As a result of his post-mortem findings Dr Ritchey has expressed the view that Ms Beswick was suffering from widespread cancer and that she was actively dying at the time of her presentation at the MCH. I accept this opinion. It is consistent with the opinion of Dr Bell that Ms Beswick's cancer was the principal cause of her death. It is my view, and I find, that Ms Beswick's death was imminent when she arrived at the MCH on 23 October 2014, that palliation was the only realistic treatment option and that there was little prospect of her survival beyond the short term. The morphine overdose played a secondary role in her death in that it accelerated its arrival but only by a relatively short but inderterminable period.

This case highlights serious shortcomings within the MCH pharmacy concerning the safeguards required to prevent the dispensing of medications for prescriptions which are clearly questionable. It is my **recommendation** that the MCH undertake a review of its pharmaceutical protocols with a view to implementing practices which reduce the risk of drug overdoses such as has occurred on this occasion.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how her death occurred and the particulars needed to register her death under the *Births, Deaths and Marriages Registration Act* 1999. I do not consider that the holding of a public inquest would elicit any significant information further to that disclosed by the investigation

conducted by me. The circumstances of the death do not require me to make any further comment or to make any recommendations.

I convey my sincere condolences to Ms Beswick's family and loved ones.

Dated: 8 day of August 2016 at Hobart in the State of Tasmania.

Rod Chandler
Coroner

APPENDIX A (CORONER'S REPORTS (XIII)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Ian Patrick Summerfeldt

Find that:

(a) The identity of the deceased is Ian Patrick Summerfeldt.

(b) Mr Summerfeldt was born at Brisbane in Queensland on 25 May 1966 and was aged 48 years.

(c) Mr Summerfeldt died at the North West Regional Hospital (NWRH) in Burnie on 23 October 2014;

(d) The cause of Mr Summerfeldt's death was sepsis and renal failure due to a perforated gastric ulcer.

Background

Mr Summerfeldt was single and had lived alone at Queenstown since early September 2014. He had previously lived in Launceston. He had been the partner of Ms Therese Dora Orme and they had a daughter, Millie. Although Mr Summerfeldt and Ms Orme separated in about 1991, they remained close friends and continued to live in close proximity. Mr Summerfeldt's past medical history included hepatitis C, schizo-affective disorder, chronic back pain and chronic obstructive pulmonary disease.

Circumstances Surrounding the Death

On Monday 20 October 2014, Mr Summerfeldt was visiting his daughter and grandson. After lunch he said that he felt sick and that he had worsening stomach pain. He declined her offer to call an ambulance. However, later that day he phoned his daughter and asked that an ambulance be called. He was conveyed to the West Coast District Hospital (WCDH) arriving at about 3.15pm. The hospital records show that Mr Summerfeldt indicated that his pain was located in the right upper quadrant and that he rated it as 8/10. A nurse recorded that Mr Summerfeldt's pain radiated to his right shoulder tip, that his abdomen was tender all

over, especially the upper abdomen, and he described the pain as 'continuous' rather than 'colicky'. This further entry has been made in the nursing notes:

> *"Whilst awaiting review patient left the facility "due to pain". Patient also stated staff here have 'poor bedside manner – why would I want to stay here in pain.' Following having left patient entered the staff dining room and disturbed another patient (inpatient) in the process. Author and nurse unit manager had to convince patient to get into a wheelchair and return to the A & E department so as the doctor could review. Analgesic was given and we continued to monitor patient as per doctor's instructions. Patient later left as he had 'other things to do' and stated to author he could get 'black market medications if he wanted'. Doctor informed and patient left the facility at 17.50. Impression upon discharge was ? Gallstones or ? Drug-seeking."*

Whilst in the WCDH clinical observations were made of Mr Summerfeldt on three occasions and were within normal limits. He had no signs of fever, and was given ketorolac for pain relief.

The following morning Ms Summerfeldt visited her father. She said he *"seemed a bit better"* but *"you could tell that he has (sic) still unwell."* At about 5.00pm she visited him again. This time she said; *"He was in so much pain that he couldn't get up to answer the door. I could hear him crying out in pain."* However, Mr Summerfeldt would not invite his daughter into his home and did not want her to call an ambulance.

At 10.50am on 22 October, Mr Summerfeldt re-presented at the WCDH. He was transported by ambulance having been found lying on the ground outside his home. The ambulance case report indicated that his speech was slurred and he *"had excessive jaw movement."* His skin was described as mottled. He had trouble standing and an abnormal gait. It was recorded that he appeared intoxicated although there was no smell of alcohol. At the WCDH his presentation was recorded as: *"Brought in by ambulance, found lying in path - slurred speech, complaining of pain 10/10."* He was described as pale. Clinical observations were: pulse ~128, respiration ~19, blood pressure 150/100, oxygen saturation 99% and temperature 35.6-36.3°.

Nursing notes indicate that Mr Summerfeldt was reluctant to be admitted but did agree after discussion with family members.

After admission, Nurse Practitioner Arthit Barnes took over Mr Summerfeldt's care. In the afternoon Mr Barnes requested Dr Alastair Currie to assess Mr Summerfeldt. He reports that at that time *"his abdomen was generally mildly tender but was soft with no guarding or rebound. His colon felt to be distended and Mr Summerfeldt stated that he felt constipated. Bowel sounds were present and a per rectum examination was normal. Except for a tachycardia of 125/min, his other vital observations were acceptable. He also appeared quite dehydrated at that time. A working diagnosis of constipation or gallstones was made and he was treated with IV rehydration of 3L of fluid and analgesia along with management for constipation."* Abdominal x-rays showed faecal loading of the colon and small bowel. Blood tests showed a raised urea and creatinine along with an elevated hemoglobin and a

normal white cell count. Dr Currie regarded these results as indicating a moderate level of dehydration.

During the afternoon of 22 October Mr Summerfeldt continued to complain of ongoing and worsening abdominal pain. Dr Currie was kept informed. He was visited by Ms Orme. She gives this description; *"Ian couldn't talk. He was almost unrecognisable. His forehead was all puffed up, he had a massive bruise (blood coming to the surface) on his right hand side stomach and chest."*

Dr Currie re-examined Mr Summerfeldt at 5.25pm, and was content to continue with the diagnosis of constipation with the intention to *"Re-check path in morning."* The nursing notes show that at 8.00pm Mr Summerfeldt was complaining of worsening abdominal pain which was treated with IV morphine. At 9.00pm he was found on the floor. Dr Currie was called. He states that Mr Summerfeldt was *"clearly in shock with a distending abdomen suggestive of a ruptured bowel."* He decided that at that point it was necessary for Mr Summerfeldt to be transferred to the NWRH.

Ambulance Tasmania records show that it received a call for Mr Summerfeldt's transfer at 10.40pm, and that the ambulance actually departed the WCDH at 11.40pm. En route to Burnie Mr Summerfeldt was positioned supine with his legs elevated. A notation was made that he did not have a radial pulse and only a weak brachial pulse.

The ambulance arrived at the NWRH at 1:10am on 23 October. Mr Summerfield was seen immediately. Bloods were taken and a central line inserted into his right groin. Ketamine was administered, an arterial line was inserted, an ECG was taken and he was intubated. He was promptly transferred to the operating theatre for a laparotomy. The surgeon was Mr Alsaffar. He reports; *".... there was about 3 litres of faecal matter in the peritoneal cavity and a lot of gas. There was a 10 mm perforation in the pyloric area. The distal half of the small bowel and the right colon looked ischaemic but viable. Procedure; ...all the faecal matter was suctioned out. The pyloric perforation was closed with an omental patch....."*

Mr Summerfield was transferred to the intensive care unit. At about 7:30am he was reviewed by the Intensive Care Unit registrar, and a Code Blue was called because of heart dysfunction. Mr Summerfeldt required resuscitation but could not be revived. He was declared deceased at 7:45am.

Post-Mortem Examination

This was carried out by State Forensic Pathologist, Dr Christopher Lawrence. He reports:

> *"This 48-year-old man, Ian Patrick Summerfeldt, died as a consequence of sepsis and renal failure due to a perforated gastric ulcer. Other significant contributing factors include emphysema.*
>
> *The decedent.....presented to Queenstown Hospital with a two-day history of abdominal pain. He was taken late in the evening to North-West Regional Hospital where the ulcer was oversewn however he died the next morning. Autopsy reveals an oversewn pyloric ulcer with what appears to be a dehiscence or re-perforation*

adjacent to the ulcer. Some of the pyloric wall appears necrotic and appears to have re-perforated. There is pus in the abdomen. There are changes in the kidneys consistent with acute renal failure."

Investigation

This has included:

1. Consideration of affidavits provided by Ms Orme and Ms Millie Summerfeldt.

2. Consideration of reports provided by Registered Nurses Anu Daniel and Ashley Burgess, Nurse Practitioner Arthit Barnes and Dr Currie.

3. A review of Mr Summerfeldt's records at the WCDH and the NWRH carried out by research nurse, Ms L K Newman.

4. Consideration of a report provided by Dr A J Bell as medical adviser to the coroner.

5. Meetings attended by me, Ms Newman, Dr Bell, forensic pathologist Dr Donald Ritchey, and Dr Lawrence to review the investigation.

Dr Bell expresses these opinions:

- Sudden onset severe abdominal pain, with radiation to the shoulder tip, along with generalised tenderness of the abdomen are signs of concern.

- The first diagnoses that must be considered in patients with acute abdominal pain are those that may require surgical intervention. These include bowel obstruction and peritonitis.

- There was an underestimation of the degree of Mr Summerfeldt's illness when he first presented to the WCDH.

- The history suggests that Mr Summerfeldt was suffering the effects of increasing peritonitis when attended by ambulance officers on 22 October.

- Mr Summerfeldt's presentation at the WCDH on 22 October was strongly suggestive of a patient suffering acute renal failure, as indicated by his elevated creatinine with a proportional rise in urea levels. His relatively normal vital signs were typical of the ability of a relatively young and healthy patient to compensate for severe illness.

- In the morning of 22 October Mr Summerfeldt required immediate treatment with IV fluid, antibiotics, and emergency transfer to the nearest surgical centre.

- Mr Summerfeldt's best chance of surviving his faecal peritonitis required his immediate transfer from the WCDH to the NWRH in the morning of 22 October. However, it has to be recognised that he was suffering a life threatening condition and the prospects of surgical intervention saving his life were no greater than 50%.

Findings, Comments and Recommendations

I accept Dr Lawrence's opinion upon the cause of death. It is apparent, with the benefit of hindsight, that Mr Summerfeldt was suffering from a perforation in the pyloric area of his stomach when symptoms first presented on 20 October 2014. Regrettably, the seriousness of his condition was not appreciated when he first attended the WCDH. However, I acknowledge that, on this occasion, Mr Summerfeldt appears to have been a less than compliant patient and an extended monitoring of his health was frustrated by his self-discharge.

However, I accept the opinion of Dr Bell that when Mr Summerfeldt re-presented to the WCDH on 22 October he had increasing peritonitis after suffering a perforation of a pyloric ulcer, and was in urgent need of surgery to maximise his prospects of survival. Unfortunately this situation was not recognised when he attended at WCDH, and there was a significant delay before his eventual evacuation to the NWRH. I am not able to find that Mr Summerfeldt would have survived his peritonitis if he had been conveyed to the NWRH at the earliest opportunity on 22 October 2014. However, I am satisfied, and so find, that the delay in his evacuation did, to an appreciable extent, reduce his prospects of survival.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how his death occurred, and the particulars needed to register his death under the *Births, Deaths and Marriages Registration Act 1999*. I do not consider that the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me.

I convey my sincere condolences to Mr Summerfeldt's family and loved ones.

Dated: 3 January 2017 at Hobart in the State of Tasmania.

Rod Chandler
Coroner

APPENDIX A (CORONER'S REPORTS (XIV)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Simon Cooper, Coroner, having investigated the death of Mary Weir

Find That:

(a) The identity of the deceased is Mary Weir;

(b) Mrs Weir died in the circumstances described in this finding;

(c) Mrs Weir died as a result of a subdural haematoma due to a closed head injury following a fall from bed whilst hospitalised;

(d) Mrs Weir died on 6 September 2014 at Calvary Hospital, Hobart in Tasmania; and

(e) Mrs Weir was born in Hobart on 18 January 1934, was aged 80 years at the time of her death; she was a widow whose occupation was a retired small business owner.

Circumstances Surrounding the Death:

Mrs Mary Weir was admitted to Calvary Hospital as the result of a referral from her general practitioner on 25 August 2014. Aged 80 years her health was poor with, in particular, chronic heart failure and cellulitis of grossly swollen legs.

Upon admission, Mrs Weir was assessed by staff as being a patient at high risk of falls and, appropriately, a high risk falls prevention plan was initiated.

Subsequent to her admission an improvement in her general medical condition was noted.

Unfortunately, in the early hours of 30 August 2014, Mrs Weir suffered an unwitnessed fall in the bathroom of her room. She had, according to notes in her medical record, neither rung for assistance (as she had been instructed) nor used her four wheel walking frame (also as she had been instructed).

Nursing staff were concerned that Mrs Weir had hit her head in the fall. She was observed for four hours. Her observations were stable. At 4.50am on 30 August

2014, Mrs Weir was reviewed by a House Medical Officer who concluded, wrongly as subsequent investigations would reveal, that Mrs Weir had not suffered any injury.

Subsequent reviews later that day and the next led to notes being made in her medical records that Mrs Weir was progressing well.

At 1.20am on 1 September 2014, Mrs Weir was found by nursing staff to be unresponsive and a 'Code Blue' emergency response was initiated. She was resuscitated and administered Digoxin IV for a rapid heart rate and the anticoagulant drug Clexane (60 mg) for what was presumed to be a thrombotic stroke.

At 2.10am Mrs Weir suffered a seizure. On the advice of an intensive care specialist doctor, the anti-convulsive drug Dilantin and Amiodarone for heart arrhythmia were administered to Mrs Weir. Another seizure followed and a CT scan of Mrs Weir's brain was carried out which showed an acute left cerebral convexity subdural haematoma without mass effect. A follow up CT scan seven hours later showed no change. A neurosurgeon reviewed Mrs Weir and concluded that because there was no mass effect from the haematoma, surgery was not required.

Responsibility for Mrs Weir's care was then taken over by an intensive care specialist. Unfortunately, she continued to suffer further seizures despite her receiving anti-convulsive therapy. Her condition continued to deteriorate, developing chronic renal failure and poor cardiac function. It was concluded that further treatment was futile, life support was withdrawn, and shortly after about 3.00pm on 6 September 2014, Mrs Weir died.

A report was prepared for the Office of the Coroner and an investigation commenced pursuant to the *Coroners Act* 1995 into the circumstances surrounding Mrs Weir's death.

That investigation included, after formal identification of the body, an autopsy at the Royal Hobart Hospital. The autopsy was performed by Dr Donald Ritchey MD, MSc, FRCPA, a forensic pathologist. Dr Ritchey's opinion was that the cause of Mrs Weir's death was a subdural haematoma caused by a closed head injury sustained in a fall in a hospital. I accept this opinion.

The care afforded to Mrs Weir whilst a patient at Calvary Hospital was reviewed as part of the coronial investigation by Clinical Professor Anthony Bell MD FRACP FCICM. Prof. Bell concluded, and I accept, that no fault can be attached to the nursing staff in respect of Mrs Weir's fall.

However two issues arose from a consideration of her care. First, given her age and the fact that Mrs Weir was taking antiplatelet agents, aspirin and clopidogrel, as well as a small prophylactic dose of the anticoagulant drug Clexane, a CT scan of her brain should have been carried out on the morning of her fall. It was not, and as has already been indicated, no CT scan was carried out for two days.

Second, and of very serious concern, was the decision after the discovery of Mrs Weir being non-responsive at 1.20am on 1 September 2014, to treat her with a full dose of Clexane on the basis of a presumed stroke due to atrial fibrillation, without first carrying out a plain CT scan. It is a basic principle of treatment in the circumstances that then pertained to carry out a plain CT scan before any thrombolytic therapy or anticoagulation, so as to exclude a cerebral haemorrhage. Moreover, to have administered a full dose of anticoagulant medication where there was clear evidence of a fall and likely resultant head injury was not sound practice. By the time the CT scan was carried out, belatedly in my view, the damage caused by the full dose of Clexane resulted in significant and accelerated bleeding from the subdural haematoma on Mrs Weir's brain (which in turn caused seizures).

It seems clear in the circumstances a decision was made to treat Mrs Weir with Clexane on the basis of a presumed stroke due to atrial fibrillation. The decision was wrong. It was unsound medical practice. The making of the decision by a House Medical Officer without consulting with the appropriate treating specialist was also unsound practice.

I am satisfied that the administration of a full dose of Clexane in the circumstances outlined contributed to the cause of Mrs Weir's death.

I record that Calvary Hospital was afforded the opportunity to comment upon this case during its investigation and comment upon my proposed draft findings. The Hospital replied by indicating that it 'takes the matter very seriously', that Mrs Weir's death is the subject of a review by the hospital's Clinical Review Committee and that recommendations arising from that review would be implemented.

Comments and Recommendations:

The circumstances of Mrs Weir's death require me to remind all hospitals and medical practitioners of the need not to administer anticoagulant medication to any patient with a history of a fall without first having a plain CT scan of the patient's brain done.

In conclusion I wish to convey my sincere condolences to the family of Mrs Weir.

Dated: 7 September 2015 at Hobart in the state of Tasmania.

Simon Cooper
Coroner

APPENDIX A (CORONER'S REPORTS (XV)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Nicolle Clare Hingston

Find that:

(a) The identity of the deceased is Nicolle Clare Hingston;

(b) Nicolle was born in Burnie on 8 January 2000 and was aged 14 years;

(c) Nicolle died at the North West Regional Hospital (NWRH) in Burnie on 9 August 2014;

(d) The cause of Nicolle's death was cardiorespiratory arrest resulting from end-stage pulmonary hypertension and cor pulmonale secondary to congenital heart disease (ventricular septal defect).

Background

Nicolle was a secondary student. She resided with her parents and sister in Penguin. She had a complex cardiac history as evidenced by this chronology:

- At aged 6 months Nicolle was diagnosed with ventricular septal defect, colloquially known as a 'hole in the heart'. She was referred to consultant cardiologist, Dr Brian Edis at the Royal Children's Hospital (RCH) in Melbourne, who confirmed the diagnosis but noted the absence of any evidence of heart failure. Thereafter she was reviewed every 6 months, initially by Dr Edis and then by paediatric cardiologist, Dr L Fong, also from the RCH.

- In October 2001, when Nicolle was aged 21 months, testing showed that she had developed pulmonary hypertension with right ventricular hypertrophy. One month later she underwent the surgical closure of her ventricular septal defect at the RCH. Her post-operative course was complicated by significant pulmonary hypertension.

- Following her surgery Nicolle was reviewed on a 6 monthly basis by consultant cardiologists up to March 2007. At about this time there was an incident at school when Nicolle collapsed whilst training for Little Athletics. She was seen by Professor Dan Penny, Director of Cardiology at the RCH, who recognised that she was becoming symptomatic with decreasing exercise performance and episodes of

fainting. Testing at the RCH confirmed significant pulmonary hypertension and she was placed on bosentan and Warfarin.

- The 6 monthly specialist reviews continued over the following years. During this time Nicolle participated in a 12-month clinical trial of bosentan which required her to take the medication thrice daily. According to her parents Nicolle did not appear to benefit from this trial.

- In early 2014 it was noted that Nicolle was becoming lethargic. There was a deterioration in her exercise tolerance and her general wellbeing. She was prescribed Sildenafil which seemed to be beneficial. She was also given iron supplements in recognition of the commencement of menstruation.

- Nicolle's father David reports that around 4 and 5 August Nicolle was menstruating and had *"very heavy bleeding."* In accordance with earlier medical advice her Warfarin was stopped.

Circumstances Surrounding the Death

At about 4.00am on 7 August Nicolle began to have difficulties breathing. She complained to her father that she had *"a very tight chest."* Mr Hingston called for an ambulance. However, by the time it arrived Nicolle's symptoms had abated, and her breathing had returned to normal. She was reluctant to go to hospital and the ambulance left. However, at about 7.00am Nicolle collapsed when she was walking to the car with her father and sister. Mr Hingston says that *"she lost consciousness for a minute or so."* He then drove her to the NWRH where she was admitted to the Paediatric Unit. She was assessed to be significantly anaemic with haemoglobin of 52. However, her vital signs were stable and she did not have any signs of significant cardiorespiratory compromise. She was transfused two units of packed red blood cells with no deterioration in her cardiorespiratory signs. Nicolle stayed in hospital overnight. Mr Hingston says that the next day *"she started to look better"* but that she *"was still menstruating heavily.....as my wife was taking her to the bathroom regularly."*

At the NWRH Nicolle's care was overseen by Consultant Paediatrician, Dr Bert Shugg. He was concerned by Nicolle's anaemic state and was unsure of its cause. He did not think it could be explained by her menstrual blood loss. Dr Shugg raised with interstate specialist, Associate Professor Michael Cheung, whether it may be attributable to the bosentan but he did not believe this to be likely. In the afternoon of 8 August Mr and Mrs Hingston were advised that they could take Nicolle home.

