MAGISTRATES COURT TASMANIA

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 s57(1)(c) of the Coroners Act 1995)

I, Robert Webster, Coroner, having investigated the death of RI

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

a) The identity of the deceased is RI;

b) RI died in the circumstances set out below;

c) RI's cause of death was community acquired pneumonia; and

d) RI died on 12 August 2022 at Hobart, Tasmania.

In making the above findings I have had regard to the evidence gained in the comprehensive investigation into RI's death. The evidence includes:

- The Tasmania Police Report of Death for the Coroner;
- Tasmanian Health Service (THS) Death Report to Coroner;
- Affidavit as to identity;
- Affidavit of the forensic pathologist Dr Donald Ritchey;
- Affidavit of the forensic scientist Juliette Tria of Forensic Science Service Tasmania;
- Affidavit of JR the daughter of RI and his senior next of kin;
- Medical records of RI obtained from the THS and his general practitioner; and
- Reports of the coronial medical consultant Dr Anthony Bell MD FRACP FCICM.

Background

RI was born in South Africa on 21 April 1954 and he had one sister who still resides in that country. The records of RI's general practitioner suggest his father died of a cerebral haemorrhage at 47 years of age whereas his mother lived until she was 82 years old. RI went to Queens College in Queenstown in South Africa after which he served in the Army for 12 months. He subsequently married KS and they immigrated to Western Australia in 1982 before moving to Tasmania in 1989. RI and KS had three children namely JT, NV and OP. During his working life RI worked as a clerk for South African Railways but he subsequently worked in the insurance industry. He enjoyed playing rugby as a youth and he loved to socialise and be around his friends.

At the date of his death RI was 68 years of age and single his marriage having ended in divorce in or about 2003.

RI's daughter, JT, says they had horses when she was a teenager. An incident occurred in about 1988 when RI was riding a horse and was thrown into a tree. As a result of this accident RI suffered numerous orthopaedic injuries including fractures to his wrists, cheekbone and ribs. A number of surgeries were required at the time and RI remained an inpatient at a hospital in Western Australia for in excess of three weeks. This incident shaped his subsequent years because he was left with limited function of his left arm, chronic pain and recurrent migraines. In 1988 and 1990 part of his ulnar bone was removed however his chronic pain symptoms and migraines continued.

Medical Records

The records of the general practitioner commence on 21 November 1991 and his last visit with his general practitioner was on 18 July 2022. Because of his chronic pain and severe migraines he was a frequent attendee. From the year 2000 RI attended his GP in excess of 360 times and on average he saw his general practitioner on 15 occasions per year.

During the 1990s and into the early 2000s RI's general practitioners made a number of applications to what is now known as the Pharmaceutical Services Branch of the Department of Health for permission to prescribe and/or administer powerful narcotics in order to treat RI's chronic pain. Those applications were in almost all cases granted. There is also evidence of a number of reports being provided to what was then known as the Department of Social Security.

In 1993 RI was referred to the neurologist Dr Yeo who over a number of attendances altered RI's medications. It was thought that a component of his headaches was caused by stress because at about that time RI had been unemployed and was being pressured by the bank in relation to his home loan. RI however managed to obtain alternative manually based employment.

In 1999 RI was referred by his general practitioner to a rheumatologist Dr Francis in order to improve RI's sleep pattern. At that time his job was under threat and he was separated from his wife. Dr Francis' reports reveal at the time of the horse riding accident RI had his own business as an insurance agent which he had to sell because of his injuries. Dr Francis believed RI suffered from post-traumatic stress disorder or a cumulative stress disorder and he implemented a strategy which was aimed at reducing RI's reliance on narcotics on a long-term basis. It seems in addition to all these difficulties RI experienced guilt because of his disability and his inability to work and provide for his family as he had done prior to the accident. There was an admission to the Royal Hobart Hospital (RHH) under the care of Dr Francis between 29 August and 2 September 2000 whereby he received treatment for his chronic migraines. Dr Francis indicated he would provide this treatment again if RI ceased taking narcotics. I have no record of this treatment being provided so the inference is RI did not cease taking narcotics.

