Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Patricia Joy Russell

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

a) The identity of the deceased is Patricia Joy Russell;

b) Mrs Russell was born on 4 March 1942 and was aged 74 years;

c) Mrs Russell died on 19 January 2017 at the Launceston General Hospital (LGH) in Launceston; and

d) The cause of Mrs Russell’s death was empyema/sepsis complicating oesophageal perforation following a complicated elective hernia repair. Significant contributing factors were Type II diabetes, obesity (BMI 38.1), emphysema and atherosclerotic coronary vascular disease.

Background

Mrs Russell was married for 53 years to Terrence Roy Arthur Russell. They resided at Egg Lagoon on King Island.

Mrs Russell’s past medical history included diabetes mellitus, hypertension, chronic obstructive pulmonary disease with asthma and renal impairment. She was an ex-smoker and weighed 118kg.

In late 2016 Mrs Russell was diagnosed with a large umbilical hernia. She had ongoing discussions with her general practitioner concerning the symptoms before making the decision to have it surgically repaired. Arrangements were then made for the procedure to be undertaken by surgeon Mr Trevor Leese at the North West Regional Hospital (NWRH) in Burnie on 4 November 2016. However, in the days immediately prior to the surgery Mrs Russell suffered a lower respiratory tract infection. She was reviewed by her general practitioner on 2 and 3 November and was assessed to be sufficiently well to travel to Burnie and to have the surgery.
Circumstances Surrounding the Death

On 4 November 2016 Mrs Russell underwent her surgery. The hernia was located and repaired. However, during the operation a cancerous tumour was unexpectedly found in Mrs Russell’s transverse colon. It was surgically removed. Mrs Russell was then admitted to the Critical Care Department (CCD) and her condition remained stable overnight. The following day Mrs Russell was able to mobilise and was clinically stable. She was transferred to a ward at around 10.30am.

In the morning of 6 November Mrs Russell was seen by an anaesthetist clinician. Later she was reviewed by Mr Leese. Her vital signs were satisfactory. However, a MET call was made at 8.00am because of low oxygen saturations and shortness of breath. She was given a nebuliser and placed on an ‘Airvo’ oxygen delivery system. Later a chest x-ray showed right sided atelectasis but otherwise the lungs appeared normally aerated. Her renal function declined. At around 10.25am she was transferred back to CCD. Overnight she deteriorated further. Her creatinine level was 334 micromoles/l and the albumen was 19g/l.

During a ward round at about 10.00am the next morning, Mrs Russell was noted to be drowsy and complaining of shoulder tip pain on the left side. A chest x-ray done at about 10.50am reported a “Left sided pneumothorax.” Later an intercostal catheter was inserted and 700ml of brown fluid was drained from the pleural cavity over a 7 hour period. A pathology analysis reported “…Brown fluid…Albumin 3 g/l…Lactate Dehydrogenase 969 U/L…pH-insufficient specimen.” A cytology analysis of the brown fluid reported: “Negative. There is no cytological evidence of malignancy.”

During 8 November Mrs Russell continued to require circulatory support. Nursing notes record that thick blood stained secretions were suctioned from her endotracheal tube. Light brown fluid was also draining from the intercostal catheter. It was noted that Mrs Russell was not tolerating naso-gastric feeds and these were ceased that evening. A measurement of the pleural fluid showed a pH of 6.98.

In the morning of 9 November Mrs Russell had a CT scan of her chest, abdomen and pelvis. It showed no evidence of an oesophageal pleural fistula. However, there was evidence of fluid collections in the abdomen suggesting an anastomatic dehiscence or breakdown. A methylene blue swallow was considered but not proceeded with. In the afternoon Mrs Russell underwent an exploratory laparoscopy which confirmed a faecal leak at the site of the previous transverse colon surgery. It was repaired. The surgeon reports that faecal contamination appeared limited to the central part of the abdomen. In the days following Mrs Russell remained seriously unwell. She remained in CCD. A nursing note made on 10 November records that during an infusion of calcium via a peripheral intravenous catheter Mrs Russell’s left arm became discoloured, reddened, blistered and began to peel. The infusion was ceased and the arm dressed.

On 13 November some methylene blue was added to Mrs Russell’s naso-gastro feeds. The methylene blue was then noted to come out of the intercostal catheter which led to a diagnosis of a perforated oesophagus. Mrs Russell’s condition continued to worsen. Input
was sought from intensivists at the Royal Hobart Hospital (RHH) and the decision was then made for Mrs Russell to be transferred to Hobart. She arrived at the RHH at 11.45pm on 14 November and was directly admitted to the Intensive Care Unit (ICU). Mrs Russell remained at the RHH until 19 January when she was transferred to the LGH to enable her to be closer to family. At both hospitals attempts were made to manage Mrs Russell’s ongoing sepsis, renal failure and declining cardiovascular and respiratory condition. However, Mrs Russell continued to deteriorate and on 19 January, after consultation with family members, the decision was taken to cease active treatment. Mrs Russell died at about 6.50pm.