August 9 was a Saturday. Nicolle got up to go to the toilet but complained of still feeling tired. She went back to bed and Mrs Hingston then gave her breakfast-in-bed. This was at about 9.00am. Sometime between 11.00 and 11.30am Nicolle started having breathing difficulties. She again complained of a tight chest. Mr Hingston called the ambulance. At the moment it arrived Nicolle stopped breathing. The paramedics immediately began CPR. A second ambulance arrived to assist. Nicolle was then conveyed to the NWRH with CPR being continued en route. The ambulance arrived at the Emergency Department at

12.50pm. Attempts to revive Nicolle were unsuccessful. She was declared deceased at 1.04pm.

Post-Mortem Examination

This was carried out by pathologist, Dr Terry Brain. In his opinion the cause of Nicolle's death was cardiorespiratory arrest resulting from end-stage pulmonary hypertension and cor pulmonale secondary to congenital heart disease (ventricular septal defect).

Investigation

This has been informed by:

1. An affidavit obtained from Mr Hingston.

2. A report provided by Mr Dominic Morgan, Chief Executive Officer of Ambulance Tasmania, along with Patient Care Reports of Ambulance Tasmania.

3. A report from Dr Shugg.

4. A review of Nicolle's records at the NWRH carried out by Research Nurse, Ms L K Newman.

5. A report upon Nicolle's medical care and management compiled by Dr A J Bell, medical adviser to the coroner.

6. Meetings to monitor the investigation attended by myself, Ms Newman, Dr Bell and Forensic Pathologists, Dr Christopher Lawrence and Dr Donald Ritchey.

In his report Dr Bell includes this advice:

- That in his opinion Nicolle's sudden death appears to be related to continued blood loss and under transfusion in a patient with significant pulmonary hypertension.

- That Nicolle's treatment on 7 August with transfusion was appropriate. Before her discharge on that day Nicolle's vital signs appeared to be stable and her haemoglobin level had been increased to 71 g/L. However, a blood test done on 9 August showed a haemoglobin level of 58 g/L suggesting continuing blood loss.

- That patients with pulmonary hypertension who experience arrest rarely survive.

- That he agrees with the post-death assessment made by Dr Shugg and expressed in these terms: *"In the retrospective analysis of Nicolle's admission to the NWRH I consider that it may have been appropriate to transfuse her to a higher haemoglobin level as the volume of menstrual blood loss was under revealed but the risks of transfusion were understood by the medical staff and Nicolle's parents."*

- That also with the benefit of hindsight it may have been prudent to have kept Nicolle in hospital for a second night to monitor her blood loss and haemoglobin level.

- That in his opinion the level of care provided by the NWRH was of a good standard.

Findings, Comments and Recommendations

I accept Dr Brain's opinion upon the cause of death.

It is apparent that Nicolle's menstruation and consequential blood loss adversely impacted upon her underlying and pre-existing pulmonary hypertension. It is apparent too, as Dr Shugg has since acknowledged, that the extent and continuation of Nicolle's blood loss was not fully appreciated when she presented at the NWRH on 7 August 2014. In the result she was under-treated during this admission and perhaps prematurely discharged. However, this is an assessment made with the benefit of hindsight and does not cause me to be critical of the hospital care provided to Nicolle. Rather, I accept the opinion of Dr Bell that overall the level of care provided at the NWRH was of an acceptable standard.

Nicolle's death has been a tragic event and I extend my sincere condolences to her family and loved ones for their loss.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how her death occurred, and the particulars needed to register her death under the *Births, Deaths and Marriages Registration Act 1999*. I do not consider that the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me.

Dated: 17 November 2016 at Hobart in the State of Tasmania.

Rod Chandler
Coroner

APPENDIX A (CORONERS REPORTS (XVI)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Heather Mary Bird

Find that:

(a) The identity of the deceased is Heather Mary Bird;

(b) Mrs Bird was born in Hobart on 31 March 1948 and was aged 66 years;

(c) Mrs Bird died at the Royal Hobart Hospital (RHH) in Hobart on 12 July 2014;

(d) The cause of Mrs Bird's death was a retroperitoneal haemorrhage due to anticoagulation following right cerebrovascular infarct following intra-arterial cannulation of the right carotid artery.

Background

Mrs Bird had been married to Kevin James Bird for 48 years. They resided at Murdunna and had 3 children. She had a complex medical history including a perforated diverticulum requiring extended hospitalisation, a stroke with significant residual effects, Type 2 diabetes, obesity and severe rheumatoid arthritis requiring the use of a wheelchair.

Circumstances Surrounding the Death

On 5 June 2014 Mrs Bird was an emergency presentation to the RHH with vomiting and abdominal pain. She was admitted to a ward. A CT scan demonstrated a small bowel obstruction but this resolved within 36 hours. However, clinical signs suggested a pneumonic process and by 7 June a diagnosis of left lower lobe pneumonia (with possible aspiration) was made. Mrs Bird made a steady improvement over the following days and by 13 June was keen to go home. However, her temperature spiked overnight on 14/15 June. The following day a decision was made to place a central venous catheter (CVC) into the right internal jugular vein. The purpose was to facilitate blood sampling and to enable the administration of intra venous fluids and antibiotics to treat the pneumonia. The procedure was undertaken by consultant anaesthetist, Dr Peter Peres, under ultrasound guidance. It proved to be difficult and required multiple attempts. The records show some confusion

upon the site of insertion, the medical record indicating the left internal jugular vein was used whilst the radiology shows it to have been the right jugular vein. The colour of blood extracted via the line was considered by the anaesthetic registrar to be consistent with venous blood. A sample of blood was subject to a blood gas analysis at 5.02am on 17 June which clearly showed it to be arterial and not venous. However, this was not recognised by the medical staff at this time. Over the following days Mrs Bird's condition deteriorated. On 18 June she had a further temperature spike and became tachypnoeic (quick and shallow breathing) necessitating a medical emergency team (MET) call. The following day medical staff met with family members to discuss Mrs Bird's goals of care. Despite a poor prognosis both Mr and Mrs Bird were adamant that full treatment be maintained.

By 23 June there had been some improvement in Mrs Bird's condition. However, that morning it was noted by the radiology registrar, on a follow-up x-ray, that the CVC had been mal-positioned and was in the carotid artery and extending to the aorta. Medical staff met with Mr and Mrs Bird and explained the complication and that it had not been realised earlier. They were told that the catheter had to be removed and that this procedure involved some risk including the risk of stroke. The following day the CVC line was removed without incident. However, imaging at the time showed that a fibrin sheath had formed in the carotid artery and Mrs Bird was commenced on a full anti-coagulant dose of clexane to reduce the risk of cerebral embolism and stroke.

On 27 June a CT scan of Mrs Bird's brain showed a sub-acute stroke in the primary motor cortex. The plan was to continue the therapeutic clexane and to ready Mrs Bird for transfer to the rehabilitation ward. A MRI scan on 30 June confirmed that Mrs Bird had suffered a stroke which was *"likely secondary common carotid artery line"* thereby confirming an association with the CVC's insertion. On 2 July Mrs Bird was transferred to the rehabilitation ward. Imaging on 7 July showed that the fibrin sheath was still present and was mobile.

On 9 July a MET call was made for Mrs Bird due to a blood pressure of 90/55 mmHg and a tachycardia of 120 bpm. Her Glasgow Coma Score had fallen and there was no urine output for 2.5 hours with a urinary catheter in situ. The MET team stood down after the blood pressure rose to 110 mmHg systolic following IV fluid treatment. Later that day she had a percutaneous inserted central catheter (PICC) line put in place. At 4.45pm the resident medical officer reviewed Mrs Bird. Her blood pressure was 110/70 mmHg, heart rate was 90bpm and respiratory rate was 18bpm. At 7.30pm Mrs Bird was reviewed by an intern. A tender abdomen was noted with a large haematoma. The intern was concerned that Mrs Bird had either an intracerebral haematoma or an intra-abdominal haematoma. Her condition was discussed with the medical registrar and the CT radiographer. At 9.35pm a further MET call was made, again because of hypotension and tachycardia. It was noted by an attending doctor that her haemoglobin was 75 being *"likely dilutional."* It was recorded that a transfusion would be considered if her condition deteriorated. She was then transferred to an acute ward for one-on-one nursing care. Over the next few hours Mrs Bird's condition stabilised but at about 3.30am the next day she again became hypotensive and tachycardic. Her blood pressure was 80/50 mmHg and her heart rate was 130bpm. A diagnosis of septic shock was made and further IV fluid was administered. The blood pressure rose to 102/83 mmHg and the pulse rate was 137bpm. The intensive care unit

(ICU) registrar was contacted and advised that Mrs Bird required admission to the High Dependency Unit (HDU). The registrar agreed to review Mrs Bird.

That review took place at 7.20am on 10 July. Mrs Bird remained in shock. Blood pressure was 95/50 mmHg, heart rate was 130 bpm and haemoglobin was noted to be 71 g/L. A bolus of intravenous fluid was administered. Mrs Bird remained in the rehabilitation ward. At 9.46am a further MET call was made due to hypotension and tachycardia. On this occasion her blood pressure was unrecordable, she was peripherally shut down, had no radial pulse and her haemoglobin was found to be 35. An abdominal examination revealed a dense left-sided mass. She was transferred to ICU.

In ICU Mrs Bird was rapidly resuscitated and treated for shock. An urgent CT angiogram of the abdomen showed a large retroperitoneal collection thought to be a haematoma. An arterial bleeding source was demonstrated on the left side. Anticoagulation was reversed and blood transfusion given. Mrs Bird's condition continued to deteriorate over the following two days and she died at 9.45am on 12 July 2014.

Post-Mortem Examination

This was carried out by State Forensic Pathologist, Dr Christopher Lawrence. In his opinion the cause of Mrs Bird's death was a retroperitoneal haemorrhage due to anticoagulation following right cerebrovascular infarct following intra-arterial cannulation of the right carotid artery.

I accept this opinion.

In his report Dr Lawrence includes this comment: *"Autopsy reveals a large retroperitoneal haematoma which appears to be arising from lower intercostal or lumbar arteries. There is an old left occipital stroke, a subacute right occipital stroke and an acute right frontal stroke. There does not appear to be any residual fibrin in the right carotid artery. The decision to anti-coagulate after removal of the cannula in the right carotid artery to prevent a further stroke probably contributed to the intra-abdominal bleeding. However, it is not exactly clear what caused the lumbar/intercostal arteries to bleed. I cannot see any real evidence of trauma."*

Investigation

The investigation has been informed by:

- Medical reports/statements provided by Dr Peres, Professor Jens Froelich, Dr Anthony Beasley, Clinical Associate Professor Andrew Turner, Dr Andrea Laborde and Mr David Cottier.

- A statement from Mr Craig Watson as Executive Director of Services - Southern Region, Tasmanian Health Service.

- An affidavit from Mr Bird.

- A review of Mrs Bird's records at the RHH carried out by Research Nurse, Ms L K Newman.

- A report upon Mrs Bird's medical management and treatment compiled by Dr A J Bell as medical adviser to the coroner.

- Meetings to review the investigation attended by myself, Ms Newman, Dr Bell, Dr Lawrence and Forensic Pathologist, Dr Donald Ritchey.

In his report Dr Bell:

1. Advises that arterial puncture occurs in 3 to 15% of venous access procedures. As such it is a known complication and does not necessarily represent sub-standard medical practice.

2. Advises that unrecognised arterial cannulation with subsequent dilation and catheter placement within the artery can be associated with life-threatening haemorrhagic and neurologic complications.

3. Opines that the cerebrovascular brain injury or stroke suffered by Mrs Bird and observed on 27 June was attributable to a portion of the fibrin sheath within the carotid artery breaking off and causing a vascular occlusion in the brain.

4. Advises that the catheter used was not the standard type used for insertion into the jugular vein. It had a smaller internal diameter than the usual catheter and may have produced a lesser flash back of blood when inserted into the artery. As a result the recognition of the arterial insertion may have been less obvious.

5. Opines that it represents poor medical practice to use a catheter which was not designed for the insertion site.

6. Points out that the radiologist who reported on the check x-ray of the catheter's position following its insertion failed to recognise its misplacement. This represented poor medical practice.

7. Advises that immediate recognition and management of an arterial puncture usually prevents subsequent complications.

8. Retroperitoneal haemorrhage is most commonly seen in association with patients with anticoagulation therapy, bleeding abnormalities or haemodialysis and represents one of the most serious and potentially lethal complications of anticoagulation therapy.

9. Opines that it was an error at the time of the second MET call to consider Mrs Bird's fall in haemoglobin from 138 g/L to 71 g/l to be 'dilutional,' particularly as she was

being anti-coagulated. Mrs Bird was obviously bleeding but there was no visible blood. In these circumstances retroperitoneal haemorrhage should have been considered as a likely explanation.

10. Opines that the MET service was deficient because of:

    a. The failure to seek appropriate consultant input. Dr Laborde, as a staff specialist in Rehabilitation Medicine, was not sufficiently experienced in acute medicine to provide such input.

    b. Its failure to seek appropriate consultant input at the time of the second and third MET calls.

    c. Its delay in making the diagnosis of haemorrhagic shock.

    d. The four hour delay in having Mrs Bird medically reviewed following the diagnosis of haemorrhagic shock being made.

11. Advises that haemorrhagic shock represents a medical emergency. When this diagnosis was made Mrs Bird required immediate admission to ICU for emergency treatment including the cessation of her anticoagulation.

12. Advises that the failure to promptly diagnose Mrs Bird's haemorrhagic shock and have her appropriately treated in ICU denied her any prospect of survival.

Findings, Comments and Recommendations

It is evident that Mrs Bird's admission to the RHH on 5 June 2014 was a consequence of a small bowel obstruction. This was promptly diagnosed and successfully treated. However, it seems clear that secondary to this condition Mrs Bird developed pneumonia which, in its initial stages, was successfully managed but had relapsed by 15 June. This led to the need for the placement of a CVC. It is at this point Mrs Bird's medical care and management went awry directly leading to her death. In this context I make these findings:

- The CVC was wrongly positioned in the carotid artery rather than the jugular vein.

- The radiologist wrongly reported the catheter to be in the correct position.

- The anaesthetic registrar wrongly considered the sample of blood taken following the CVC insertion to be venous when it was arterial.

- A blood gas analysis of the sampled blood performed at 5.02am on 17 June clearly indicated it to be arterial and not venous. This was not recognised by the medical staff at this time thus no steps were taken to remove the catheter and thereby reduce the risk of any associated complications.

- Chest radiographs taken daily for four days from 16 June were not reported upon until 24 June. Earlier reporting would have identified the malposition of the CVC and led to its earlier removal.

- Eight days were allowed to elapse before the malpositioned catheter was removed. By this time a fibrin sheath had developed in the carotid artery necessitating anticoagulation treatment to reduce the risk of cerebral embolism and stroke.

- Notwithstanding the anticoagulation Mrs Bird suffered a stroke which was directly attributable to the fibrin sheath which had developed in her carotid artery.

- There was a delay, most particularly on the part of the MET team, in recognising that Mrs Bird was suffering from a retroperitoneal haemorrhage and shock attributable to her anticoagulation. This delay reduced the prospect of her being successfully treated for this condition. Her death followed.

I accept that the malpositioning of a CVC is a known complication of that procedure and I make no criticism of it in this instance. However, thereafter a litany of errors and shortcomings followed which I have set out above and which ended in a death which was almost certainly preventable.

In his statement Mr Craig Watson has advised me that Mrs Bird's death was the subject of review by the RHH's Death Review Committee and that such review did not lead to any recommendations being made. To my mind this is an extraordinary outcome given the multiple failings associated with Mrs Bird's death. In the very least it is my view that this death highlights serious deficiencies associated with the hospital's MET team and it is my **recommendation** that the RHH initiate a review of that team with a focus upon consultancy input and supervision.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how her death occurred and the particulars needed to register her death under the *Births, Deaths and Marriages Registration Act 1999*. I do not consider that the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me.

I convey my sincere condolences to Mrs Bird's family and loved ones.

Dated: 20 March 2017 at Hobart in the State of Tasmania.

Rod Chandler
Coroner

APPEDIX A (CORONER'S REPORTS (XVII)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

(These findings have been de-identified in relation to the name of the deceased, family, friends and others by direction of the Coroner)

I, Olivia McTaggart, Coroner, having investigated the death of Mr D

Find, pursuant to Section 28(1) of the Coroners Act 1995, that:

a) The identity of the deceased is Mr D;

b) Mr D died after collapsing at his home in Southern Tasmania in December 2015 after injecting methamphetamine in the circumstances further described in this finding;

c) The cause of Mr D's death was hypoxic encephalopathy complicating a cardiac arrest caused by methylamphetamine toxicity;

d) Mr D died in December 2015 at the Royal Hobart Hospital in Tasmania; and

e) Mr D was born in Hobart and was aged 21 years.

In making these findings I have had regard to the evidence gained in the investigation into Mr D's death. The evidence comprises an opinion of the forensic pathologist who conducted the autopsy, results of toxicological samples, relevant police and witness affidavits, medical records and forensic evidence.

Mr D and his partner lived in Southern Tasmania. They had a daughter. Mr D worked as a labourer.

Mr D had suffered from eczema and asthma as a child and continued to suffer these conditions until his death. He smoked cigarettes and was a casual user of cannabis. The evidence indicates that in the months before his death he commenced to use methylamphetamine. However, he did not disclose his use to his partner or close family members.

On an evening in December 2015, Mr D and his uncle were at Mr D's house, consuming some alcoholic drinks together. Subsequently, at about 11.30pm, they left the residence to purchase alcohol and/or cigarettes. In the course of the journey, Mr D asked his uncle to take him to a

residence in Bridgewater. His uncle did so and waited in the car whilst Mr D entered the residence. At the time Mr D was at the residence, his uncle observed a vehicle arriving with the occupant of the vehicle going inside. Mr D was in the residence at Bridgewater for about five minutes. He did not communicate to his uncle his reasons for visiting the residence. At about 1.00am they both returned to Mr D's home.

At about 2.15 am, Mr D's uncle was in the shed on the property waiting for Mr D to return from inside the house. At that time he heard Mr D's partner yelling *"He can't breathe, he can't breathe".* He went inside and saw Mr D in the shower starting to collapse and turn blue. Mr D's partner stated in her affidavit for the investigation that she had been asleep, and had awoken to find Mr D struggling to breathe and heading to the bathroom where he collapsed in the bath tub. She telephoned for an ambulance and Mr D's uncle commenced CPR upon Mr D until the ambulance arrived and took over resuscitation efforts.

Mr D was conveyed to the Royal Hobart Hospital in the ambulance and resuscitation attempts continued en route.

Ongoing life support and treatment was continued at the Intensive Care Unit (ICU) of the Royal Hobart Hospital until clinical changes indicated Mr D's condition had deteriorated. Clinical brain testing was conducted which confirmed Mr D was brain dead.

Mr D's life support was switched off at 11.27am, two days after his admission to ICU, and he was pronounced deceased.

The results of toxicology testing of Mr D's ante mortem blood showed an elevated methylamphetamine concentration of 0.4 mg/L.

An autopsy was conducted by forensic pathologist, Dr Donald Ritchey. He formed the opinion that the cause of death was hypoxic encephalopathy complicating a cardiac arrest caused by methylamphetamine toxicity. I accept his opinion as to cause of death.

Mr D denied his use of methylamphetamine to his partner and to other family members, all of whom were concerned about him. Several months before his death Mr D was approached by a close family member who suspected that he had commenced to use "ice" (being methylamphetamine). He did admit to that family member that he had used "ice". However, he stated that he no longer did so as he was aware of "what it did to people" and that it was expensive. From then on, Mr D did not talk further to that family member, or anyone else, about his drug-taking. His partner stated that he had become secretive and there had been a change for the worse in his behaviour in the months before his death.