Despite seeing and being treated by both Dr Yeo and Dr Francis RI's migraines continued as did his chronic pain. He was prescribed medications for these conditions. In or about the middle of 2002 the records reveal that a bone density study was carried out. That study was carried out to determine whether or not RI had osteoporosis. It is clear that in subsequent years this condition developed and he was prescribed medication to treat it.

In 2010 RI was working because there are some workers compensation certificates on file which certify he suffered from mid thoracic back pain as result of an incident that occurred on 28 January that year when he was moving a fridge. He does not appear to have been certified as totally incapacitated for work. He was certified as fit for modified duties between 4 February and 3 May 2010.

In September 2017 RI underwent chest, rib and thoracic spine x-rays as a result of a fall he had suffered 10 days prior. Compression fractures to the thoracic spine at T5 and T7 were diagnosed as were fractures to 3 ribs on the left-hand side. These fractures were treated conservatively.

There were a number of admissions to the RHH. The first 2 on 13 August and then between the 17 and 19 of August 2012 relate to a condition unrelated to RI's death. He was then admitted between 19 December 2013 and 3 January 2014 because of a burn he had suffered

to his right foot when he fell asleep in front of a heater while intoxicated. The wound did not heal well and he contracted an infection. This required a debridement of the wound and a split thickness skin graft which took place on 27 December 2013.

RI also attended the Emergency Department (ED) at the RHH on a number of occasions. He was treated for a fractured calcaneus in December 2007 and for his foot in December 2013. In relation to the burn to his foot RI also received treatment in the burns, occupational therapy and physiotherapy outpatient clinics. On 24 June 2022 RI was taken to the ED as a result of him fainting at a local hotel he used to frequent. Fortunately people he was with prevented him falling and striking his head. He had experienced a similar episode the day before but did not present to the ED. The history was he was more frail and weak and he had lost weight. The notes record RI declined any imaging and an admission to hospital. He was discharged against medical advice. The notes record a general decline in RI's condition over the last year and he admitted that he was too lazy to look after himself and he acknowledged his alcohol intake was excessive.

Circumstances Leading To Death

RI's final admission to hospital occurred between 9 August 2022 and when he passed away on 12 August 2022. A housemate had called an ambulance because RI had suffered from 2 syncopal episodes and he was cachectic; that is he had lost body weight and mass. He weighed approximately 35 kg. It was thought RI may have suffered an accidental paracetamol overdose due to the fact he was underweight for the amount of medication he was taking. This was said to be secondary to chronic back pain and malnourishment in a setting of multifactorial delirium, alcohol misuse and functional decline. The notes suggest RI consumed alcohol to excess and smoked 30 to 40 cigarettes per day. He had osteoporosis, hypertension and multiple previous spinal fractures due to falls. He also had caecal angiodysplasia and iron deficiency. Over the first two days treatment records suggest RI improved however on 11 August 2022 there was a medical emergency team call because RI deteriorated due to metabolic acidosis and respiratory failure. A decision was made in conjunction with the family to implement palliative care. RI passed away the next day.

Investigation

RI's daughter, JT has expressed concerns about the treatment he received from both his long-term general practice and also from the RHH. She felt he was treated like it was "all too hard" for his general practitioner and hospital staff. She is concerned that mistakes were made by the hospital that may have resulted in his death. Accordingly I arranged for the coronial medical consultant Dr Anthony Bell to examine the records.

(i) THS Death Report

This document suggests the cause of death is decreased GCS[1] and saturations of unknown cause in a setting of accidental paracetamol overdose, malnourishment and functional decline with several falls in the preceding months without investigation. Two issues are raised which included a CT of the brain had been booked but not performed before palliation and therefore it was questioned whether RI had suffered a sub arachnoid haemorrhage due to recent falls. It was also noted RI had decreased saturations despite the fact his chest x-ray was normal. It was questioned whether he had suffered from an aspiration event.