A thorough investigation into the circumstances of Mr D's death was conducted by police. Police officers located drug paraphernalia at the property and in his car consistent with his use of methylamphetamine.

I am satisfied that, at a time close to death, Mr D ingested a quantity of methylamphetamine. I am not able to determine how this substance was ingested but it had the effect of causing his death. I also am not able to find positively that he obtained the methylamphetamine during his visit to the Bridgewater residence. I suspect that he did so but he may also have already been in possession

of the substance. I am not able to determine who supplied Mr D with the fatal quantity of the substance.

Comments and Recommendations

In recent findings concerning the death of two young males[1] as a result of ingesting methylamphetamine, I observed that the substance stimulates the central nervous system, producing behavioural and physiological effects. It has a high potential for abuse and addiction. It may result in fatality in some individuals when used alone on a single occasion.

I also commented in those findings that of particular concern is the higher purity crystalline form of methylamphetamine, known as "ice". This is the substance most likely consumed by Mr D before his death. The increased availability and use of crystal methylamphetamine have been associated with increased regular use and harms.

Mr D was a young man with a supportive family, an infant child and prospects for a full life. His life ended tragically and prematurely as a result of his ingestion of a quantity of methylamphetamine, to which he had become addicted in the months before his death. It appears from the evidence that he wished to break his addiction but was unable to do so.

I convey my sincere condolences to Mr D's family and loved ones.

Dated: 11 of November 2016 at Hobart in the State of Tasmania.

Olivia McTaggart
Coroner

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2016 TASCD 312 Paul Marcus White
2016 TASCD 313 Tyler John Broomhall

APPENDIX A (CORONERS REPORTS (XVIII)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

IN THE MATTER OF THE *CORONERS ACT* 1995

AND

IN THE MATTER OF AN INQUEST TOUCHING THE DEATH OF ALEXANDER PASINSKI

FINDINGS, RECOMMENDATIONS AND COMMENTS of Coroner Rod Chandler following an inquest held in Launceston on 2 and 15 June 2016.

August 2016

PREAMBLE

On 25 February 2014 Alexander Pasinski died at the Launceston General Hospital ('LGH') in Launceston. At this time Mr Pasinski was an inpatient at Northside, a division of the LGH which provides treatment and care for persons suffering from mental disorders. His presence in this facility was pursuant to a Continuing Care Order made on 27 December 2013 and confirmed by the Mental Health Tribunal on 9 January 2014.

On 2 and 15 June 2016 an inquest was held concerning Mr Pasinski's death and these are my findings arising from it.

BACKGROUND

Mr Pasinski was born at Ouse in Tasmania on 30 May 1954 and was aged 59 years. He was one of five children. He was educated in Tasmania but did not advance beyond Year 8. He worked in unskilled positions, initially in Tasmania and then in South Australia. However, for many years prior to his death he had been unemployed and dependent upon a disability pension. He was unmarried and did not have any children.

Mr Pasinski had a long history of psychiatric illness. That history is summarised in a report provided to the inquest by consultant psychiatrist, Dr Ian Sale, and made following a review of Mr Pasinski's medical and hospital records. The salient features of that history follow:

- In 1988 Mr Pasinski had his first contact with mental health services when he was hospitalised in South Australia following a self-harm incident. About two years after this event he returned to Tasmania where he sought psychiatric treatment. He initially consulted psychiatrist Dr Rosemary Schneider and then became a patient of psychiatrist, Dr Ian Martin. Documentation from Dr Martin indicates that problems with sexual behaviour were the main focus of treatment at this time.

- In 1993 Mr Pasinski was admitted to the LGH because of concerns about the risk of suicidal behaviour. He remained in hospital for three weeks where he was diagnosed with a non-specific depressive disorder in a setting of dependent personality traits. He was briefly re-admitted to the LGH later that year after he had taken two overdoses of prescribed medication. His self-harm behaviour was viewed as largely attention seeking in nature, aimed at obtaining hospital admission. He was discharged into Dr Martin's care.

- In 1994 Mr Pasinski again attended the LGH because of motor restlessness. This problem was assessed as reflecting a side-effect of the medication prescribed by Dr Martin.

- In 2000 Mr Pasinski was referred to psychiatrist, Dr S J Hyde when Dr Martin ceased practice. Dr Hyde remained involved in Mr Pasinski's care through to his hospital admission in late 2013.

- Dr Hyde reported a long history of symptoms of anxiety, depression, irritability, and difficulty with relationships. Borderline intelligence was also advised. He also described how Mr Pasinski complained of various physical symptoms, e.g. blackouts. He also noted complaints of suicidal thoughts.

- From Dr Hyde's viewpoint there was a deterioration in Mr Pasinski's well-being in mid-2013. Initially there were complaints of altered taste and dry mouth and later an altered sensation of smell. For this latter condition he was assessed by an ENT specialist but nothing unusual was found. Mr Pasinski also experienced weight loss.

- As of late 2013 psychotropic medications prescribed to Mr Pasinski comprised Nitrazepam, a benzodiazepine used for night sedation and Imipramine, a tricyclic antidepressant.

- On 11 December 2013 Mr Pasinski self-presented to the LGH complaining of being unable to smell or taste. He also complained of being unable to sense hot or cold. He indicated that he would take a drug overdose if he was unable to obtain a solution to these problems. He also said that he wanted to die, that he was paranoid and that he had lost 10 kg of weight over the previous five months. He was admitted to Northside and a provisional diagnosis was made of an acute psychotic disorder or depression with psychotic features. Within a short time of his admission Mr Pasinski was overheard voicing suicidal threats. This led to him being transferred to the high dependency unit ('HDU'). This period of hospitalisation lasted 12 days during which Mr Pasinski continued to complain of lack of sensation, that he did not exist and that he was unable to feel pain. He also continued to claim an intention to kill himself or to abscond from the unit.

- By the time of his discharge on 23 December 2013 Mr Pasinski's psychotropic medication arrangements had been revised. These now comprised Imipramine 75 mg daily, Nitrazepam 10 mg at night and the anti-psychotic agent Quetiapine 25 mg twice daily.

CIRCUMSTANCES LEADING TO DEATH

In the early evening of Christmas Day 2013 officers of Tasmania Police took Mr Pasinski to the LGH following an incident where he claimed to have jumped from a bridge into the Tamar River. An examination made at this time noted him to be restless, dishevelled, irritable and abusive. It was decided to re-admit him as an involuntary patient pursuant to an Initial Order made under s24 of the *Mental Health Act* 1996. (This order was followed by the Continuing Care Order referred to in the preamble to these findings.) In Northside Mr Pasinski was assessed by a locum psychiatrist who considered schizophrenia a distinct likelihood. A trial of the antipsychotic Paliperidone was commenced.

Mr Pasinski continued to present a challenge with aggressive and disruptive behaviour. He continued to voice bizarre somatic complaints: e.g. *"my guts have disappeared."* Sedative medications were employed. On 29 December oral Risperidone was commenced.

In his submissions counsel-assisting provides a summary of Mr Pasinski's presentation over the following weeks. I adopt that summary. It follows:

> *"Ongoing psychiatric assessments during his admission revealed increasing evidence of a co-morbid depressive illness with melancholy features along with psychotic symptoms including a thought disorder, delusional beliefs which were both persecutory and nihilistic. His nihilistic delusions for example included that he had no feeling in his body or head, that he was not real, that he had no arms or legs (and) questioning whether others around him were in fact real or not. These delusions and disorganised thinking were associated with a decreasing capacity for self-care, a demonstrable lack of insight, depressive and suicidal thinking, aggression and disinhibition at times.....He remained what Dr Sale referred to as a 'diagnostic dilemma'."*

Various treatment regimens were employed over this period. On 12 January 2014 the tricyclic antidepressant agent Imipramine was switched to Amitriptyline, another tricyclic agent. The dosage was increased to 150 mg daily. It was at about this time that electroconvulsive therapy (ECT) was discussed as a treatment option. It was first administered on 31 January after approval had been obtained from the Guardianship Board. It was then continued three times weekly and in all was administered on 10 occasions. However, it seems that there was no apparent benefit from the procedure. Close nursing observations were maintained and were on a one-for-one basis at the time of death as Mr Pasinski was then staying in the High Dependency Unit. ('HDU')

On Tuesday, 25 February 2014 registered nurse Dane Flynn was assigned to Mr Pasinski's care. He had cared for Mr Pasinski previously and he was well known to

him. Nurse Flynn reports that at about 8.00 am Mr Pasinski got out of bed and went to the bathroom. He then urinated over himself and the floor before returning to his bed. Nurse Flynn asked him to return to the bathroom, change his clothes and have a shower. Mr Pasinski did not respond to this request. He remained in bed for a further 15 minutes before his breakfast tray was delivered to the unit. He then got out of bed, looked at his breakfast, and then stood up and paced the room. After this Mr Pasinski began eating some Sultana Bran. There was also some toast on the tray and Nurse Flynn spread it with margarine and Vegemite. Mr Pasinski took a piece of the toast and began eating it as he walked around the room. He then returned to the table, sat down and continued eating the piece of toast. However, before he had finished that piece he picked up another and also placed it in his mouth. At this time Nurse Flynn suggested that he finish eating the first piece before beginning the second but there was no response to this suggestion. Mr Pasinski then stood up and began pacing the room again. Nurse Flynn then heard him gagging. He suggested that he go with him to the bathroom to spit out some of the toast. This advice was ignored. Mr Pasinski then gagged again. By this stage Nurse Flynn had sought assistance from another colleague, Nurse Georgia Freeman. Both nurses tried to encourage Mr Pasinski to spit out the food and were rubbing his back. It was then noted that his face had become pale and that he was unable to stand unaided. At this point the nurses assisted Mr Pasinski to the floor and placed him in the recovery position. A Code Blue was called. Mr Pasinski remained conscious and Nurse Flynn was able to open his mouth and remove some of the toast. At one stage he stood up again and began walking around unaided. When the Medical Emergency Team ('MET') arrived Mr Pasinski was again placed in the recovery position and oxygen was administered. The MET then monitored Mr Pasinski for about 15 minutes before leaving the unit. MET's attendance had been supervised by Dr Scott Parkes, the Director of the Intensive Care Unit ('ICU'). He recommended that Mr Pasinski remain in Northside and be closely supervised in its HDU.

From 9.00 am on this day a weekly Clinical Review meeting was being held in Northside. It was being chaired by Dr Franco Giarraputo, the consultant psychiatrist with clinical responsibility for Northside's inpatients. Also present was Dr Ben Elijah, the unit's Clinical Director. At the meeting Mr Pasinski's treatment plan was revised and included the decision to cease the ECT, to replace the tricyclic antidepressants with a more contemporary antidepressant and to cease all benzodiazepines. However, it seems that these changes were not in response to the choking incident, although both Drs Giarraputo and Elijah were aware of it. In fact no consideration was given as to whether that event in itself required a re-assessment of the appropriate care and treatment for Mr Pasinski. In particular the choking incident did not provoke a decision to introduce a soft diet for Mr Pasinski pending a further investigation of the event and its possible cause(s).

Mr Pasinski largely spent the remainder of the morning in his room, seemingly asleep. There was another occasion when he went to the bathroom and again urinated on his clothing and the floor. On Nurse Flynn's prompting he showered and changed his clothes.

Mr Pasinski's lunch was delivered to the unit at about noon. It included a chicken dish. Nurse Flynn observed Mr Pasinski through the window in the adjoining office. He saw him begin his meal by picking pieces of chicken from a bone and placing them in his mouth. He thought that he appeared to be eating normally. He was then seen to leave the table and go to his room. He then returned to his meal. Mr Pasinski continued eating but made two further brief visits to his room. Nurse Flynn then observed him again sit down at the table and then noticed *"Alex place his head down, he was leaning forward. I didn't think it looked right."* Nurse Flynn then left the office to attend Mr Pasinski. Nurse Janita Roberts was with him. Mr Pasinski was still at the table and was gagging. The nurses encouraged him to try and regurgitate the food in his mouth. Nurse Flynn assisted him to stand but he then appeared to lose consciousness. A Code Blue was called and Mr Pasinski was laid on the floor in the recovery position. Nurse Flynn attempted to remove food from his mouth but was unable to do so because his jaw was clenched shut. (It was Dr Parkes' evidence that the clenched jaw was probably attributable to Mr Pasinski suffering an hypoxic event.)

The MET responded immediately and arrived at Northside as soon as it could. It comprised Dr Armit Gangli, an ICU registrar and two ICU nurses. Dr Gangli was unable to clear the airway although by this time Mr Pasinski's jaw had relaxed. Specialist Intensivist Dr Vikram Patil then arrived and took charge of the situation. He attempted to clear the airway using a pair of Magill forceps but could not do so whilst Mr Pasinski was lying on the floor. He was then lifted onto a bed where Dr Patil was able to obtain a better view of his airway. He could see that it was completely obstructed by a large mass of meat which he was able to remove piece by piece with the forceps. He describes the mass of meat *"as being equivalent to a 'fistful,' ie. a 50-60 millimetre pulp mass, which consisted of multiple pieces of meat partially mashed or chewed up. It was solid and compacted."* It needs to be noted that Northside was equipped with a suction device but Dr Patil advised that its use *"would have been pointless because of the size and compaction of the solid mass."*

CPR, which had been initiated by Nurse Flynn and his nursing colleagues, was maintained for approximately 30 minutes but Mr Pasinski could not be revived. He was pronounced deceased at 1.15 pm.

POST-MORTEM EXAMINATION

This was carried out by forensic pathologist, Dr Donald Ritchey. In his opinion the cause of Mr Pasinski's death was consistent with asphyxia due to choking on food. Significant contributing factors were a clinical history of major depression and paranoid schizophrenia. Dr Ritchey comments; *"The finding of food in the posterior pharynx adjacent to the airway supports the clinical observation of apparent asphyxia due to choking on food."* He further notes; *"Significant natural disease was not identified at autopsy."*

WAS DEATH ACCIDENTAL OR INTENTIONAL?

The circumstances surrounding Mr Pasinski's death, including his medical history and the choking incident at breakfast, raise the question whether his death was the consequence of a deliberate act on Mr Pasinski's part to take his own life. On this subject Dr Sale has made these observations:

- That choking incidents, including fatalities, are not rare for persons who suffer a significant mental disorder or are in a psychiatric facility.
- That whilst threats of self-harm had been a prominent feature of Mr Pasinski's history those threats appeared to have been a means of prompting the concern of others.
- Studies indicate that risk factors associated with choking as a cause of death include polypharmacy, age, the presence of organic conditions and 'fast eating.' As to this latter factor it was the evidence of Mr George Pasinski, a brother of the deceased, that Mr Pasinski ate his food *"very very fast,"* on one occasion he told him to *"slow it down"* but that it was a habit which *"was in his character."*
- Mr Pasinski was being prescribed Amitriptyline, a tricyclic anti-depressant, along with antipsychotics Paliperidone and Chlorpromazine. Dr Sale says that studies suggest that these antipsychotics may have an adverse impact upon the mechanism of swallowing. Further he says that a side-effect of Amitriptyline can be a drying of secretions including dry mouth which may impact upon swallowing.

Dr Sale provides this concluding opinion: *"While the possibility that choking was self-induced cannot be entirely excluded, the more probable explanation is that this man's choking was accidental, but was made more likely by his treatment regime which at that stage included amitriptyline, multiple antipsychotic agents such as chlorpromazine, and repetitive anaesthetics necessary for administration of ECT."*

Dr Giarraputo was also of the view that death was accidental. This evidence leads me to conclude that Mr Pasinski's death was accidental and was not a consequence of an intentional act on his part to take his own life.

THE EMERGENCY RESPONSE

It was the evidence of Dr Parkes that a patient with a completely blocked airway will lose consciousness within 2 minutes and will suffer a cardiac arrest within *"no more than 5 minutes."* After this, it is my understanding that death will occur within a further 5-7 minutes if oxygen is not provided and cardiac function resumed. These facts made it critical for Mr Pasinski's airway to be cleared of the chicken mass at the earliest opportunity if his life was to be saved.

I am satisfied that the Code Blue call was made in a timely manner and that Nurse Flynn and his colleagues at Northside acted appropriately when Mr Pasinski's airway became obstructed. I am satisfied too that MET made a timely response to the Code Blue. However, the members of MET are attached to ICU which is located in the body of the hospital and a considerable distance from Northside which is sited on a different level on its western perimeter. Because of this geography at least 5 minutes had elapsed by the time the MET arrived at Northside. By this time it seems clear that Mr Pasinski had experienced a cardiac arrest with hypoxia as evidenced by his clenched jaw. It is clear too that the MET, with the involvement of Dr Patil, acted promptly in removing the mass of chicken meat and clearing Mr Pasinski's airway. However, I am satisfied that by the time this was achieved any realistic opportunity of successfully resuscitating Mr Pasinski had passed.

Dr Parkes described the circumstances surrounding this choking event as a *"perfect storm"*, a description which I consider to be most apt given:

- The rarity of an event where a patient suffers a completely blocked airway. It was Dr Parkes' evidence that he had not encountered a similar event in 20 years practice as a consultant intensivist. Similarly Dr Giarraputo, in his 23 years of practice as a psychiatrist, had not encountered such a circumstance.
- Mr Pasinski's hypoxia causing his jaw to clench thereby preventing the Northside nursing staff from clearing his airway, either manually or with a suction unit and thereby mandating the urgent need for MET's attendance.
- MET's location within the hospital which prevented it from attending Mr Pasinski before at least 5 minutes had elapsed.
- The need to use Magill forceps to remove the chicken mass in a piecemeal fashion.

I am satisfied that this *"perfect storm"* created a situation where Mr Pasinski's life could not be saved, despite the prompt and appropriate endeavours of both the Northside staff and the members of MET.

During the course of the inquest some consideration was given upon whether I could make any recommendations which may assist to avoid another death occurring in similar circumstances. From the outset I accept that it would not be feasible, or in the interests of LGH patients generally, for the MET to be attached to a ward other than ICU and/or for its members to be based elsewhere in the hospital. Too, I accept that it is not feasible for a second MET to be established which was located in closer proximity to Northside. This leaves for consideration whether Northside staff could be realistically trained to provide an on-the-spot MET service to its patients who suffer a choking event which causes a complete obstruction of the airway. On this subject it was Dr Parkes' view that it may be possible to train clinicians in Northside in the use of Magill forceps to remove obstructions from a patient's airway. However, Mr Pasinski's case was complicated by his clenched jaw. This phenomenon required the administration of adrenaline or other muscle relaxants by injection to force the jaw's release. Dr Parkes opined that the administration of such injections require a level of special skill which could not realistically be delegated to either the medical or nursing staff within a psychiatric ward such as Northside.

The evidence of Dr Parkes, which I accept, leads me to **recommend** that the LGH give consideration to putting in place a programme to train members of the Northside unit in the use of Magill forceps. (I need to note that it was Dr Parkes' understanding that Northside had, subsequent to Mr Pasinski's death, been equipped with Magill forceps. However, Nurse Flynn *"had not seen them."* This is a situation that requires clarification.)

REPORT IN ACCORD WITH S28(5)

At the time of his death Mr Pasinski was being detained at Northside as an involuntary patient under a Continuing Care Order made pursuant to s28(1) of the *Mental Health Act* 1996. As such Mr Pasinski qualified as a person held in care as defined by s3 of the Act. In this circumstance I am required by s28(5) to report upon the care, supervision or treatment provided to Mr Pasinski. This requirement raises several issues for me to consider.

The first concerns the level of observation in place for Mr Pasinski. The evidence is that his management plan required him to be accommodated in HDU and for one-on-one observation to be maintained. This plan was, in my view appropriate, and was in place at the time of the fatal event. At this time Nurse Flynn was the person responsible for maintaining observation and the evidence shows, in the period leading up to the choking, that he was either inside the HDU with Mr Pasinski or

alternatively in the adjoining nursing station from where he maintained a view of his patient via its glass partition. When he realised that something was amiss he responded quickly by attending Mr Pasinski, attempting to help him regurgitate the chicken and by making a Code Blue call. Overall I make no criticism concerning Mr Pasinski's observation.

Another matter to consider is whether a soft diet should have been introduced for Mr Pasinski following the choking incident at breakfast? It was Dr Parkes' evidence that in hindsight a soft diet should have been adopted following the morning incident. On this subject, Dr Sale in his substantive report comments; *"It would have been preferable to restrict Mr Pasinski's oral intake to fluids until this situation was clarified."* This comment was made in the context of a recommendation made following a Significant Incident Review ('the Review') and which I will refer to in more detail in a moment. Specifically the recommendation stated that; *"RMO review post event. This entails physical/neurological assessment or any other assessments deemed necessary and relevant to the incident."*

In my view the choking incident at breakfast was a significant event which caused Mr Pasinski to fall unconscious and which generated a Code Blue and the involvement of MET. In these circumstances it is my view that it would have been prudent for Mr Pasinski to have been placed on a soft diet pending the outcome of the assessment which has since been determined as being warranted following the Review. This course would have reduced the risk of a subsequent choking event, a risk which was not necessarily inconsequential in the light of Dr Sale's assertion, based upon a review of the literature, that; *"Choking incidents including fatalities are not rare in individuals who suffer significant mental disorder or who are in psychiatric facilities."*

What of Mr Pasinski's medical management?

As I have already noted Dr Sale undertook a comprehensive review of Mr Pasinski's two admissions to Northside in 2013 and this included the consideration of his medical management. He acknowledges that his behaviour was *"difficult and challenging"* and that he represented, as I have already said, a *diagnostic dilemma."* Dr Sale does not assert that the diagnoses and treatment plans adopted by Mr Pasinski's treating psychiatrists were wrong or inappropriate and I accept this to be so. However, he does point out that the history shows that over an extended period Mr Pasinski did not appear to be responding positively to his treatments and that in fact his condition may have been deteriorating. He postulates that it may have been appropriate, at an earlier stage, for an overall review of his treatment including the continuation of ECT (he suggested this therapy should possibly have been limited to 5 procedures) and the maintenance of his antipsychotic medications.

In his evidence Dr Giarraputo explained that in his view ECT is often an effective therapy, more so than antidepressants. He explained too that in Mr Pasinski's case

two of the procedures were not considered to be valid because of *"problems"* related to the anaesthesia. Finally, and coincidentally he explained, as I have already noted, that at the Clinical Review meeting conducted on the morning of Mr Pasinski's death both Drs Giarraputo and Elijah agreed to revise the treatment plan by ceasing ECT and adjusting Mr Pasinski's medications including the tricyclic antidepressants.

I make no criticism of Mr Pasinski's diagnosis and treatment as overseen by Dr Giarraputo. Mr Pasinski was a particularly difficult patient and the decisions made by Dr Giarraputo and his colleagues were, in my view, properly considered and appropriate to the patient's presentation. They were of course aided and informed by Dr Giarraputo's and the Northside staff's daily contact with Mr Pasinski. I accept that there may be some merit in Dr Sale's observation that Mr Pasinski may have benefited from an earlier re-assessment of his management plan but this is an opinion made with the benefit of hindsight and does not warrant a criticism of the real-time decisions made upon Mr Pasinski's care and management whilst in Northside.

In summary, for the purposes of s28(5) of the Act I am satisfied that the level of observation in place for Mr Pasinski was appropriate and properly maintained. I am satisfied too that his diagnoses and treatment plans were reasonably suited to Mr Pasinski's presentation and do not warrant criticism. My one criticism relates to the failure, following the choking incident at breakfast, to initiate a comprehensive review of that event and for Mr Pasinski, in the meantime to be placed on a soft diet pending its completion. This is a shortcoming identified by the Review and which has led to the specific recommendation which I have referred to and which I endorse.

THE REVIEW

This was chaired by Associate Professor Len Lambeth, Chief Civil and Forensic Psychiatrist. In addition to that recommendation which I have already set out, the Review made these further recommendations:

1. *"Update and upgrade of resuscitation equipment on Northside.*
2. *Lab results to be signed as cited by medical team.*
3. *Development and implementation of an extensive suicide risk assessment where indicated.*
4. *Complex case review for all patients after 30 days admission.*
5. *Swipe access for code blue team members."*

I accept these recommendations to be appropriate and support them.

Allied to recommendation number 4, Dr Sale, during the course of his evidence, made the suggestion, which Dr Giarraputo supported, that a high risk and complex case panel be established to review long-term psychiatric patients, most particularly

those held on an involuntary basis and to be at high risk from a clinical viewpoint. It was suggested that the panel could be Statewide, that it comprise two senior clinicians not associated with the patient, and that at 28 day intervals it provide review and feedback on a patient's diagnosis and management to the treating consultant. I believe this suggestion to have merit and **recommend** its consideration by the Tasmanian Health Service.

FINDINGS IN ACCORD WITH s28(1)

I find:

1. The identity of the deceased is Alexander Pasinski;
2. Mr Pasinski died at the LGH (in Northside ward) in Launceston on 25 February 2015.
3. The circumstances of Mr Pasinski's death are set out in these findings.
4. The cause of Mr Pasinski's death was asphyxia due to choking on food.

CONCLUDING COMMENTS

I extend my sincere condolences to Mr Pasinski's family and loved ones.

Mr C N Dockray was counsel assisting. I acknowledge and thank him for his excellent work. I acknowledge too the attendance by members of Mr Pasinski's family at the inquest and their participation in the process. It is hoped that it may have been of some benefit to them in dealing with this sad event.

Dated: 16 August 2016 at Hobart in the State of Tasmania.

Rod Chandler
Coroner

APPENDIX A (CORONERS REPORTS (XIX)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

(These findings have been de-identified in relation to the name of the deceased, his family and friends, by direction of the Coroner pursuant to s. 57(1)(c) of the Coroners Act 1995.)

I, Simon Cooper, Coroner, having investigated the death Mr B

Find, pursuant to Section 28(1) of the *Coroners Act* 1995, that

(a) The identity of the deceased is Mr B;

(b) Mr B died in the circumstances described in this finding;

(c) The cause of Mr B's death was drowning following combined drug and alcohol intoxication;

(d) Mr B died between 19 January 2014 and 20 January 2014 at Hobart in Tasmania; and

(e) Mr B was born in Hobart, Tasmania and was aged 35 years at the time of his death; he was a single, unemployed man.

Background

Mr B suffered from mental health problems for a considerable period of his adult life. He had extensive contact with Dr David Weidmann, a consultant psychiatrist in practice in Hobart. Mr B's most significant issues related to substance abuse and secondary depression sometimes accompanied by secondary psychosis. In addition, he was diagnosed as suffering from an obsessive compulsive disorder and a panic disorder. He was also treated by a general practitioner for alcoholism.

Mr B had numerous admissions for treatment, as an inpatient, to the psychiatric ward of St Helen's Private Hospital in Hobart. His last admission to that hospital was in December 2013. He was discharged in January 2014, just 10 days before his death.

Circumstances Surrounding the Death

It is very clear that in the days leading up to his death, Mr B was significantly ill. It is also quite clear that numerous attempts were made by both friends and his family to have him admitted to hospital for treatment but those attempts were all unsuccessful.

On 15 January 2014, Mr B was visited by his father. His father said in an affidavit made pursuant to the *Coroners Act* 1995 that Mr B 'looked ok' but also that Mr B told him that 'he wasn't right and ...that he thought he would have to go back to hospital'. Later the same day Mr B called a friend late in the evening and had a conversation with that friend about a female who he (Mr B) thought was going to kill him.

The next day, Thursday 16 January 2014, Mr B's father visited him again. Mr B again told his father that he considered he needed to go to hospital. Mr B's father later told investigating police that he made phone calls to both St Helen's Hospital and Dr Weidmann in an attempt to have his son admitted. Mr B's father said he was informed that St Helen's did not have an available bed and that Mr B's admission needed to be authorised by Dr Weidmann. Mr B's father said "at [that] stage [he] was concerned about his son's health but felt there was nothing [he] could do".

Mr B contacted his brother that same evening in a distressed state. Mr B's brother visited him at about 8.30pm and noted he was upset but settled after his arrival. Mr B told his brother he had not been taking his tablets (contextually this can only mean medication prescribed for him).

The next day, Friday, 17 January 2014, Mr B's father visited his son again and made more phone calls in an attempt to have his son admitted to St Helen's Private Hospital for psychiatric treatment. Again he was unsuccessful. He repeated these attempts, again without success, on Saturday 18 January and Sunday 19 January.

On Saturday 18 January, Mr B's father again saw his son. His father told investigating police that Mr B told him that he had attended the Royal Hobart Hospital (RHH) the previous evening complaining of 'trouble breathing [and] tightness in his chest'. Mr B told his father that he had been told to come back on Monday 20 January for an ECG check. However an examination of Mr B's medical records does not support that he was given this advice. The records show that Mr B visited the RHH Department of Emergency Medicine (DEM) in the early hours of 17 January. The DEM notes record that Mr B presented complaining that he 'felt he had a breathing problem'. The nursing assessment records that he told staff he had been binge drinking for a week and that he was 'going into St Helen's' that day. The same information is recorded in the notes of the doctor who saw him.

On the last day of his life, in January 2014, Mr B was visited at his home, by a friend, and later, again, by his parents. Both the friend and Mr B's father said Mr B was clearly distressed. Mr B's father again contacted St Helen's hospital. The hospital confirmed Mr B would be admitted the next day. Mr B's father described his son as not being 'his usual bubbly self' but he also said he didn't think he was drunk and that he didn't think Mr B was drinking whilst his parents were with him.

Mr B's parents left their son's home at about 7.00pm.

Mr B's friend however described Mr B as someone who appeared to have been drinking for some time and noted the smell of alcohol in the flat. He also described Mr B as appearing to be affected by the presence of his parents, describing him as "getting upset and teary". He left Mr B, alone, at about 9.30pm. Mr B was not seen alive again.

At roughly 10.00am on the date of Mr B's death police were tasked to attend Mr B's unit. Officers were met by Ms Carlee Howard who was employed by Key Property, the letting agent for the unit. She in turn had been contacted by the owner of the building, complaining about a large amount of water flowing from Mr B's unit and into the business situated therein. Ms Howard made her way to Mr B's residence where she saw water flowing from under the door of the unit, under the door of the business, over the footpath and into the gutter. She could tell immediately that it was coming from Mr B's unit. Ms Howard banged on the door of the unit. After a short time with no answer she used her key to open the door and called out for Mr B (whom she knew). There was no answer. She could see water running from the bathroom at the top of the stairs. Ms Howard went to the bathroom and found Mr B lying dead in the bath. She immediately telephoned police.

Mr B's body was formally identified at the scene and removed by mortuary ambulance and transported to the mortuary at the Royal Hobart Hospital.

An investigation into Mr B's death was commenced at the scene. His unit was noted to be untidy but there were no signs of disturbance, violence or forced entry (I note that Ms Howard needed to use a key to enter the unit). Officers from Hobart CIB and police forensic services attended the scene. No circumstances of suspicion were identified. A number of items were taken possession of by police including Mr B's computer and a quantity of medication. Several hand written notes capable of being interpreted as 'suicide' notes were also located at the scene. A subsequent forensic analysis of his computer revealed nothing relevant to his death.

An autopsy on Mr B's body was carried out by Dr Christopher Hamilton Lawrence, the State Forensic Pathologist. Dr Lawrence's opinion, which I accept, was that the cause of Mr B's death was drowning following combined drug and alcohol intoxication. The presence of drugs namely Quetiapine (Seroquel), alcohol and diazepam (valium) were detected as a result of a toxicological analysis of samples taken at autopsy. Quetiapine, an atypical antipsychotic agent used in the treatment of schizophrenia, bipolar disorder and as an adjunct in treatment resistant major depression and in generalised anxiety disorder, was found to be present at a level within the reported toxic lower fatal range. Alcohol was detected as being present at a level of 0.237g in 100 mL of blood. Diazepam was also found to be present in therapeutic levels but it is noted that in combination with other central nervous system depressants, such as Quetiapine and alcohol, the sedative effects of diazepam are enhanced.

I am satisfied that Mr B's death occurred in the circumstances set out in this finding. I am satisfied on the balance of probabilities that Mr B took his own life and that no other person was involved in his death.

It is very clear that Mr B was very ill and in need of proper medical treatment in the days leading up to his death. Numerous attempts were made to obtain that treatment and none were successful. Had Mr B been treated and, in particular, had he been admitted as an inpatient for psychiatric care, then I am satisfied he would not have died in the circumstances in which he did.

Mr B and his family sought treatment for him but were constantly turned away. It is difficult to imagine any person in our community gravely ill with any condition, other than mental illness, being refused treatment when it was so obviously needed. The treatment received in the lead up to Mr B's death has been comprehensively reviewed as part of this investigation.

Suicide in persons suffering from mental illness is very common in Australia. Mr B's mental illness meant he was obviously a serious suicide risk. Dr Weidmann, in a report to the coroner relating to the investigation of Mr B's death, acknowledged that Mr B was at 'chronic risk of suicide'. He also said 'unfortunately the St Helen's Private Hospital is sometimes full and has a waiting list. If this ever happens, individuals are advised to contact the Royal Hobart Hospital. His father rang St Helen's and was so advised'. This statement, in so far as it suggests that an absence of beds was the reason for Mr B not being admitted to the St Helen's Private Hospital, is misleading.

It is plain, and I find, that the claimed failure on the part of the St Helen's Hospital to admit Mr B on the basis of an absence of sufficient beds is inaccurate. Data provided by St Helen's Hospital relating to the relevant ward (Hampden) simply does not support the assertion that there was an absence of suitable beds or that it was 'full'.

The Hampden Unit was, in January 2014, a ward with a capacity for 30 patients. Mr B's father sought Mr B's admission over several days. Those days were 16 January to 19 January 2014, inclusive. It is quite clear from the information obtained as a consequence of this investigation that on 16 January 2014 the ward had 25 patients, on 17 January 2014, 26 patients, on 18 January 2014, 22 patients and on 19 January 2014, 20 patients. The material supplied by the hospital satisfies me that at all relevant times staff levels were appropriate to the number of inpatients in the unit. In short, the Hampden Unit was never full. It follows that the assertion that there were insufficient beds is not made out.

Both St Helen's Private Hospital and Dr Weidmann were afforded the opportunity to comment in relation to the findings set out above. The hospital accepted that its records indicated it had beds available during the relevant period. However it explained that patient admissions were only accepted if authorised by a patient's "privately treating Doctor". The hospital went on to say that Dr Weidmann authorised Mr B's admission for 20 January 2014. The hospital said that this was the earliest date that it was able to admit Mr B.

The hospital also pointed out that it does not have an Emergency Department and all patients need to be under the care of their admitting doctor.

Dr Weidmann replied personally as well as through solicitors instructed by him. The substance of both replies was essentially the same. I note also that he had provided an earlier report in response to a request from the coroners' office on 25 March 2014 (which has already been referred to). In his reply of 6 September 2016, Dr Weidmann repeated his assertion that "the hospital is sometimes full". He indicated he stood by that "assertion". He offered as an explanation the fact that the period in question was "summer school holidays and during this time the hospital has reduced professional staff". Like the hospital, Dr Weidmann highlighted the fact that St Helen's does not provide an emergency service.

In summary, both the treating psychiatrist and the hospital pointed to an absence of staff, and not beds, as the reason Mr B was not admitted. It is unfortunate that this was not made clearer when a report was first sought early in the investigation into Mr B's death.

Comments and Recommendations:

The circumstances of Mr Mr B's death are not such as to require me to make any recommendations pursuant to section 28 of the *Coroners Act* 1995.

I do, however, comment that this is yet another tragic suicide of a young person in Tasmania suffering from mental illness and unable to access adequate treatment. It is clear that Mr B and his family were crying out for assistance in the immediate lead up to his death but none was provided. A civilised society has a duty to ensure proper treatment is provided to anyone suffering a medical condition whether physical or psychiatric. As I have already observed, it is difficult to imagine a person suffering from a physical ailment as serious as the psychiatric illness Mr B was suffering from in the lead up to his death being unable to access appropriate treatment.

I convey my sincere condolences to the family and loved ones of Mr B.

Dated: 3 October 2016 at Hobart in the state of Tasmania.

Simon Cooper
CORONER

APPENDIX A (CORONER'S REPORTS (XX)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Margaret Wynne Newett

Find that:

a) The identity of the deceased is Margaret Wynne Newett;

b) Mrs Newett was born in Queenstown on 27 January 1936 and was aged 77 years;

c) Mrs Newett died at the North West Regional Hospital (NWRH) in Burnie on 5 January 2014; and

d) The cause of Mrs Newett's death was the combined effects of ischaemic heart disease and calcific aortic valve sclerosis.

Background:

Mrs Newett resided at 14 Fysh Street in Queenstown. She was a widow, her husband Milton having died in January 2013. They had four children. Mrs Newett's medical history included a splenectomy in 1995 and hypertension.

Circumstances Surrounding the Death:

In mid-2011 Mrs Newett attended her doctor in Queenstown for a medical assessment required for the renewal of her driving licence. On examination, a systolic cardiac murmur was detected and Mrs Newett was referred to cardiologist, Dr Jozef Durech in Burnie. Dr Durech saw Mrs Newett on 29 June 2011. In his view Mrs Newett was suffering from moderate aortic stenosis which was asymptomatic. He recommended medical therapy and further review if she became symptomatic or if her doctor thought it warranted.

In mid-2013 Mrs Newett was again medically assessed for the purposes of her driving licence. On this occasion her doctor noted that she had two years previously been diagnosed with aortic stenosis. The notes went on to state; "*Does not have any dizzy spells/blackouts. No consult in past two years with any symptoms relating to dizziness/blackouts. BP 140/80.*"

It seems that Mrs Newett did not report or demonstrate any symptoms or signs of aortic stenosis until 30 December 2013. On this day she collapsed in a Queenstown supermarket. She was attended by officers from Ambulance Tasmania. She had been apparently unconscious for several minutes. There was no associated chest pain or breathlessness.

She had become incontinent during the collapse. She was taken by ambulance to the West Coast District Hospital (WCDH). On examination an aortic stenosis murmur was noted. Vital signs and neurological examination were normal. An ECG was taken which was abnormal. Advice was then sought by telephone from the cardiology unit at the Launceston General Hospital (LGH). Contact was made with cardiology registrar, Dr Ramanathan Parameswaram. He asked to see the ECG and a digital copy was provided. His interpretation of the ECG was: "*Sinus rhythm, Heart rate 81/min Left bundle branch block with secondary ST-T changes.*" The notes at the WCDH also include this entry; "*Discussed with Dr Ran(?) at LGH. He advised against getting Troponins done, advised to be referred for echo on outpatient basis…*"

Mrs Newett remained in the WCDH for 24 hours for observation. She remained stable and pain free. Her clinical observations were within normal limits. When discharged the plan was for her to see her general practitioner and to arrange a referral for a carotid Doppler scan and an echocardiogram.

On 2 January 2014 Mrs Newett attended General Practitioner Dr Vincent Jain. Her recent syncope (faint) was noted along with her moderate aortic stenosis. He suggested; "*that Mrs Newett should not drive and that she be again reviewed by Dr Durech.*" A letter of referral was prepared.

On 3 January Mrs Newett's son Scott telephoned Dr Durech's rooms to enquire whether he had received a referral from Dr Jain and whether an appointment had been made for his mother. Mr Newett in fact spoke to Dr Durech in person who advised him that he would be on leave until 1 April 2014 and thus would not be able to see Mrs Newett prior to this date. Subsequent to this, members of Mrs Newett's family made contact with the Cardiac Centre at the Calvary Hospital in Hobart and arranged an appointment for Mrs Newett to see Cardiologist, Dr Andrew Black on 8 January 2014.

On 4 January 2014 Mrs Newett was travelling in a motor vehicle with family members when she became unconscious. An ambulance was called and she was conveyed to the NWRH. At the hospital Mrs Newett's ECG was unchanged. The blood troponin level was elevated indicating myocardial damage. Mrs Newett was treated for acute myocardial infarction with aspirin, clopidogrel and therapeutic heparin. The noted plan was for the hospital to liaise with the cardio-thoracic unit at the Royal Hobart Hospital the following morning.

At 3:50am on 5 January Mrs Newett was transferred to the NWRHs High Dependency Unit following a further syncope episode. At this time the ECG record showed left bundle branch block with severe ischaemic changes. She was severely hypotensive. Thereafter, Mrs Newett developed rapid atrial fibrillation and suffered a cardiac arrest. Resuscitation efforts were maintained for about 25 minutes but Mrs Newett could not be revived. Her death was recorded at 4:54am on 5 January 2014.

Post Mortem Examination:

This was carried out by State Forensic Pathologist, Dr Christopher Lawrence. Dr Lawrence has recorded that the "*autopsy reveals calcific aortic valve sclerosis with moderate aortic stenosis. There is also severe ischaemic heart disease with 80% narrowing of the left*

anterior descending coronary artery with haemorrhage into a plaque." In his opinion Mrs Newett died from the combined effects of ischaemic heart disease and calcific aortic valve sclerosis.