(ii) Post-Mortem and Toxicology

The forensic pathologist Dr Donald Ritchey performed an autopsy on 15 August 2022. In his affidavit he says there is no evidence of any haemorrhage or aspiration and so these possibilities as to the cause of death raised in the THS Death Report can be ruled out. The fact the CT scan of the brain was not performed had no bearing on RI's outcome. An important finding by Dr Ritchey is an oblique surgical scar which he observed on RI's left upper abdomen which suggests at some stage RI had undergone a splenectomy[2]. This is referred to below.

Dr Ritchey goes on to say, after conducting his examination, considering the medical records and the results of histology and toxicology, the cause of death in this case was community acquired pneumonia in a setting of advanced centriacinar emphysema, alcoholism and asplenia which is the absence of normal spleen function. He says significant contributing factors were the paracetamol overdose, and ketoacidosis which was caused by excessive alcohol consumption and starvation. The pneumonia was evidenced by widespread consolidation of the left lung with grey hepatisation and the results of histology were florid acute and organising pneumonia. In addition post mortem lung cultures grew moderate Streptococcus. Toxicology results for paracetamol which were reported upon by Ms Tria confirm it was detected at greater than therapeutic levels. I accept the opinions of both Dr Ritchey and Ms Tria.

(iii) Dr Bell's Reports

Dr Bell noted the following relevant history:

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[1] The GCS or the Glasgow Coma Scale is used to objectively describe the extent of impaired consciousness in all types of acute medical and trauma patients. The scale assesses patients according to three aspects of responsiveness: eye-opening, motor, and verbal responses.

[2] That is had his spleen removed.

- Smoked 30 to 40 cigarettes daily
- Alcohol 8 standard drinks per day, alcohol misuse disorder
- 1988: Fell off horse, multiple vertebral fractures, probably had a splenectomy at this time, no records known
- Osteoporosis
- 1998: Panadeine forte (paracetamol 500 mg, codeine 30 mg) continuously prescribed since this time, for chronic back pain which had been endured for 30 years but which has worsen after a fall 6 weeks prior, multiple crush fractures in mid-thoracic spine with significant progression since 2010, exaggerated kyphosis at T9 and T10
- 2010: CT scan of the abdomen showed an irregular small spleen, suggested haematological review
- 17.08.2012: Full blood examination showed anaemia, the red blood cells were assessed by microscope which showed dimorphic red blood cells with a hypochromic microcytic and a normochromic normocytic population. Also present were target cells, Howell Jolly bodies with occasional acanthocytes, a pattern which is consistent with spleen dysfunction and liver damage
- 2012: burn foot, became infected with gram positive cocci
- Four month history of unusual behaviour
- Three week history weight loss, BMI 14, weight 32.5 kg and blurred vision
- Needs assistance with activities of daily living

The past medication history of significance was:

Panadeine forte daily for years, dose 8 to 10 daily (4 to 5 g paracetamol daily)

Olmesartan, Amlodipine

The current medical history was:

On 09.08.2022 at 12.30pm Ambulance Tasmania was called to RI's residence. He was confused with a GCS of 14/15. Vital signs were an un-recordable blood pressure, heart rate 113 bpm, respiratory rate 14 bpm and a cool periphery, oxygen saturation was unobtainable due to poor peripheral perfusion. During the journey to the RHH RI's blood pressure was recorded at 109/72.

At the ED of the RHH there was a history of decreased oral intake due to nausea and difficulty swallowing. RI said he was too lazy to care for himself, and acknowledged excessive alcohol use. He was able to follow conversation and retain information but he was disorientated to time and place. Systematic questions revealed no additional symptoms. RI was cachectic, blood pressure 130/80 mmHg, heart rate 101 bpm with normal heart examination. The respiratory rate was 16 bpm, and oxygen saturation 95% on ambient air, anterior chest auscultation was normal. His temperature was 36.5 degrees Celsius. There was midline spinal tenderness at T8.