I accept Dr Lawrence's opinion upon the cause of death.

Investigation:

This has included the following:

1. A review of Mrs Newett's records at the Queenstown General Practice, the WCDH and the NWRH undertaken by research nurse, Ms Libby Newman.
2. The obtaining of affidavits from Christopher Newett and Scott Newett, both sons of the deceased.
3. Consideration of a report provided by Dr Durech.
4. Consideration of a report provided by Dr Parameswaram.
5. Compilation of a report upon Mrs Newett's medical and hospital care made by Dr A J Bell as medical adviser to the Coroner.|
6. A meeting attended by myself, Dr Bell, Ms Newman, Dr Lawrence and Forensic Pathologist, Dr Donald Ritchey to review the investigation.

In his report Dr Bell provides this advice and opinion:

- The most common presenting symptoms for aortic stenosis are decreased exercise tolerance, exertional dizziness and exertional angina. However, some patients can be asymptomatic for a prolonged period. Heart failure, syncope and angina are all 'classic' symptoms reflecting the end-stage of the disease.
- Serial transthoracic echocardiogram (TTE) play an integral role in the longitudinal management of patients with aortic stenosis and the frequency of routine follow up studies is determined by the severity of the disease. When Mrs Newett saw Dr Durech in June 2011 she was diagnosed with moderate asymptomatic aortic stenosis. The 2006 American College of Cardiology/American Heart Association guidelines recommend that a patient with this diagnosis receive a follow-up TTE every 1-2 years.
- It was unrealistic to expect the medical staff at the WCDH to be able to fully interpret the ECG taken on 30 December 2013 and it was good practice for advice to be sought from the LGH's cardiology unit.
- Dr Parameswaram's interpretation of the ECG was not complete. Dr Bell opines; *"In the leads V5 and V6 in LBBB the T wave should be in the opposite direction from the QRS complex. The T waves were upright. This is called pseudonormalization of the T wave. This suggests there is a secondary problem, and this is not just LBBB. The most likely issue is ischaemia and/or infarction of the heart tissue. The sign is not diagnostic but suggestive."*

- Mrs Newett required a blood test on 30 December to determine her troponin level. It is most likely that the test would have shown an increased troponin level indicative of some heart damage. The test could have been done at the WCDH and involves a five minute process.

- Mrs Newett's syncope, her previous diagnosis of aortic stenosis, the ECG and a likely elevated troponin level required her immediate transfer to the Royal Hobart Hospital (RHH) on 30 December 2013 for treatment in its cardiac unit.

- Had Mrs Newett been transferred to the RHH it is likely that she would have been assessed for possible aortic valve replacement and coronary artery angioplasty or grafting. The prospects of survival are excellent during the prolonged asymptomatic phase but decline rapidly after the development of symptoms. Aortic valve replacement prevents this rapid downhill course.

Findings, Comments and Recommendations:

The evidence clearly shows that Mrs Newett was diagnosed with moderate aortic stenosis in mid-2011. It is my understanding that this condition is progressive but the rate of progression is variable. It is for this reason that it requires monitoring, most particularly with the aid of TTE, which enables the identification of a deteriorating patient who will benefit from surgery before end-stage symptoms appear.

It is evident that the seriousness of Mrs Newett's condition was not fully recognised post its diagnosis and in the result opportunities were lost to possibly avoid her regrettable death.

Firstly, it seems clear that no steps were taken for Mrs Newett to have a follow-up TTE within 2 years of her diagnosis contrary to the guidelines identified by Dr Bell. Given the findings upon autopsy it is likely that a TTE would have revealed a worsening of Mrs Newett's aortic stenosis and been a prompt for further investigation and possible surgery.

Secondly, I accept that Mrs Newett's syncope on 30 December 2013 was, as advised by Dr Bell, a sign of end-stage aortic stenosis with or without coronary artery disease. Its occurrence, in the context of Mrs Newett's earlier diagnosis, required Mrs Newett's immediate referral to a hospital properly resourced to evaluate her condition and to implement appropriate treatment. The cardiology unit at the RHH was the most suitable venue. I am satisfied that this did not occur largely because of the telephone advice provided by Dr Parameswaram. That advice was less than optimal for several reasons. Firstly, I accept Dr Bell's opinion that Dr Parameswaram should have advised that Mrs Newett's troponin level be tested. It's likely that the test would have been positive reinforcing the need for an urgent response to Mrs Newett's situation. Secondly, I accept Dr Bell's criticism of Dr Parameswaram's interpretation of the ECG. Again, a more fullsome interpretation should have helped to make clear the seriousness of Mrs Newett's situation. Irrespective of these matters it is my view that Dr Parameswaram, as a cardiology registrar, should have known that a syncope occurring in the presence of diagnosed aortic stenosis indicated a serious situation which mandated a referral to a hospital such as the RHH.

The third lost opportunity was on 2 January 2014 when Mrs Newett attended Dr Jain. His decision made on that day to refer Mrs Newett back to Dr Durech on a date to be determined was not an appropriate course to take in the light of the seriousness of Mrs Newett's

condition. However, I do recognise that Dr Jain was, in all probability, influenced in this decision by the advice received from Dr Parameswaram 3 days previously.

In the result, I have come to the view that Mrs Newett's death may have been avoided if those persons involved in her medical care had recognised the seriousness of her condition and responded in a more urgent and pro-active manner. Her tragic death should serve as a reminder that aortic stenosis is a life threatening condition which requires careful monitoring and an urgent response to clear signs of its progression.

The communication between the WCDH and Dr Parameswaram leads me to **recommend** that the LGH give consideration to adopting protocols around its telephone advice to outlying health facilities. Those protocols should include:

- A requirement to keep a written record of the communication.
- A requirement for a consultant to be informed of the advice at the first opportunity in those instances where the advice has not been provided by a consultant.
- A requirement that ECGs be reported upon by a consultant.

It is my understanding that the more remote parts of Tasmania such as the West Coast are serviced by short-stay medical practitioners. In the result patients often do not see the same doctor twice. This makes very difficult the management of chronic progressive diseases such as aortic stenosis. This in turn leads me to **recommend** that the State's health authority gives consideration to adopting a practice whereby all patients diagnosed with moderate to severe aortic stenosis are referred to the cardiology unit at the RHH for an annual review of their condition.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how her death occurred and the particulars needed to register her death under the *Births, Deaths and Marriages Registration Act* 1999. I do not consider that the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me. The circumstances of the death do not require me to make any further comment or to make any further recommendations.

I extend my sincere condolences to Mrs Newett's family and loved ones.

Dated: 21 April 2016 at Hobart in the State of Tasmania.

Rod Chandler
CORONER

APPEDIX A (CORONER'S REPORTS (XXI)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

(These findings have been de-identified in relation to the name of the deceased, family, friends and others by direction of the Coroner pursuant to s. 57(1)(c) of the Coroners Act 1995.)

I, Simon Cooper, Coroner, having investigated the death of Mr S

Find, pursuant to Section 28(1) of the Coroners Act 1995, that:

(a) The identity of the deceased is Mr S;

(b) Mr S died in the circumstances outlined further in these findings;

(c) Mr S died as a result of multiple injuries due to a motor vehicle collision with a tree;

(d) Mr S died on 2 January 2014 at Powranna Road, Powranna in Tasmania; and

(e) Mr S was born in Dunedin, New Zealand on 7 May 1970, and was aged 43 years at the time of his death; he was separated from his first wife and was employed as a truck driver when he died.

Introduction

Mr S died in the circumstances set out below in this finding. A preliminary issue needs to be considered. The issue is whether in terms of the *Coroners Act* 1995 an inquest is mandatory. If Mr S died in circumstances in which he was, for the purposes of the Act, "in custody" then an inquest would be mandatory (see section 24(1)(b) of the Act).

For reasons which will emerge further in this finding I am satisfied that at the time of his death Mr S was not in custody in terms of the Act. As a consequence I am satisfied that an inquest is not mandatory.

Circumstances Surrounding the Death

Mr S commenced a relationship on the internet with Ms P in December 2009. The relationship became permanent in December 2010. Near Christmas 2012 Ms P told police Mr S assaulted her and a police family violence order was made against Mr S. Ms P and Mr

S continued to live together but their relationship was punctuated by further incidents of domestic violence.

After an argument in June 2013 Ms P says Mr S threatened to commit suicide by driving a car into a tree.

On 21 August 2013 Mr S badly assaulted Ms P and the relationship came to an end.

There was a short reconcilation in September, but the relationship ended, again, in Novemeber 2013 when Ms P left Mr S and reported aspects of his behaviour to police. Those complaints led to Mr S being arrested and, *inter alia*, an interim family violence order being made. Mr S was bailed. A condition of his bail was that he was required to live at the residence of a friend, Mr R.

At the time of his death a full "non-contact" interim family violence order was in place against Mr S. In addition to charges of family violence, he was also facing charges of breaching an interim family violence order and various breaches of bail.

Mr R's daughter lived at the residence of Mr R from time to time (she divided her time between her mother and father who were separated).

On 12 December 2013 Mr. R's daughter disclosed to her father conduct on the part of Mr S which, if proved, would have amounted to an indecent assault upon her. Mr R asked Mr S to leave his address. Mr R reported the matter to police and the daughter made a statement.

On 23 December 2013 Detective Senior Constable Barrett of the Launceston CIB contacted Mr S. He had a conversation with him by telephone. In that conversation Detective Senior Constable Barrett outlined the allegations that Mr R's daughter had made against him, and made arrangements for Mr S to attend the Launceston Police Station on 2 January 2014 for an interview.

Between Christmas and New Year Mr S was in contact with Ms P. That contact involved a trip to Deloraine and to Tasmania Zoo. On 1 January 2014 Ms P and Mr S went to the cinema together. Ms P reports on that occasion Mr S was "crying and upset saying that he was fearful of going to the police and was afraid of going to jail and wasn't a paedophile".

Ms P spoke with Mr S by telephone between noon and 1.00pm on 2 January 2014. A little later Mr S rang and spoke with Detective Senior Constable Barrett and another detective. During this phone call, Mr S expressed his intention to commit suicide by driving his car into a tree but refused to divulge his location. Detective Senior Constable Barrett, a trained negotiator, attempted to negotiate with Mr S. He recorded the conversation he had with Mr S on his personal iPhone. Contact was made with police radio dispatch services (RDS) and efforts were made immediately to locate Mr S. That was done by mobile phone triangulation. Two police units were dispatched to the general area of Mr S's location, which was determined to be in the junction of the Midland Highway and Powranna Road. The units tasked were specifically instructed not to approach Mr S's car but to await instructions. The evidence from the officers involved, as well as objective evidence obtained during the investigation in relation to the circumstances surrounding Mr S's death from, *inter alia*, the police AVL system, demonstrates that they followed these instructions. No unit approached to within a kilometre of Mr S's vehicle.

It is clear from the conversations had between Mr S and Detective Senior Constable Barrett that at no stage did Detective Senior Constable Barrett ever indicate to Mr S that it was his intention to arrest him or take him into custody. In fact Detective Senior Constable Barrett indicated quite the opposite.

While Mr S seems to have been conscious that police were in his general vicinity it is quite clear to me, and I find, that that awareness had no impact whatsoever upon his decision to take his own life.

Throughout his conversations with Detective Senior Constable Barrett, Mr S resolutely indicated an express intention to commit suicide by driving his vehicle into a tree at high speed. He indicated that he had already had a "practice run". Unfortunately, Mr S terminated the call despite admirable efforts by Senior Constable Barrett to dissuade him. Mr S then drove at very high speed in a westerly direction on Powranna Road straight into a large gum tree. One of the attending officers describes the car as erupting into flames after the collision. The vehicle was unrecognisable and debris was scattered over a wide area. Mr S was killed instantly. So severe were the flames at the scene, attending police could do nothing to assist him.

Uniform, forensic and CIB officers all attended. So did personnel from Tasmania Fire Service and the on-call coroner. An investigation was commenced at the scene.

Mr S's body was subsequently removed from the wreck of the vehicle and transported to the mortuary at the Royal Hobart Hospital. After formal identification utilising DNA, an autopsy was undertaken. The autopsy was carried out by Dr Christopher Hamilton Lawrence, the State Forensic Pathologist. Dr Lawrence expressed the view after autopsy that Mr S died of multiple injuries due to a motor vehicle collision. The autopsy revealed that he had suffered traumatic injuries to his aorta, chest, pelvis and possibly to the skull. The absence of clear soot in the upper airway and carbon monoxide in his blood indicated that he had been killed instantly and did not die as a result of smoke inhalation. I accept Dr Lawrence's opinion.

Samples taken at the autopsy were subsequently analysed at Forensic Science Service Tasmania and nothing of any significance emerged from that analysis.

I am satisfied that Mr S died in the circumstances described in this finding. Specifically, I am satisfied that on no view of his interaction with Tasmania Police on 2 January 2014,was Mr S in custody in terms of the *Coroners Act* 1995. He was not under arrest. No attempts were made to arrest him or intercept him; indeed police at all times kept well clear of him. At its highest, his interaction with police involved Detective Senior Constable Barrett negotiating with him with a view to attempting to persuade Mr S not to commit suicide. For these reasons I am satisfied that Mr S was not "in custody" at the time of his death.

I am satisfied that the acts which caused his death were voluntarily undertaken by him with the express intention of ending his own life.

Comments and Recommendations:

Nothing about the circumstances surrounding the death of Mr S requires me to make any recommendations or comments pursuant to section 28 of the *Coroners Act* 1995.

Dated: 30 October 2015 at Hobart in the State of Tasmania.

Simon Cooper
Coroner

APPEDIX A CCORONER'S REPORTS

XXII

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, **Rod Chandler**, Coroner, having investigated the death of Paul Lucien Henri

Find That:

(a) The identity of the deceased is Paul Lucien Henri;

(b) Mr Henri was born in Hobart on 5 August 1935 and was aged 78 years;

(c) Mr Henri died at the Royal Hobart Hospital ('RHH') in Hobart on 15 October 2013; and

(d) The cause of Mr Henri's death was cerebral infarction and hypoxic brain damage due to the combined effects of ischaemic heart disease, aspiration pneumonia, Parkinson's disease and obstructive sleep apnoea.

Background:

Mr Henri was married to Shirley Marie Henri. They had two children. He was an auditor and had been employed for 38 years by the Hydro Electric Commission, retiring at age 55 years. His medical history included chronic obstructive pulmonary disease, myocardial infarction, depression, Parkinson's disease, hypertension, back, neck and shoulder pain and obstructive sleep apnoea. For some years Mrs Henri had helped care for her husband at home.

Circumstances Surrounding the Death:

On 9 September 2013 Mr Henri was admitted to the Huon Eldercare Nursing Home ('the Home') whilst his wife had surgery. At this time Mr Henri's principal health issues related to his lack of mobility arising from his Parkinson's disease and neck and shoulder pain. He was taking Tramadol for analgesia as well as regular Panadol Osteo. His care was being overseen by Dr John Riley of the Huon Valley Health Centre. On 23 September Dr Riley increased Mr Henri's Tramadol dose because of his persistent pain.

On 24 September 2013 Mr Henri was reviewed by Professor Peppard, a consultant neurologist who specialises in Parkinson's disease. He considered Mr Henri to be suffering from anxiety related to his changed residential situation and the apparent inability of his wife to take him home. Professor Peppard prescribed quetiapine. A subsequent review of Mr Henri's drug chart shows that this drug was given in the correct dosage from the evening of 24 September until his discharge. Professor Peppard did not change Mr Henri's Madopar dosage. The nursing notes record that Dr Riley advised that the quetiapine was to be ceased immediately for over-sedation or other observed side effects. However, it seems that Mr Henri remained active over the next few days with no evidence of a sedation difficulty.

On 1 October 2013 Dr Riley decided to cease the Tramadol and trial Mr Henri on MS Contin (10mg, 3 times a day). MS Contin is a time released formulation of morphine sulphate usually taken to manage chronic pain. Dr Riley recorded in the notes that whilst Mr Henri was staying at the Home this was a "*great place for a trial. He has CPAP for his OSA (obstructive sleep apnoea) so a bit of night time sedation would not be worrisome.*" (Dr Riley has since advised that it was not his belief that the use of a CPAP machine would mitigate against the risk of night-time over sedation. Rather it was his view that the MS Contin may cause a general drowsiness which may worsen the sleep apnoea for which Mr Henri was equipped with a CPAP device.)

From 1 October to 5 October there are no medical notes within the Home records and only minimal nursing notes. The observation chart provides minimal information only. It indicates that Mr Henri's blood pressure remained low, as did his oxygen saturation on room air.

On 5 October Mr Henri was noted to be sedated. His oxygen saturations were 89% on room air and his blood pressure was 90/52mmHg. When given supplemental oxygen via nasal prongs Mr Henri became more easily roused. He received his morning dose of MS Contin. Later in the day the nursing notes indicate that Dr Riley was notified that Mr Henri's blood pressure was low and his oxygen saturations had dropped to 76% (on oxygen). His blood pressure was 78/56mmHg. Dr Riley directed that Mr Henri's MS Contin be ceased and that he commence oral antibiotics and his CPAP. The nursing notes indicate that difficulties were experienced by the nursing staff when trying to fit the CPAP mask, although it appears that this was eventually achieved. Early on 6 October Dr Riley was again contacted by nursing staff that were concerned by Mr Henri's condition. At this time his blood pressure was recorded at 90/52mmHg and the oxygen saturation was 76% on CPAP. Chest auscultation revealed moist crackles. A decision was taken to call an ambulance.

In the Emergency Department of the RHH Mr Henri was noted to be very unwell. He had an unrecordable blood pressure, his heart rate was 130bpm and he was unresponsive. Blood tests indicated that he was in acute renal failure. His limbs were noted to be extremely tremulous. A metaraminol infusion was commenced

along with intra-venous antibiotics. Mr Henri was then noted to be much more alert with an increased blood pressure. At this time a possible diagnosis was made of opiate related sedation. He was admitted to the Intensive Care Unit.

In the following days there was some improvement in Mr Henri's condition. By 10 October he was considered sufficiently recovered for transfer to a Ward. However, the following day he was noted to be more drowsy and confused. He was febrile and blood tests showed a sodium level of 154 and a mild respiratory alkalosis. A chest x-ray showed right sided basal atelectasis with a small effusion. A family meeting was held and the goals of care for Mr Henri were discussed. It was decided that if he deteriorated further then palliative care would be considered.

Mr Henri continued to slowly deteriorate over the following days. On 13 October, after consultation with family members, a decision was taken to initiate palliative care. This was maintained until Mr Henri's death at 5.05 am on 15 October 2013.

Investigation:

A post-mortem examination was carried out by State Forensic Pathologist, Dr Christopher Lawrence. In Dr Lawrence's opinion the cause of Mr Henri's death was cerebral infarction and hypoxic brain damage due to the combined effects of ischaemic heart disease, aspiration pneumonia, Parkinson's disease and obstructive sleep apnoea. I accept this opinion.

In his report Dr Lawrence makes this comment: *"Clinically hypoxic injury secondary to opioid use for chronic pain on the background of obstructive sleep apnoea was thought to be a possible contributing factor to the death but this cannot be explained by toxicology."* Dr Lawrence goes on to explain, *"Unfortunately it does not appear that any toxicology has been done during the admission (to the RHH) and the bloods from admission appear to have already been discarded, so it is not possible to establish at this stage what the blood levels of the opiates were."*

The investigation has also included the following:

(a) Consideration of an affidavit provided by Mrs Henri.

(b) Consideration of a statement obtained from Ms Sally Hodder, Mr Henri's daughter.

(c) A review of Mr Henri's records at the RHH and at the Home undertaken by Clinical Nurse Specialist, Ms Libby Newman.

(d) Consideration of a report provided by Dr Riley.

(e) Consideration of correspondence received from Dr Riley's solicitors.

(f) Consideration of a report provided by the Home.

(g) Consideration of reports provided by Dr A J Bell, as medical adviser to the Coroner.

(h) A meeting attended by myself as Coroner, Dr Bell, Forensic Pathologists, Dr Lawrence and Dr Donald Ritchey, and Ms Newman where a full review of the circumstances surrounding Mr Henri's death and its investigation was considered.

The focus of this coronial investigation was upon the medical care received by Mr Henri whilst resident at the Home and its supervision by Dr Riley, most particularly with respect to the safe management of his medication. Upon these matters Dr Bell has expressed these opinions:

- Older adults generally have an increased pharmacodynamic sensitivity to morphine and initial doses are generally decreased from those recommended for younger patients. A reasonable approach is to decrease the 'usual' dose by 50% given at the same intervals and slowly titrate up until effective.