Dr Bell goes on to say *"[a]n electrocardiogram showed sinus tachycardia, marginal P pulmonale, and borderline right sided electrical axis deviation 87 degrees. The biochemistry showed chronic kidney damage with a calculated glomerular filtration of 30 ml/min (normal greater than 90 ml/min), although the serum creatinine was within the normal range. Liver function tests showed an elevated AST but normal ALT, with elevated GGT and normal bilirubin. The albumin was 34 g/L (normal 35 to 52 g/L). There was anaemia with normal sized red blood cells but a wide red cell volume distribution suggestive of combined iron deficiency anaemia (was iron deficient) with macrocytic anaemia, folate and B12 levels were normal, thus probably an alcohol effect on the size of the red blood cells. The white cell count and neutrophil counts were elevated. The C reactive protein was 18 mg/L, not suggestive of bacterial infection. Coagulation profile was normal. The liver synthetic function appeared reasonable. A paracetamol level was 218 micromol/L (blood sample dated 09.08.2022 at 08.15am, toxic range 1300 micromol/L 4 hours after ingestion)."*

RI was admitted to the hospital under the care of a general medical team. The management plan was to treat the paracetamol overdose with N-acetyl cysteine intravenously, paracetamol was ceased. Then RI's hydration would be corrected and the dysphagia[3] investigated. Dr Bell says RI may well have had Plummer Vinson syndrome[4] due to chronic iron deficiency which might have been causing the dysphagia.

On 10.08.2022 RI was reviewed, and he developed fluent confabulation[5] and confusion.

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[3] Difficulties swallowing.
[4] A disorder marked by anaemia caused by iron deficiency, and a web-like growth of membranes in the throat that makes swallowing difficult.
[5] A problem that makes someone produce false memories about events. This condition differs from lying in that the person is not consciously attempting to deceive.

On 11.08.2022 at 09.20am cognition was improving but there was still episodic confusion. RI was noted to be picking at his sheets and bed. At 4.20pm a medical emergency team was called due to oxygen saturation falling to 70%. The oxygen saturation improved to 88% on high flow oxygen. RI's conscious state had decreased. Arterial blood gases showed oxygen of 60 mmHg, carbon dioxide 49mmHg, pH 7.20 and bicarbonate 19 mmol/L consistent with a metabolic acidosis with failure of respiratory compensation that is respiratory failure. A chest x-ray showed no consolidation. There was a normal glucose level with elevated ketone levels indicating a ketoacidosis. The blood lactate was normal. Insulin glucose infusion was commenced. An antibiotic was commenced for a possible chest infection. After a family discussion RI was commenced on palliative comfort care.

Dr Bell noted Dr Ritchey's opinion and highlighted the following from Dr Ritchey's affidavit:

"Liver [histology showed] *there is widespread but patchy central zone lobular necrosis and congestion characterised by necrotic hepatocytes and central zone congestion."*

"Splenic remnant; is predominantly composed of blood vessels with only weak splenic type follicle formation and fibrosis."

Dr Bell says it follows RI had minimal if any functional splenic tissue. Dr Bell says Dr Ritchey's opinion as to the cause of death is correct however it was the lack of functional splenic tissue, chronic liver disease and malnutrition that were the drivers of the infection which caused RI's death.

Dr Bell says *"an absent or reduced splenic function can be due to anatomic absence of the spleen due to surgical removal. Absent or reduced splenic function may be suspected in individuals with conditions known to affect the spleen or if characteristic red blood cell (RBC) findings such as Howell-Jolly bodies, RBC pits, or nucleated RBCs (NRBCs) are on the blood smear or acanthocytes. However, as noted above, these findings are relatively nonspecific. The spleen is usually removed by an oblique upper left sided abdominal incision*[6]*.*

In this patient's medical record there is no reference to the oblique upper left sided abdominal scar noted at post mortem examination. With the history of trauma with multiple fractures (from a fall from a horse) this scar should have raised consideration of splenectomy. In 2010 and 2012 CT scans of the abdomen showed a small irregular spleen. The only blood film to assess the red blood cells is recorded above in past history (17.08.2012). This film was consistent with asplenia but is relatively non-specific and may represent liver dysfunction.