- The usual dose of morphine, in any form, is 30mg per day in divided doses. In this case the initial dose provided to Mr Henri was 30mg per day. A more prudent approach would have been to initiate the dose at 15mg per day.

- It seems that Dr Riley considered the dosage of morphine prescribed for Mr Henri to be appropriate on the basis that he was not opiate naïve, presumably because he considered the drug Tramadol to be an opiate. However, clinical studies suggest that clinically significant cross tolerance between Tramadol and morphine does not occur.

- The safe approach to starting morphine on a patient chronically taking Tramadol is to start at the recommended dose for the patient and consider that patient opiate naïve.

- The addition of morphine to quetiapine may have exacerbated the effects of the morphine.

- The normal ventilator response of the central nervous system to hypercapnia and hypoxemia is diminished with age and may lead to an exaggerated respiratory depressant of opioids. Patients may also be at risk of clinically significant respiratory depression when opioids are titrated rapidly, are administered in the setting of sleep apnoea syndrome or some other serious cardiopulmonary comorbidity that limits ventilator reserve or when the opioid is combined with a sedative/hypnotic. The cautious selection of the initial dose and conservative incremental dose titration limits the risk of respiratory depression.

- The records from the Home indicate a practice of carrying out clinical observations on a daily basis, usually around 10am. They further indicate that

no changes were made to this practice even after quetiapine and later morphine were added to Mr Henri's medication regime.

- That CPAP is not a form of mechanical ventilation and is not useful for the treatment of respiratory depression due to morphine. Rather, the role of CPAP is to provide positive pressure inside the airway to keep it open when there is airway obstruction during sleep. CPAP does not protect against sedation induced hypoventilation and hypoxia.
- The level of renal function assessed at the time Mr Henri presented at the RHH indicates at least a 4 day course of renal injury related to hypotension.

I accept these opinions.

Findings, Comments and Recommendations:

As advised by Dr Lawrence, the RHH did not retain a sample of Mr Henri's blood taken at the time of his presentation to the Emergency Department and hence it has not been possible to establish, by toxicology testing, the level of opiates in his blood at that time. This circumstance prevents me from making a positive finding that opioid overdose either caused or contributed to Mr Henri's death. Nevertheless, there are some aspects related to Mr Henri's drug management over the days preceding his hospitalisation which are concerning and upon which I should comment. They are:

- The decision by Dr Riley to administer morphine with a starting dose of 30mg per day when Mr Henri was elderly and opiate naïve. In my view, based upon the opinion of Dr Bell, a more appropriate course would have been to use a starting dose of 15mg.
- Dr Riley's expectation that the nursing staff at the Home would closely monitor Mr Henri after he was commenced on morphine. It seems this expectation was not realised as the Home records indicate that it did not alter its previous observation regime and only made clinical observations on a daily basis.

It is clear that the management of Mr Henri's pain presented a very real difficulty for Dr Riley and the Home's staff. With the benefit of hindsight it may have been prudent to have involved a pain-management specialist in his care.

For the reason explained, the absence of any ante-mortem blood samples has hindered this investigation. This leads me to **recommend** that all hospitals in this State investigate and, if reasonably practical, adopt protocols to ensure the identification of those patients who present with a possible drug-related illness and the retention of a sample of their blood, either until their discharge or, in the case of death, for provision to the State Forensic Pathologist or his assignee.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, and cause of death, relevant circumstances concerning how his death occurred and the particulars needed to register his death under the *Births, Deaths and Marriages Registration Act* 1999. I do not consider that the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me.

I conclude this matter by conveying my sincere condolences to Mr Henri's family and loved ones.

Dated the 12th day of November 2015 at Hobart in the State of Tasmania.

Rod Chandler
Coroner

APPENDIX A (CORONERS REPORTS (XXIII)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Olivia McTaggart, Coroner, having investigated the death of Tyler John Broomhall

Find, pursuant to Section 28(1) of the *Coroners Act* 1995, that:

(a) The identity of the deceased is Tyler John Broomhall;

(b) Mr Broomhall died in the circumstances set out in this finding;

(c) Mr Broomhall died as a result of intra-cerebral haemorrhage following methylamphetamine use;

(d) Mr Broomhall died on 20 April 2013 at the Royal Hobart Hospital, Hobart in Tasmania; and

(e) Mr Broomhall was born in Devonport, Tasmania on 15 February 1990; he was aged 23 years and was employed as a factory hand at the date of his death.

In making the above findings I have had regard to the evidence gained in the comprehensive investigation into Mr Broomhall's death. The evidence comprises an opinion of the forensic pathologist who conducted the autopsy, relevant police and witness affidavits, medical records and reports, and forensic evidence.

Mr Broomhall was a fit, young man who played Australian Rules football with the Wilmot Football Club. He worked as a casual factory hand at Harvest Moon, a vegetable processing farm at Forth. He was a habitual cannabis user. It is unclear on the evidence as to the extent that he used other illicit drugs.

Mr Broomhall received a minor head injury at work about 4 to 6 weeks before his death, which he did not report. In the weeks leading up to his hospitalisation and death it would appear that this injury caused Mr Broomhall to become forgetful, emotional, and lethargic and to lose motivation. He was described as "not being himself." He did not seek medical attention for these symptoms. It would appear that

his use of cannabis, and possibly methylamphetamine, exacerbated the effects of the head injury, giving him severe headaches.

On 13 April 2013, Mr Broomhall played football and received a heavy knock during the first half of the game. Post mortem examination indicated that this caused a rib fracture.

Mr Broomhall's mother and girlfriend stated in their affidavits for the investigation that they were told by one of Mr Broomhall's friends that Mr Broomhall had used the drug "ice" (being methylamphetamine) at half time to assist with keeping him on the ground. There were no eye witnesses to Mr Broomhall using this substance. However, from the result of blood tests taken on his presenting to hospital, I am able to find that he did use the substance. I am unable to say if this use was a one-off event or if he had been taking the drug for any extended period before the football match.

At 5.05pm the same day, being 13 April 2013, Mr Broomhall was transported by ambulance to the emergency department of the North West Regional Hospital (NWRH). His chest trauma suffered in the football game was noted by medical staff. His history of vomiting and coughing up blood-streaked sputum was also documented. It was recorded that he had consumed methylamphetamine and that he was confused and agitated.

Serial electrocardiograms (ECG) were conducted upon Mr Broomhall. The doctors were of the opinion that the results showed ECG changes consistent with acute myocardial injury (heart muscle injury). Mr Broomhall was therefore treated with aspirin, being a standard treatment for a myocardial infarction. Over a period of two hours he returned to his normal self and insisted on discharge.

At 10.30pm Mr Broomhall was admitted again, on this occasion to the Mersey Community Hospital, (MCH), upon request of medical staff at the NWRH. Due to concerns about myocardial injury he was treated enoxaparin (an anti-coagulant). The admission was for monitoring and management of amphetamine induced myocardial injury.

At 3.00am on 14 April 2013 Mr Broomhall was noted to be hyper-vigilant, slightly agitated and to have dilated pupils.

At 5.00am Mr Broomhall was noted to be confused and disorientated, with his condition deteriorating. At 6.15am he was reviewed by Dr David Selvanayagam. Intracranial pathology was suspected and a CT scan of the brain was therefore requested.

Later that morning, a CT scan was conducted which showed a large frontal lobe haemorrhage. He was therefore transported to the Royal Hobart Hospital at 3.15pm that afternoon. At the Royal Hobart Hospital Mr Broomhall underwent further CT

scans and was subsequently taken to surgery. A left frontal craniotomy and evacuation of the hematoma was performed. Over the following days his condition did not improve, and he remained in hospital.

On 20 April 2013 Mr Broomhall suffered further intracranial hypertension despite full medical management. Therefore a left sided decompressive hemicraniectomy was performed. However, his condition deteriorated and, unfortunately, he passed away.

On 22 April 2013 Dr Christopher Lawrence performed an autopsy upon Mr Broomhall at the Royal Hobart Hospital.

In his report Dr Lawrence stated:

"Autopsy reveals an intracranial haemorrhage in the frontal area which appears to be a primary intracranial haemorrhage rather than extension of a cerebral contusion. The heart shows mild enlargement and contraction band necrosis consistent with methylamphetamine cardiac damage.

Possible explanations for the cerebral haemorrhage include:

1. *An earlier injury complicated by hypertension due to methylamphetamine. The neurosurgeon Mr Jens Peter Wilke thought that there was some older haemorrhage present at the craniotomy on 15 April 2013. However, no haemosiderin was detected in the specimen sent to Anatomical Pathology to confirm this observation.*
2. *Combined effects of an injury sustained during the football game and the effects of methylamphetamine. While there was a left rib fracture there was no evidence of head trauma.*
3. *Hypertensive haemorrhage due to the effects of methylamphetamine.*

His presentation at NW Regional Hospital and later at the Mersey include cardiac features as well as neurological features which I think fit best for cardiac features of methylamphetamine toxicity. Methylamphetamine was identified on drug screen and on toxicology analysis of ante-mortem blood. I think that methylamphetamine is the major factor in the intracranial haemorrhage.

This case highlights the danger of recreational use of methylamphetamine in sport."

Dr Lawrence cited the cause of death as intra-cerebral haemorrhage following methylamphetamine use. I accept his opinion.

It was during the course of the coronial investigation that it was discovered that Mr Broomhall had sustained the head injury, referred to earlier, while at work 4 to 6 weeks prior to his hospitalisation on13 April 2013. This involved him hitting his head on a piece of machinery while cleaning underneath it. He did not report this as a work injury despite being urged to by a workmate who witnessed the incident. Although he had a visible lump on his head as a result of the knock, it did not break

the skin and he considered it too minor to report. The circumstances of the injury were investigated by Workplace Standards. As a result of the evidence in the investigation I am satisfied that the injury was minor and the piece of machinery concerned did not present any particular risk to an employee operating the machinery. I note that the employer fully cooperated in the investigation and has now taken steps to cover the sharp edge in question.

A drug squad investigation has not been able to determine the source of the methylamphetamine used by Mr Broomhall.

Comments and Recommendations

Late diagnosis of head injury

I note that investigations initially led to Mr Broomhall being treated for a cardiac problem caused or exacerbated by drug use. There was a 6 hour delay in him being correctly diagnosed with an intracranial haemorrhage.

Dr Anthony Bell, coronial medical consultant, has reviewed Mr Broomhall's medical care and provided an opinion at my request. In his report, Dr Bell notes that Mr Broomhall's ECG changes had not progressed as would be expected with myocardial injury. Thus the diagnosis should have been reviewed before the administration of the anti-coagulant medication. The non-specific ECG changes without progression actually led to treatment of an acute myocardial infarction which was not present. Dr Bell stated that the treatment was with anti-coagulation and aspirin both of which are contra-indicated in cerebral haemorrhage due to increased risk of bleeding. Thus, the poor interpretation of the ECGs led to inappropriate treatment. However, he further notes that the diagnosis of a cerebral haemorrhage was not possible by clinical examination and there was no specific reason to perform a CT scan of Mr Broomhall's brain at initial presentation.

The diagnosis would have been especially difficult as Mr Broomhall appeared to have minimal signs, if any, of cerebral haemorrhage and gave no history of his earlier head injury. Therefore, given the difficulty of the diagnosis of intracranial pathology, I am not able to say that the standard of care was inadequate.

Director of emergency at North West Regional Hospital, Dr Marielle Ruigrok, has provided a helpful report for the coronial investigation regarding the hospital's review of the delay in diagnosis. She noted that, upon analysis, an emergency physician rather than an anaesthetist, should have been on-call, which, as a result of a review, is something that now occurs. Dr Ruigrok also noted that locum doctors on duty were unfamiliar with the MCH systems and equipment. If it has not already done so, the MCH might consider reviewing whether the orientation system in place for locums is sufficiently robust.

I am not able to find that Mr Broomhall's outcome would have been different had he been correctly treated at an earlier time.

Dangers of methylamphetamine use

This finding is published together with my finding in respect of the death of another male who also died after consuming methylamphetamine whilst playing football. It is therefore appropriate to make the following comments regarding the dangers of the consumption of methylamphetamine generally and, more particularly, whilst playing sport or after exertion. I hope that publication of these findings will emphasise the tragic consequences and significant harm that results from its use.

Methylamphetamine (also known as methamphetamine) belongs to the stimulant class of drugs, which also includes amphetamine, ecstasy, and cocaine. These drugs stimulate the brain and central nervous system, resulting in increased alertness and physical activity.

There are three main forms of methylamphetamine: powder (speed), base, and crystal. The research shows that of particular concern is the crystalline form of methylamphetamine, known as "ice". This is the substance likely consumed by Mr Broomhall before his death.

In 2013, 7% of Australians reported that they had used methylamphetamine in their lifetime, and 2% reported using in the past 12 months. Whilst the same proportion of Australians use methylamphetamine now as in 2007, the frequency, form and method of administration has changed, resulting in greater harms.[1]

The availability of crystal methylamphetamine has increased, with more users reporting using crystal methylamphetamine rather than lower purity powder methylamphetamine and more regular use. Amphetamine related helpline calls, drug treatment, arrests and hospital admissions per amphetamine disorders and psychosis have all increased steeply since 2010. The increased availability and use of crystal methylamphetamine have been associated with increased regular use and harms.[2]

It is estimated that in 2013-2014 there were 268,000 regular methylamphetamine users aged 15 to 54 years in Australia. This equates to population rates of 2.09% as regular users and 1.24% for dependent use. This study also noted that the rate of dependent use had increased since 2009–2010, most markedly among young adults aged 15 to 34 years.[3]

The National Coronial Information System records that from the period 1 January 2011 until 31 December 2015 there were 1193 deaths reported to a coroner in Australia where consumption of methylamphetamine was determined to be the primary cause of death. Males comprised the majority of these deaths. The National

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[1] Roche et al; Methamphetamine Use in Australia; National Centre for Education and Training on Addiction, Flinders University.

[2] Degenhardt et al; Crystalline methamphetamine use and methamphetamine-related harms in Australia; Drug Alcohol Rev 2016 Jun 11;

[3] Degenhardt et al; Estimating the number of regular and dependent methamphetamine users in Australia, 2002 – 2014; MJA 204 (4) 7 March 2016

Coronial Information System report notes that the actual number of deaths is likely to be greater due to the number of cases still open.

Dr Bell has provided me with a summary of the effects of the use of methylamphetamine, derived from the medical literature. I have, below, extracted the main points from his summary.

Methylamphetamine stimulates the central nervous system, producing behavioural and physiological effects. It has a high potential for abuse and addiction. It may result in fatality in some individuals when used alone on a single occasion.

Use is associated with serious cardiovascular events, including sudden death in patients with pre-existing structural cardiac abnormalities or other serious heart problems. At higher doses there may be dramatic increases in heart rate, blood pressure, respiration and temperature. Hypertensive crisis, hyperthermia, and refractory arrhythmias are associated with severe intoxication.

Methylamphetamine use can cause or exacerbate focal neurologic deficits (impairments of brain, spinal cord and nerve functions) such as central nervous system ischemia, infarction, or haemorrhage. Use may lead to new onset or breakthrough seizure activity. Headaches, nausea and insomnia are also commonly associated with use.

Both acute and chronic methylamphetamine use is strongly associated with a variety of psychiatric symptoms, including anxiety, paranoia and psychosis, delusions, homicidal and suicidal ideation, aggressive and hostile behaviour. Choreiform movement disorders (rapid, jerky involuntary movements) are a relatively common finding in acute methylamphetamine intoxication. Hypervigilance and akathisia (a need to move constantly) may be present in mildly intoxicated persons, while severe intoxication may cause abrupt changes in behaviour, becoming extraordinarily violent. Psychiatric symptoms are often the chief complaint of patients presenting to the emergency or acute care setting.

The main psychological effects of methylamphetamine include wakefulness, alertness, a decreased sense of fatigue, mood elevation, increased self-confidence, and a decreased appetite. Ingestion prevents the safe performing of tasks which require mental alertness. Methylamphetamine use does not create extra physical and mental energy but merely affects the perception of fatigue and pain. Therefore, a person engaged in athletic activity may feel as though they are moving faster and getting stronger when this is not the case. Moreover, methylamphetamine is notable for distorting the user's perception of reality and impairing judgement, which may cause an athlete to participate while ill or injured, possibly leading to exacerbation of pre-existing injuries or illness. Methylamphetamine also predisposes the user to excessive exertion which can lead to heatstroke and rhabdomyolysis (muscle breakdown).

In Mr Broomhall's case, I am not able to determine the extent of his past use of methylamphetamine or the dose ingested during the football match. However, the

evidence at autopsy shows that his heart displayed methylamphetamine-related damage, and also that the pre-existing brain injury suffered at work was significantly worsened by his ingestion of methylamphetamine during a period of physical exertion. The worsening of his condition, being increased haemorrhage, led to his tragic death.

I extend my appreciation to investigating officer, Senior Sergeant Darren Pendlebury, for his thorough investigation and report.

I convey my sincere condolences to the family and loved ones of Mr Broomhall.

Dated: 12 September 2016 at Hobart in the State of Tasmania.

Olivia McTaggart
Coroner

APPEDIX A (CORONERS REPORTS (XXIV)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (without inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Maureen Ann Rogers

Find:

(a) The identity of the deceased is Maureen Ann ROGERS ('Mrs Rogers').

(b) Mrs Rogers died on 3 November 2012 at Unit 2/6 Kensington Gardens in Norwood.

(c) Mrs Rogers was born at Colchester in England on 24 May 1941 and was aged 71 years.

(d) The cause of Mrs Rogers' death was a cardiac tamponade due to an ascending aortic dissection.

Circumstances Surrounding the Death:

Mrs Rogers was a widow and a retired Probation Officer and Teacher's Aide. She resided alone at Unit 2/6 Kensington Gardens in Norwood. Her past medical history included osteoarthritis, hip replacement, spinal stenosis and sciatica. She also suffered from macular degeneration.

In the early hours of 2 November 2012 Mrs Rogers developed a sudden onset of epigastric or stomach pain. An ambulance was called and she was assessed by paramedics. They were concerned that Mrs Rogers had an acute coronary syndrome. Pain relief required 8mg of intravenous morphine. Mrs Rogers was transported to the Launceston General Hospital ('the LGH') and presented to the Emergency Department. ('the ED').

Mrs Rogers was seen by Dr Kannan Ramanathan at 8.19am. He recorded pain in the epigastric region with no chest pain and no radiation of the pain. It was described as burning in nature. Mrs Rogers indicated that it had been present for a month. Clinical examination noted epigastric tenderness. A diagnosis of gastritis was made. The treatment plan was for a repeat ECG, repeat troponin testing and administration of pain relief. (In total Mrs Rogers was administered 20mg of morphine whilst in the ED). Because of differential diagnoses of duodenal perforation and thoracic aortic dissection a CT scan of the abdomen was also ordered. The clinical information provided to the radiologist was, *"Suspected duodenal perforation/dissection of aorta."*

A non-contrast CT scan of Mrs Rogers' chest and abdominal aorta was done at 12.08pm. (It was non-contrast because Mrs Rogers was allergic to iodine). The radiologist was Dr Anil Gupta.

His report noted: *"No acute abnormality seen in the chest and abdomen in this non-contrast examination."*

At 3.00pm Mrs Rogers was medically reviewed. At this time her pain was described as central stabbing chest pain (severity 8/10, radiating to the back and also to the epigastrium but *"predominantly the pain was in the chest."*) It was relieved by leaning forward. Her vital signs were recorded as stable. An ejection systolic murmur was noted in the aortic area. The ECG changes were sighted by Dr Thomas David. The management plan was for no acute cardiac intervention. The troponin test was repeated and the value recorded as low.

At 3.20pm the nursing staff recorded Mrs Rogers' pain in these terms; *"9/10 pain in epigastric, retrosternal, mid-scapular more left sided."* However, by 5.30pm the pain in her chest had subsided but there remained mild pain in the posterior thorax scapular region. Dr Ramanathan discussed the level of Mrs Rogers' pain and the test results with her and her son, Ian. It was recommended that she go home. She was advised to return to the hospital if the pain persisted or increased.

Mrs Rogers was taken home by her son. He left her unit at about 6.30pm. At about 8.15am the following day a neighbour, Ms Lola Cortnage, called on Mrs Rogers to check on her. She called out to her but did not get a response. She believed that she may have been asleep. She then telephoned Mr Ian Rogers and suggested that he return to his mother's unit to check on her. At about 8.30am Mr Rogers arrived at the unit. He found his mother in her bed. She appeared deceased. An ambulance was called and promptly attended. Paramedics examined Mrs Rogers and were satisfied that she had passed away. Officers of Tasmania Police then attended and undertook a brief inspection. They were satisfied that there was no evidence of any suspicious circumstances.

Investigation:

State Forensic Pathologist, Dr Christopher Lawrence, carried out a post mortem examination. His report includes this comment; "Autopsy revealed a cardiac tamponade, as well as dissection of the ascending aorta, with a tear within the tunica intima, located immediately proximal to the origin of the right subclavian artery. The dissection extended distally into the aortic arch and descending aorta and in a retrograde direction towards the heart and around the proximal coronary arteries. A second tear within the tunica intima was present within the descending thoracic aorta. There are no aortic aneurysms."

In Dr Lawrence's opinion the cause of Mrs Rogers' death was a cardiac tamponade due to an ascending aortic dissection. I accept this opinion.

The investigation of the circumstances of Mrs Rogers' death has also included the following:

1. The consideration of affidavits provided by Dr Kannan Ramanathan, Dr Thomas David and jointly by Dr Arvind Madras and Dr Ramanathan Parameswaran.

2. An examination of Mrs Rogers' hospital records.

3. Consideration of a report provided by Dr A J Bell in his capacity as Medical Consultant and Adviser to the Coroner.

4. Consideration of a letter provided by Messrs Murdoch Clarke, solicitors, and written on behalf of Radiologist, Dr Anil Gupta. Enclosures with that letter included reports

from Dr Gupta and radiologist, Dr Darren Ault. (The report from Dr Ault had been sought by the Australian Health Practitioners Regulation Agency (AHPRA))

5. Consideration of a report provided by Dr Michael Carr, Director of Medical Imaging at the Royal Hobart Hospital.

6. Consideration of a second letter provided by Messrs Murdoch Clarke and including a further report provided by Dr Gupta.

7. Consideration of a report provided by radiologist Dr Philippa Taplin. This report had been commissioned by Dr Gupta's solicitors.

In my previous findings I commented that an affidavit had been sought from Dr Gupta but had not been received. Dr Gupta's solicitors have since provided material which satisfies me that the LGH administration failed to inform Dr Gupta of multiple requests made by the Coroner's Office for Dr Gupta to provide an affidavit. It follows that any inference that Dr Gupta has not co-operated with the coronial investigation should not be made.

It is evident that this investigation has brought to light multiple opinions upon the interpretation of Mrs Rogers' non-contrast CT scan. It is helpful for me to set out the salient points from each of them.

I begin with Dr Gupta. The representations made on his behalf coupled with the contents of his own reports indicate these matters:

- Dr Gupta acknowledges that a review of the imaging does reveal a displacement of mural calcification which he describes as a subtle indirect sign of intramural haematoma and which raises the suspicion of dissection.

- That Dr Gupta, whilst aware that the displacement of mural calcification was a sign of dissection, had not previously seen this sign in practice.

- That Dr Gupta accepts that he failed to recognise the displacement of the mural calcification and as a result a dissecting aorta was not diagnosed and recorded in his original report.

- That Dr Gupta very much regrets his error and acknowledges the distress it has caused Mrs Rogers' family and friends.

In Dr Ault's view the images demonstrate an "abnormal separation of the calcified intima in the aortic arch extending into the descending thoracic and abdominal aorta indicative of an aortic dissection." He describes the findings as "subtle" but says they should have been appreciated by Dr Gupta.

When providing his report Dr Carr had access to the reports of both Drs Gupta and Ault. He also was able to view the original images of Mrs Rogers' CT scan. He makes these comments:

- A diagnosis of dissection of the aorta was obvious from the CT scan and should have been made by Dr Gupta.

- The error made by Dr Gupta was inexplicable given that he was specifically requested by the clinician to exclude dissection of the aorta as the diagnosis.

- That Dr Ault considered the diagnosis of a dissection to be "straightforward."

- In his report Dr Ault used the word "subtle" in its usual radiological sense to mean that the signs of a dissection on the scan were clearly visible if assessed carefully. It was wrong to imply that the word "subtle" was intended to mean that the signs of a dissection were difficult to see. They were not.

In Dr Taplin's opinion an aortic dissection is visible on the CT scan. She says, "There is a hyperdense crescent in the ascending aorta, clearly visible on these non-contrast scans. This is a well recognised sign of dissection. There is also stranding and hyperdensity of the mediastinal fat, indicating leak/rupture." Dr Taplin also describes the findings as "subtle" but says a radiologist with experience in an acute hospital emergency department should not have missed the diagnosis.

In his report Dr Bell has made the following comments which I consider particularly pertinent:

- The initial assessment undertaken by Dr Kannan Ramanathan was appropriate and thorough. However, the history of sudden onset pain in the epigastric region requiring 8mg of morphine intravenously for relief is more suggestive of a cardiovascular event than it is of gastritis.

- Mrs Rogers was taking a proton pump inhibitor for treatment of her reflux. This is a class of drugs designed to reduce stomach acids. It is those acids which cause gastritis. This makes the diagnosis of gastritis unlikely in Mrs Rogers' case and this is particularly so given that her level of pain required management by administering a moderately high dose of morphine. (In the ED Mrs Rogers was administered 20mg of morphine. This was in addition to the 8mg given by the ambulance officers).

- It would ordinarily be difficult to detect a thoracic aortic dissection by a CT scan done without intravenous contrast. However, in this instance the aortic dissection is clearly visible due to the displaced calcification within the intima of the aortic wall. Also, the colour differentiation between the centre of the aorta to the outer rim area of the aorta suggests dissection. The dissection should have been recognised by Dr Gupta and reported.

- Dr Gupta should have advised the medical staff that a non-contrast CT scan was not an acceptable means of eliminating aortic dissection.

- By the time of her 3.00pm review Mrs Rogers' presentation had changed. She had severe stabbing chest pain radiating to the back and relieved by leaning forward. A systolic ejection murmur was noted in the aortic area. Right coronary artery flow difficulties were discernible on ECG. These elements were all suggestive of an aortic dissection.

- It was unsound to discharge Mrs Rogers home with a diagnosis of gastritis which had necessitated management with morphine, particularly as she had been taking a proton pump inhibitor for her reflux.

- Survival by a person suffering an aortic dissection is rare unless treated. Treatment requires an immediate reduction in blood pressure with surgery involving replacement of the aortic root and the aortic heart valve. This is known as the Bentall's procedure. It would have necessitated Mrs Rogers' transfer to the Royal Hobart Hospital.

- The Bentall's procedure is major surgery. There is a 40 to 50% mortality rate for persons of Mrs Rogers' age. There is also a 10% risk of stroke or other vascular complications. Survivors of the procedure usually make a good recovery.

Findings, Comments and Recommendations:

It is clear upon the evidence and I so find, that at the time of Mrs Rogers' presentation to the LGH she was suffering from an aortic dissection which required immediate surgery. Although that surgery involved substantial risk the failure of the LGH medical staff to properly diagnose Mrs Rogers and recommend the necessary treatment denied her all opportunity of a full recovery and an extended life.

I have set out in detail the evidence upon the CT scan. It leads me to make these points:

- The scan showed signs of an aortic dissection which should have been appreciated by Dr Gupta and the diagnosis made.

- It was an error on Dr Gupta's part to record *'no acute abnormality'* on his report of the CT scan. Dr Gupta now acknowledges this error. The report should have included the diagnosis of an aortic dissection.

- There are differing opinions on whether the signs of an aortic dissection visible on the scan should be properly described as 'subtle.' This debate is, to my mind, largely irrelevant. This is so because I am satisfied that the signs, however described, were sufficiently clear on the scan to be recognised by an experienced hospital radiologist such as Dr Gupta.

There were, as Dr Bell has pointed out, aspects of Mrs Rogers' clinical picture consistent with a diagnosis of an aortic dissection, more so than gastritis. However, it is apparent, and I so find, that the medical staff were misled by Dr Gupta's CT scan. It is a matter of concern that Dr Gupta did not make the diagnosis of aortic dissection and report upon it. It is also of concern that Dr Gupta did not include in his report a warning to the treating clinicians that the absence of contrast made it difficult to detect an aortic dissection. Had this warning been conveyed to them it is possible that the clinicians may have placed less reliance upon the scan results and more reliance on those other signs consistent with a diagnosis of an aortic dissection.

The circumstances surrounding Mrs Rogers' unfortunate death lead me to **recommend** that the LGH undertake a review of the competencies of its radiological staff with a view to putting in place, if deemed necessary, processes for their updated training and the proper supervision or monitoring of their work.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how her death occurred and the particulars to register the death under the *Births, Deaths & Marriages Registration Act 1999*. I do not consider that the holding of a public inquest would elicit any significant information further to that already

disclosed by the investigation conducted by me. The circumstances of the death do not require me to make any further comments or other recommendations.

I conclude this matter by conveying my sincere condolences to Mrs Rogers' family.

Dated: 22 April 2015 at Hobart in the state of Tasmania.

Rod Chandler
CORONER

Addendum

On 3 December 2014 Coroner Olivia McTaggart, as Delegate of the Chief Magistrate for the State of Tasmania, directed that the investigation by Coroner Rod Chandler into the death of Maureen Ann Rogers be re-opened. That direction was given following information being received indicating that radiologist, Dr Anil Gupta had not been given an opportunity (due to the failings of the management of the Launceston General Hospital) to provide the Coroner with information which he considered relevant to the circumstances of Mrs Rogers' death. The above findings of Coroner Chandler replace his previous findings made 28 April 2014, and have regard to information provided by Dr Gupta and further investigations made.

APPENDIX A (CORONER'S REPORTS (XXV)

MAGISTRATES COURT *of* TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Simon Cooper Coroner, having investigated the death of Mr P

Find, pursuant to Section 28(1) of the Coroners Act 1995, that:

(a) The identity of the deceased is Mr P;

(b) Mr P died in the circumstances set out further in this finding;

(c) The cause of Mr P's death was a small bowel infarction due to a volvulus owing to adhesions in the small intestine;

(d) Mr P died in October 2012 at the Royal Hobart Hospital, Liverpool Street, Hobart in Tasmania;

(e) Mr P was born in Tasmania in 1948 and was aged 64 at the time of his death.

Introduction:

Mr P was born in Tasmania in 1948. His parents were GJ and AP. He was raised by his aunt, IP, and had 11 siblings.

Mr P's widow reports that he had a disrupted childhood, and as a teenager liked to play football. He was educated to high school level and gained employment aged 14.

Mrs P met Mr P in November 1971 in Tarraleah where he was working and she was living. A week after they met they commenced going out. A year later they were married. Mrs P describes him as the love of her life. Together they raised four boys; three of whom were Mr P's natural sons, and the fourth, who was eight months old when Mr and Mrs P met, was raised by Mr P as if he was his own child.

Mr P worked for the same company for 32 years.

Self-evidently Mr P was a hard-working man who carted wood on weekends; often being helped by his sons. The whole family would, at Christmas time, pack up and go fishing and camping for a week at a time.

In 1993 Mr P was electrocuted at work and suffered burns to 47% of his body. Mrs P reports that after that time he could not really work.

Mr P's medical history includes suffering from sciatica in about 1980, depression due to his not being able to work, and the removal of his appendix in 2005. He also suffered from a stomach hernia, which was repaired surgically in 2008, and had some minor heart problems.

Mrs P reports that from 2009-2010 Mr P suffered from off and on abdominal pain. He also had cancer of the sinuses which was treated, again by surgery, in about 2007.

Circumstances of the Death:

At the time of Mr P's death he and Mrs P were living together in southern Tasmania, in the family home. On the morning of his death they both got up as Mrs P had two appointments in town and Mr P was intending to go to a social function with a friend. The friend picked Mr P up at 11.00am. At 11.30am Mr P rang Mrs P and asked her to pick him up saying "god I am crook". She did so and took him straight to the Department of Emergency Medicine at the Royal Hobart Hospital.

Emergency Department medical notes, obtained as part of the coronial inquiry, indicate that Mr P presented at 12.46pm on the day in question. He was then suffering from severe central abdominal pain radiating to the right lower abdomen. Mr P was complaining of nausea but had not then vomited.

The notes indicate that vital signs were recorded at 1.30pm as a temperature of 36.5°C, heart rate of 84 bpm (although an electrocardiogram around the time shows a heart rate of 106 bpm), respiratory rate of 14 bpm, blood plate pressure of 179/108mmHg, and a pain score of 8/10. In addition, cheek dustiness was noted.

Mr P was seen by an Emergency Department consultant. The records have no entry with respect to the assessment of neck veins, fluid status or heart recorded. There is a record showing the chest had coarse basal crepitations. The notes indicate that the abdomen was distended and generally tender. The diagnosis was made by the consultant of a bowel obstruction. The consultant ordered x-rays and blood tests and a surgical referral was considered.

Mr P was admitted to a ward at 7.30pm. The notes indicate that he was still indicating a pain score of 8/10 and by now he had vomited twice. The notes record that he was seen by a doctor (although regrettably, no name or designation of that doctor is recorded in those notes). The doctor apparently wrote a formal admission note. The notes have minimal information about pain. The examination (if one was conducted at all) records no vital signs, no chest examination, and no cardiac examination. There was no fluid status assessment. The abdomen is described as grossly distended with percussion tenderness and perionitic. A rectal examination showed faeces. There was no comment in the notes on chest or abdominal x-rays being conducted; though some blood test results are recorded without comment, in particular, the white blood cell count and the presence of the left shift. A high haemoglobin level is recorded but not commented upon.

Nursing notes suggest that Mr P's pain had worsened. Nurses seem to have recalled the surgical senior increasing pain relief.

The notes next record a plan which was to admit Mr P under a general surgeon, and to conduct other treatments. Once again no diagnosis is recorded, no differential diagnosis

is recorded, and there is no consideration of a number of important aspects of the case.

Specifically, there was no attempt to consider the more lethal complications of small bowel obstruction, strangulation of the bowel, perforation, and/or peritonitis. No assessment seems to have been done appropriately (or indeed at all), and if it had, and the conclusion was that Mr P had a complicated bowel obstruction, he may well have survived.

Such a diagnosis is a clinical diagnosis. It requires thought and consideration of the facts. This care was not provided to Mr P.

The Royal Hobart Hospital was afforded the opportunity to respond specifically to the findings made above. The response to that request was unsatisfactory. The consultant was also afforded an opportunity to respond to the findings as indicated above. His response, through the medico legal adviser to the Tasmanian Health Service (and not from him personally), was to advise "that he is currently [sic] not in a position to add anything further to the clinical notes as he is unable to recall any further factual matters".

The other doctor identified as being involved in Mr P's treatment, also responded through the medico legal adviser to the Tasmanian Health Service. By letter, dated 17 December 2015, the medico legal adviser stated that she had "been in contact with [that doctor] to discuss this matter and [had] taken the step of forwarding to her all the information relevant to the case". Ms Dewhurst, the medico legal adviser, went on to request that, "the timeframe for delivery of any response from [that doctor] was left open until the required [*sic*] deadline of 24 December 2015". Time was extended accordingly but no response, regrettably, has been received by, or on behalf of, the doctor within that timeframe or at all.

I am satisfied in the circumstances that the assessment in the Department of Emergency Medicine carried out by the consultant on Mr P did not consider the complications of a bowel obstruction. On the ward, the surgical registrar, plainly found signs of complications of small bowel obstruction, but either did not understand or act in relation to what was by then a critical situation.

The standard of care afforded to Mr P by his doctors and the Royal Hobart Hospital generally, was not of an appropriate standard. Mr P was admitted to hospital with a condition that had it been properly diagnosed and treated was eminently survivable.

Comments and Recommendations:

It should be clear from the foregoing that I am of the view that the treatment afforded to Mr P fell well short of an acceptable standard.

I conclude this matter by conveying my sincere condolences to Mr P's family.

Dated: 27 June 2016 at Hobart in the State of Tasmania.

Simon Cooper
CORONER

APPENDIX A (CORONERS REPORTS (XXVI)

MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION

IN THE MATTER OF THE *CORONERS ACT* 1995

AND

IN THE MATTER OF AN INQUEST TOUCHING THE DEATH OF NEVILLE ROBERT HOSKINSON

FINDINGS, RECOMMENDATIONS AND COMMENTS of Coroner Rod Chandler following an inquest held in Launceston on 6, 7 and 8 February 2017.

31 March 2017

Introduction

On 17 May 2012 Mr Neville Robert Hoskinson died at the Launceston General Hospital (LGH). The cause of his death was a cardiac tamponade resulting from a proximal aortic dissection due to hypertension. On 6, 7 and 8 February 2017 an inquest was held concerning the death. Its focus was upon Mr Hoskinson's medical care provided by the LGH. These are the findings arising from that inquest.

Background

Mr Hoskinson resided with his wife Leeanne and their son at Waterhouse in North-East Tasmania. He was aged 55 years and was employed as a farm manager. He enjoyed good health although he had a family history of ischaemic heart disease.

Circumstances Preceding Presentation to LGH

In the afternoon of Monday 15 May 2012 Mr Hoskinson telephoned his wife at her workplace to tell her that he had severe chest pain and headache. She arranged for a work colleague to collect her husband from their home and drive him to the Barnbougle Golf Course. There he was met by an ambulance and conveyed to the North East Soldier's Memorial Hospital at Scottsdale arriving at around 4.15pm. He was promptly seen by Dr R Skerka who recorded: *"Acute onset severe (10/10) chest pain, sub sternal with radiation to neck/head/jaw."* He was administered 2 mg of morphine and then returned to the ambulance to be transported to the LGH.

Events at the LGH Preceding Death

Mr Hoskinson arrived at the Emergency Department (ED) of the LGH at 6.05pm on 15 May. He was triaged as a Category 3 patient thereby requiring medical review within 30 minutes. At 6.30pm he was seen by ED consultant, Dr Erica Kreismann. She recorded that he had constant pain, increased with inspiration. He denied shortness of breath, palpitations, visual changes, abdominal pain, nausea, vomiting, diarrhoea or back pain. Her physical examination indicated that he was awake, alert and conversant. She noted no bruits on his neck, his abdomen was soft and non-tender and his heart and lung sounds were normal. Neurological examination was normal. The treatment plan was noted as; *"Pleuritic in nature, chest pain minimal at present, complaining of mild headache, unclear aetiology, will check x-ray, labs, D dimer, Troponin. ECG: normal sinus rhythm, 80, nil acute changes."*

The chest x-ray was reported to show a *"?thickened paratracheal stripe."* The troponin level was raised and a repeat test was ordered for the following morning. The D dimer test (used to rule out the presence of blood clots) was also elevated. Because of this result and the *"questionably abnormal x ray"* Dr Kreismann ordered a CT angiogram of the chest to rule out the possibility of an aortic dissection or pulmonary embolus.

The CT angiogram was carried out by radiologist, Dr Anil Gupta. He dictated a report but this was not typed until the following day. He also reported verbally to Dr Kreismann who then made this entry in the notes:

> *"CT brain normal.*
>
> *CT.A chest: stranding in mediastinal fat. Infxn/inflamm/haemorrhage."*

At 11.15pm Mr Hoskinson was seen by medical registrar, Dr Suneet Kohli. He made a very comprehensive record of the attendance in the medical notes. It included a history, symptom details and his examination findings. The symptoms, namely chest pain, jaw pain and headache were not, in his view, typical of aortic dissection. His examination findings including a slightly elevated blood pressure, mildly elevated D dimer test and an ejection systolic murmur were also not suggestive to him, either separately or in combination, of an aortic dissection. As to the CT angiogram of the chest he recorded; *"no dissection/PE. Mediastinal fat stranding? Inflammation?? Infection? Haemorrhage (verbal report by Dr Gupta)."* He noted that the *"issue"* was *"Pleuritic chest pain with headache? cause (no significant findings on imaging)."* Dr Kohli was unable to say whether his recording of the term *"no dissection"* in his notes was a reflection of the information on the aortogram conveyed to him by Dr Kreismann or whether it represented his own conclusion based upon the symptoms, his examination and the scan as verbally reported.

The treatment plan settled by Dr Kohli at this time was to admit Mr Hoskinson to the care of the Medical Team for observation with a repeat ECG and a repeat troponin test to be done the next morning. Also in the morning a transthoracic echocardiogram was to be undertaken as advised by the cardiology registrar.