[6] Which was observed by Dr Ritchey at post mortem.

Patients with impaired splenic function are at risk for severe and overwhelming infections with encapsulated bacteria (Streptococcus pneumoniae), blood borne parasites, and other infections that the spleen plays an important role in controlling. Key measures for preventing such infections include patient and caregiver education, vaccination against encapsulated bacteria, influenza, coronavirus disease 2019 (COVID-19), and use of prophylactic antibiotics."[7]

Dr Bell says the GP records make no mention of RI saying he had undergone a splenectomy. He did not give a medical history to the RHH that he had undergone a splenectomy. There is therefore no record of appropriate management being provided to a patient who had undergone a splenectomy

Since its clinical introduction in 1955, Dr Bell says paracetamol (N-acetyl-p-aminophenol; APAP; paracetamol) has become the most widely used analgesic-antipyretic in the United States. Paracetamol is a component of hundreds of over-the-counter and prescription medications used worldwide. He goes onto say the following:

"Although the drug is considered safe when taken at usual therapeutic doses (up to 4000 mg every 24 hours), an overdose of paracetamol has been recognized since 1966 to cause fatal and nonfatal hepatic necrosis. It is suspected that even repeated therapeutic or slightly excessive doses can be hepatotoxic in susceptible individuals, such as people with alcohol use disorders. Patients are also at risk for hepatotoxicity when taking prescription products that combine paracetamol with opioids.

Patients may accidentally develop clinically significant poisoning from repeated ingestions of supra-therapeutic doses of paracetamol in an attempt to relieve pain or treat fever. These patients are more likely to have established risk factors for hepatotoxicity (fasting, chronic ethanol use) and are more likely to present to medical care late, when the toxic effects of paracetamol are already established. Unlike acute paracetamol poisoning, acute alcohol-related hepatitis and chronic paracetamol poisoning in the alcohol user have an aspartate aminotransferase (AST) to alanine aminotransferase (ALT) ratio greater than two."

In this case Dr Bell says the paracetamol toxicity was recognized although the chronic nature does not appear to have been understood. RI had the typical liver enzyme elevation reversal of chronic toxicity. The administration at the RHH of N-acetyl cysteine was, in Dr Bell's opinion, sound practice in particular due to the safety of the drug. The blood level in acute toxicity was below the level for acute liver necrosis and in the acute situation would not be given.

[7] A patient with chronic liver injury or disease is also prone to the same types of infection. Patients with severe malnutrition are also prone to infection.

RI had alcohol misuse syndrome for many years which contributed to his liver dysfunction. Dr Bell also notes the medical records suggest RI was, at times, reluctant to appropriately engage with the medical profession.

Dr Bell says:

"As is typical of this type of case the characteristic signs and symptoms of infection are absent. White cell elevation was mild and distracting from a diagnosis of sepsis. The low C reactive protein (CRP) in a normal person is strong evidence against bacterial infection. CRP is liver produced and production may be diminished. Low-grade infections also diminish CRP elevations. Due to these difficulties and other symptoms present namely dysphagia, chronic renal function, malnutrition and paracetamol toxicity the diagnosis of infection was not considered."

Because of RI's poor health Dr Bell estimates RI had a mortality rate of 70 to 80% no matter what treatment was attempted. He concludes by saying RI presented with major multiple medical issues that were unable to be solved. He considers the standard of medical care at the RHH was reasonable. Dr Bell does not raise any issues or concerns with the medical treatment provided by the general practitioners who treated RI over the years.

I accept the opinions of Dr Bell.

Comments and Recommendations

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

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

Dated: 20 February 2023 at Hobart in the State of Tasmania.

Robert Webster
Coroner