The following day at 9.30am Mr Hoskinson was seen by Drs Griffin and Batta of the Medical Team. They noted that he had *"slight chest pain when breathing in worse when lying flat."* Of the CT angiogram they noted; *"?mediastinal fat stranding. No dissection."* It was their impression that Mr Hoskinson was suffering from pericarditis and the treatment plan was then noted as:

> *"1. Echo*
>
> *2. Monitor*
>
> *3. Cardiology R/V."*

Sometime on 16 May Dr Gupta's written report upon the imaging became available. As to the CT angiogram of the chest it states:

> *"Examination performed for suspected dissection.*
>
> *Examination shows good flow of contrast through the ascending, arch and ascending aorta. No evidence of dissection seen. No obvious abnormality in aorta or its major vessels noted. There is a good flow of contrast noted through the aorta and its major branches. There is some stranding and increased density of the mediastinal fat seen in prevascular space and aortopulmonary window region. Possible changes of inflammation/haematoma in the mediastinum cannot be excluded completely. Patient should be monitored clinically and followed up.*
>
> *The remainder of the chest and abdomen included in this study demonstrate no obvious/acute abnormality except for the presence of gallbladder stones.*
>
> *Impression:*
>
> *No acute intracranial abnormality seen.*

No evidence of dissection seen.

There is stranding of fat in the prevascular space and the aortopulmonary window region noted raising the possibility of inflammation/haemorrhage/haematoma in the mediastinum in the appropriate clinical context.

Note of GB Stones is made.

Clinical monitoring and follow up advised."

Mr Hoskinson's case notes for 16 May do not contain any entries made by a doctor following the entry made following the review with Drs Griffin and Batta that morning. However, the nursing notes are more forthcoming. They show:

- The repeat troponin test was normal.
- That up to 2.00pm both Mr Hoskinson and the nursing staff were expecting him to undergo the transthoracic echocardiogram.
- That at 2.45pm Mr Hoskinson was reviewed by the Medical Team. This note was made; *"Patient for echo they were rang but don't know anything about it."* The medical team then determined; *"Patient not for echo now"* and that he instead be observed overnight for pain with the possibility of him being able to be discharged the following morning.
- That Mr Hoskinson reported that he was pain free for the balance of the day.

At 9.30am on 17 May radiologist, Dr R Heng, at the request of the Medical Team, reviewed the CT angiogram carried out by Dr Gupta. They reported on that review:

"(Dr Heng) feels that there are some abnormal findings which were not reported:

- *Ascending aortic aneurysm;*
- *Fat stranding-?blood in the mediastinum and pericardial thickening +/- fluid/blood in pericardial sac suggestive of an aortic leak;*
- *Cannot rule out an ascending aortic dissection."*

Following Dr Heng's review the Medical Team sought the assistance of cardiologist, Dr Bhuwan Singh. He saw Mr Hoskinson at 10.30 that morning. He arranged for a bedside transthoracic echocardiogram. It showed normal LV function, trivial to mild posterior pericardial effusion, mildly dilated ascending aorta with mild aortic regurgitation. Dr Singh did not consider that these findings enabled a firm diagnosis of aortic dissection to be made. It was then planned for Mr Hoskinson to undergo a transoesophageal echocardiogram (TOE) which he considered to be the most appropriate diagnostic tool to diagnose aortic dissection. However, this could not be done immediately as Mr Hoskinson had had breakfast that morning.

The TOE was carried out by Dr George Koshy and was given urgent priority at the request of Dr Singh. It was commenced at 12.35pm. It identified a dissection flap during sinus valsalva thus confirming the diagnosis of aortic dissection. However, during the course of the procedure Mr Hoskinson's aorta dissected further causing bleeding into the pericardial sac impacting on the heart's ability to beat. A Code Blue was called and resuscitation carried

out. However, Mr Hoskinson could not be revived and he was declared deceased at 12.57pm.

Post-Mortem Examination

This was carried out by State Forensic Pathologist, Dr Christopher Lawrence. In his report he comments:

> *"Autopsy revealed a proximal aortic dissection with 300 mls of blood in the pericardial sac. The tear is just above the aortic valve. The aortic valve is bicuspid and this may have something to do with the dissection. There is a known association between proximal aortic dissection and bicuspid aortic valve. The proximal aorta does appear slightly dilated."*

In Dr Lawrence's opinion the cause of Mr Hoskinson's death was a cardiac tamponade due to a proximal aortic dissection due to hypertension. Cardiac tamponade occurs when blood or fluids collect in the space between the sac that encases the heart and the heart muscle. This exerts pressure on the heart preventing its ventricles from expanding fully thus limiting its capacity to pump blood to the rest of the body. This leads to organ failure, shock and death. In Mr Hoskinson's case blood from his dissected aorta was the cause for the cardiac tamponade.

Issues Arising

The circumstances surrounding Mr Hoskinson's death have given rise to multiple issues, most particularly centred upon the failure to make a timely diagnosis of Mr Hoskinson's aortic dissection and to arrange for its treatment. Those issues concern:

1. The initial interpretation of the CT angiogram.
2. Communication of the results of the CT angiogram.
3. The failure to undertake a transthoracic echocardiogram in response to a direction of a cardiology registrar made on 15 May.

I will deal with each in turn.

Interpretation of the CT Angiogram

A matter closely examined at this inquest was the adequacy of Dr Gupta's findings upon the CT angiogram performed on 15 May 2012 and in particular whether he should have made the diagnosis of an aortic dissection. Several radiologists gave evidence on this subject and it is necessary that I set out the body of their opinions.

Dr Michael Carr is a consultant radiologist and a former Director of Medical Imaging at the Royal Hobart Hospital. He was retained to provide an opinion upon the CT angiogram and Dr Gupta's report upon it. Dr Carr provided three reports which were put into evidence. He also gave evidence in person. He opined that:

- The thin slice images show an abnormal dilation of the ascending aorta measuring 5.7cm in diameter (the 'norm' is around 4cm) and the descending

aorta is tortuous or elongated. These findings are suggestive of hypertension, a condition which commonly presents in persons who develop a dissection.

- The images show areas of moderately increased attenuation in the anterior mediastinum measuring 25 to 40 Hounsfield units. This is consistent with the presence of blood.
- The images show a Stanford Type A dissection of the antero-lateral aspect of the ascending aorta with leakage of blood into the dissection and leakage of blood into the mediastinum. However, Dr Carr did make this concession; *"I concede that the changes in the ascending aorta wall were difficult to see but were there."*
- Dr Gupta was correct to report the presence of mediastinal fat stranding and his three possible explanations for this are reasonable.
- The crux of the matter to be in these terms; *".....even if Dr Gupta had missed everything else in the report he did see the mediastinal abnormality and if he'd measured the density that would have upgraded the whole matter to a vascular emergency and the actual management of Mr Hoskinson would have been completely different....."*

To summarise, it was the evidence of Dr Carr that Dr Gupta was wrong to report that there was *"no obvious abnormality in the aorta" and "no evidence of dissection seen"* because the ascending aorta is abnormally dilated, the descending aorta is tortuous and the diagnosis of an aortic dissection was not made. Too, he says that the conclusion should have been made that the changes in the mediastinum were due to haemorrhage.

Dr Darren Ault is a radiologist who was retained by the Australian Health Practitioners Regulation Agency to review Dr Gupta's report upon the CT angiogram. A written report from Dr Ault was put into evidence. However, Dr Ault did not make himself available for examination and I did not compel his attendance. In his report he states that he did not believe there was evidence of a dissection flap but agreed the images showed an abnormal increased density in the mediastinal flap as reported by Dr Gupta. He noted the dilated ascending aorta which measured at least 5cm. He considered that the finding of an aortic abnormality was subtle and that pulsation artefact significantly contributed to a lack of diagnostic confidence. He also commented; *"The final report of the CT examination is not incorrect but in retrospect there were secondary findings sufficient to raise the possibility of the presence of a dissection of the ascending aorta such that further investigations either with repeat cardiac gated CT or TOE could have been specifically recommended."*

The other opinion evidence upon the radiology was provided by consultant radiologist, Dr Michael Jones at the request of the Tasmanian Health Service. Dr Jones' evidence can be summarised as follows:

1. He would not make a diagnosis of an acute aortic dissection unless an intimal flap is unequivocally present or, in its absence, there is unequivocal

hyperdense blood in the aortic wall and motion artefact can be excluded as an explanation.

2. Based upon the CT angiogram he would make these diagnoses:

   a) There is no evidence of aortic dissection.

   b) There is calcific aortic stenosis of at least moderate severity.

   c) There appears to be oedema in the superior mediastinum which may be due to infective or noninfective inflammatory disease or trauma.

   d) There is no evidence to suggest it is due to arterial bleeding.

   e) The changes in the aorta wall reported upon by Dr Carr as indicating a dissection were all attributable to motion artefact.

   f) That the findings with respect to the mediastinum and the pericardial sac cannot be assumed to be attributable to blood as a definite pathway for such blood to reach these compartments is not apparent. These findings therefore cannot be said to support a diagnosis of an aortic dissection.

   g) That Dr Gupta's report upon the angiogram was reasonable and in accord with competent professional practice.

Finally, I have already referred to Dr Heng's review of the aortogram made on 17 May which, in his opinion, identified some abnormal findings and did not enable the diagnosis of an ascending aortic dissection to be ruled out. Because of ill-health Dr Heng was unavailable to give evidence.

The factor common to these expert opinions is the absence of unanimity. They demonstrate that radiology is far from an exact science and in cases such as this, where a diagnosis is not plainly evident, experienced practitioners can make significantly different interpretations of the same images. This leads me to conclude that the evidence does not permit me to make a positive finding that Dr Gupta should have made a diagnosis of an aortic dissection following the CT angiogram of 15 May 2012. However, the weight of evidence is, in my view, sufficient to satisfy me that that the angiogram did demonstrate, to adopt Dr Ault's phrase, *"secondary findings"* which raised the possibility of a dissection and which required further investigation by the clinicians caring for Mr Hoskinson. This brings me to consider issues related to the communication of Dr Gupta's findings upon the angiogram and the actions taken.

Communication of the CT Angiogram Findings

The angiogram in question was carried out by Dr Gupta in the evening of 15 May and outside of normal working hours. This meant that there was not any administrative staff available to type up the report after it was dictated by Dr Gupta and to enter it into the

Picture Archiving and Communication System (PAC) so that it could be readily accessed and considered by those persons treating Mr Hoskinson. In the result all communication in the initial stages concerning the results of the scan had to be verbal. I need to consider the consequences which flowed from this state of affairs.

I have set out Dr Kreismann's note of her conversation with Dr Gupta. The first observation to make is that she has recorded infection/inflammation/haemorrhage to explain possible changes shown on the scan in the mediastinum when Dr Gupta's written report makes no reference to infection but instead attributes the possible changes to inflammation/haemorrhage/haematoma.

What of the communication around the possible presence of an aortic dissection? A stated purpose of the CT angiogram was to rule out this possibility and the note made by Dr Kreismann of her conversation with Dr Gupta does not show it to be ruled in or out. In fact it makes no reference to it at all. This is contradicted by subsequent entries in the records with Dr Kohli's notation indicating that Dr Gupta had verbally reported *"no dissection/PE"* and the replication by the Medical Team when they recorded the CT angiogram to show *"no dissection."* There is no evidence to explain how the Medical Team came to make its entry. As I have already noted Dr Kohli explained his entry as perhaps being either a record of the information passed onto him by Dr Kreismann or a reflection of his own opinion of Mr Hoskinson's status based upon the symptoms, his examination and the radiology. In my view the former explanation is more likely given that it is written alongside the phrase 'CT of the chest' and is followed by a further description of Dr Gupta's interpretation of the scan as recorded by Dr Kreismann.

Finally, I need to make comment upon some specific evidence from Dr Kohli. He told the inquest that if he had had access to Dr Gupta's written report when he saw Mr Hoskinson he would have sought further information from Dr Gupta around the sentence; *"Possible changes of inflammation/haematoma in the mediastinum cannot be excluded completely."* Particularly he would have been anxious to have an understanding of the likelihood of any blood being in the mediastinum as this would indicate a serious medical condition requiring an urgent response. If Dr Gupta was unable to be more informative then he would have enquired of other radiological investigations that he would recommend to assist with a diagnosis.

There are, in my view, several conclusions to be drawn from the foregoing. The first is the increased risk of misunderstanding, misinterpretation or wrongful emphasis which can arise from a verbal report upon radiology which in this case is evidenced by:

- Dr Kreismann's apparent understanding that infection was a possible explanation for changes shown in the mediastinum when this was not shown to be so by Dr Gupta's written report. It's noted that Dr Kohli, in his written record, repeated the reference to 'infection.'

- Dr Kreismann's failure to record haematoma as a possible explanation for changes shown in the mediastinum contrary to the written scan report.

- The inconsistency between Dr Kreismann's note which makes no reference to dissection and the written report which stated; *"No evidence of dissection seen."*

- The inconsistency between Dr Kreismann's note of the scan results and the subsequent notes made by Dr Kohli and the Medical Team which record *"no dissection."*

- The contrast between the positive assertion recorded by Dr Kohli and the Medical Team of *"no dissection"* and the less definitive statement made by Dr Gupta in his report of *"No evidence of dissection seen."*

- The absence in Dr Kreismann's report of any reference to the possibility of blood in the mediastinum as described in the written report.

The second conclusion concerns the consequences which can follow when a clinician does not have access to a full written report upon the radiology. Here, as I have noted, it was the evidence of Dr Kohli that if he had had access to Dr Gupta's written report and its reference to the possibility of blood in the mediastinum he would have sought further information of Dr Gupta. A likely outcome of this enquiry would have been Dr Gupta's reinforcement of the decision, already taken by the cardiology registrar, for Mr Hoskinson to undergo a transthoracic echocardiogram.

Dr Kohli impressed me as careful, conscientious and able physician who made meticulous patient notes. I have no doubt that the outcome of any discussion he had with Dr Gupta would have been fully recorded in the notes including any advice concerning an echocardiogram. It may be speculative to say it but if such an entry was made it is likely in my view that it would have militated against the decision later taken by the Medical Team to abandon the request for a transthoracic echocardiogram.

The Delayed Transthoracic Echocardiogram

When Dr Kohli saw Mr Hoskinson at 11.15pm on 15 May he recorded that part of Mr Hoskinson's treatment plan was for a transthoracic echocardiogram to be carried out in accord with the advice of a cardiology registrar. That advice was acted upon the following day by the Medical Team via a referral document which is on Mr Hoskinson's file. It is dated 16 May 2012 and signed by a Dr Jackson as the requesting doctor and by Dr Griffin on behalf of the Team consultant. The time that this document was created is not apparent but the relevant nursing notes which I have already referred to strongly suggest it to have occurred that morning.

It is clear that despite the referral document the echocardiogram had not been done by 2.30pm on 16 May and it has not been possible to establish how this came about. All that is evident is that at that time the Medical Team reviewed Mr Hoskinson, learned that the echocardiogram had not been done and then cancelled the referral.

Dr A J Bell is a specialist intensivist and a former Chief Medical Officer at the Royal Hobart Hospital. He provides advice to the coroner's office upon medical issues. In this instance it was his evidence that the factors warranting an echocardiogram, as identified by Dr

Kreismann and confirmed by Dr Kohli, had not changed and the decision to cancel the procedure was unwise and flew in the face of standard medical practice. In his further view the echocardiogram should have been undertaken immediately. I accept this evidence from Dr Bell.

It was the next day, as I have recorded, that Dr Singh promptly organised a transthoracic echocardiogram following Dr Heng's review of the CT angiogram. When its results did not, in Dr Singh's opinion, permit a diagnosis of aortic aneurysm to be made he arranged for an urgent TOE. However, it was not until 12.35pm that this procedure commenced.

In the result a procedure which began just beyond noon on 17 May would, in all likelihood, have been undertaken a full day earlier if the request completed by Dr Jackson had been acted upon. This raises the question whether the outcome for Mr Hoskinson would have different if the TOE had been undertaken a day earlier and the aortic dissection diagnosed.

Some Findings upon the LGH's Care and Treatment

I am satisfied that Mr Hoskinson received proper and appropriate care and treatment when he presented in the ED on 15 May and no criticism is made of those practitioners who oversaw him at this time, namely Dr Kreismann and Dr Kohli. Of the latter I commend him for the medical notes made of his attendance upon Mr Hoskinson. They are of a standard rarely seen by me and would make a suitable benchmark for all other practitioners. I have previously set out the competing interpretations made of the CT angiogram. For the reason stated I cannot be satisfied that Dr Gupta's failure to make a diagnosis from the scan of an aortic dissection mandates criticism. However, some comment is necessary concerning the reporting of the scan.

As I have noted the LGH did not in 2015 have the capacity to provide contemporaneous written reports upon radiology performed out-of-hours. In the result Dr Gupta had to provide Dr Kreismann with a verbal report on Mr Hoskinson's CT angiogram. I have set out the difficulties that arose in this case which, in my view were directly attributable to the unavailability of a written report. Ordinarily this circumstance would have led me to recommend that the LGH implement a system change to ensure the immediate availability of written radiology reports. (Such a recommendation was in fact made by me in March 2015 following an inquest into the death of Donald John Clarke). However, at this inquest evidence of the current situation upon this subject was provided by Mr Garth Faulkner, the LGH's Chief Radiographer. It was his evidence that in October 2016 the hospital's radiology services were outsourced to a private provider. He said that it was a term of the agreement with the provider that all CT and MRI scans taken of patients in ED, whether during working hours or outside of those hours, required a written report to be provided within one hour of the procedure being completed. This makes otiose any repeat recommendation by me concerning radiology reports.

I now return to the difficulties attributable to the absence of a written report on the scan. I accept Dr Kohli's evidence that if Dr Gupta's written report had been available to him he would have consulted with him concerning the possibility of blood being in the mediastinum. As I have already noted this would, in all likelihood have led to him recording in the medical notes detail of his conversation including advice upon the further radiology required. Such

an entry should have helped to dissuade the Medical Team from its decision, made in the afternoon of 16 May, to abandon the echocardiogram.

Mr Hoskinson had been scheduled to undergo a transthoracic echocardiogram, at least by late morning on 16 May. It did not occur because of an unexplained administrative bungle. The Medical Team, in my view, was in error for the reasons stated by Dr Bell, in cancelling the request for the echocardiogram. It was only after Dr Heng's review of the CT scan the following morning that an echocardiogram was re-ordered. It was then done promptly. However, it was in my view approximately 24 hours later than a proper level of care required.

I accept the evidence of Dr Singh that the results of the echocardiogram were not sufficiently conclusive to make the diagnosis of an aortic dissection and thus commit Mr Hoskinson to major cardio-thoracic surgery. I accept too the evidence that it was appropriate to proceed to a TOE. However, it was delayed by several hours because Mr Hoskinson had been permitted to have breakfast. When it did take place his aorta further dissected leading to his cardiac tamponade and death. I need to record that it was the evidence of Dr Singh, which I accept, that the cardiac tamponade was not attributable to the procedure but was co-incidental to it.

The foregoing raises the question whether Mr Hoskinson's death would have been avoided if the diagnostic procedures had been undertaken a day previously and the diagnosis made of an aortic dissection. It was the evidence of both Dr Bell and Dr Singh that had the diagnosis been made one day earlier Mr Hoskinson would have required urgent and major surgery but that he had a 60% prospect of surviving and resuming an ordinary life. I accept this evidence. It leads me to conclude that because of those shortcomings in Mr Hoskinson's hospital management which I have set out, the diagnosis of aortic dissection was delayed by approximately 24 hours and that as a result of that delay he was denied that 60% prospect of survival.

Findings Required by s28 of the Coroners Act 1995

In compliance with this s28 (1) I formally find:

a) The identity of the deceased is Neville Robert Hoskinson.

b) The death occurred in the manner and circumstances detailed in these findings.

c) The cause of death was a cardiac tamponade due to a proximal aortic dissection due to hypertension.

d) Death occurred at the LGH in Launceston on 17 May 2012.

The circumstances of the death do not, in my opinion, require me to make any comment or any recommendation as permitted by s28 (2) and (3).

Concluding Comments

I extend my sincere condolences to Mr Hoskinson's wife, family members and loved ones. Particularly, it is hoped that this inquest and the participation in the process by Mrs Hoskinson has been of some assistance in helping her to cope with this tragedy.

Finally I acknowledge the excellent work of counsel-assisting, Mr C N Dockray and coroner's associate, Sgt Hamish Woodgate. Their efforts in preparing and presenting the evidence enabled the hearing to proceed smoothly and efficiently and was of considerable assistance to me.

Dated: 31st day of March 2017 at Hobart in the State of Tasmania.

Rod Chandler
Coroner