**Post-Mortem Report**

This was carried out by Dr Donald Ritchey. In his opinion the cause of Mrs Russell’s death was empyema/sepsis complicating oesophageal perforation following a complicated elective hernia repair. Significant contributing factors were Type II diabetes, obesity (BMI 38.1), emphysema and atherosclerotic coronary vascular disease.

In his report Dr Ritchey includes this helpful comment: “Oesophageal perforation is always a serious complication. When perforation occurs in the distal oesophagus (as in the present case) there is high morbidity and mortality associated with leakage of oesophageal and gastric contents into the peritoneum and pleural spaces. The resulting sepsis is difficult to control and is frequently fatal.”

**Investigation**

This has been informed by:

2. An affidavit provided by Mrs Russell’s husband.
3. A review of Mrs Russell’s hospital records carried out by research nurse, Ms L K Newman.
4. A report provided by Dr A J Bell, as medical adviser to the coroner.
5. A report provided by Associate Professor Robert Pegram of Tasmanian Health Service-North West.

In his report Dr Bell expresses these opinions:

- That Mrs Russell was sufficiently recovered from her respiratory infection to undergo her hernia surgery on 4 November 2016.
- That it was appropriate for Mr Leese to proceed to remove the tumour when it was unexpectedly detected.
- That a central vein should be used for calcium infusion and that it was poor medical practice to use a peripheral vein. This error caused significant damage to Mrs Russell’s left arm which should have been avoided.
- That Mrs Russell’s oesophageal perforation was in all likelihood unrelated to the insertion of a naso-gastric tube. Perforation by a naso-gastric tube is extremely rare. Further, the usual site of such a perforation would be the upper oesophagus.
However, Mrs Russell’s perforation was sited in the lower portion which is consistent with a rupture by effort.

- Almost certainly Mrs Russell’s oesophageal perforation was spontaneous and was related to effort rupture possibly when attempting to vomit. It probably occurred at an unknown time on 6 or 7 November 2016, that is one week before it was actually diagnosed.
- A chest x-ray in the morning of 6 November did not indicate a pneumothorax. However, that night her condition deteriorated with signs of progressive sepsis. She complained of difficulty breathing. A chest x-ray the following day showed a left sided pneumothorax and a small left sided pleural effusion. This history is consistent with Mrs Russell suffering her oesophageal perforation sometime between the two x-rays.
- The insertion of the intercostal catheter on 7 November should have led to the diagnosis of oesophageal perforation because the brown coloured liquid which was extracted was strongly suggestive of gastric content, a circumstance most consistent with this diagnosis. The pH test result measured the following day was confirmatory proof of the diagnosis.
- That effort rupture of the oesophagus or Boerhaave syndrome is associated with high morbidity and mortality. It is fatal if untreated. Any delay in commencing treatment increases the likelihood of a poor outcome. In Mrs Russell’s case the delay in initiating treatment after the likely onset of the condition increased the mortality rate to over 90%.
- That the treatment provided to Mrs Russell by both the RHH and the LGH was appropriate and of a good standard.

Findings, Comments and Recommendations

When Mrs Russell was admitted to hospital for hernia surgery on 4 November 2016, neither she nor her family could have envisaged the horror scenario which followed. Her difficulties began with the unexpected presence of a tumour mass in her transverse colon. Quite properly this was surgically removed. Unfortunately, this set in train a cascading series of events which eventually led to death. First, the evidence shows that Mrs Russell developed a faecal leak due to a breakdown at the site of her colon surgery. (It must be acknowledged that Mrs Russell’s pre-existing health issues made her more vulnerable to that development and impeded her recovery from it.) This condition required treatment including surgical repair. Contemporaneous to this complication was the unrelated and spontaneous development of an oesophageal perforation. It is the opinion of Dr Bell, which I accept, that this perforation in all likelihood occurred sometime on 6 or 7 November. I also accept that this is a particularly serious condition which requires immediate treatment to maximise the prospect of survival. However, the diagnosis was not made until 13 November following a methylene blue swallow and I accept that this interval very significantly reduced Mrs Russell’s prospects of survival.

I acknowledge that perforation of the oesophagus is not a common condition and in all likelihood is rarely seen in a regional hospital such as the NWRH. Nevertheless, I accept, for the reasons explained by Dr Bell, that the diagnosis of a perforated oesophagus should have been made by 7 November. (I need to record that the NWRH has since accepted that its
diagnosis of this condition was delayed). It must be acknowledged that a perforated oesophagus is a particularly serious ailment which often has fatal consequences even when promptly diagnosed and optimal treatment commenced. This leaves me to find that if a diagnosis of a perforated oesophagus had been made at the first reasonable opportunity then Mrs Russell’s survival could not have been guaranteed. Rather, I can only find that a prompt diagnosis would have increased her prospects of survival.

I note and accept Dr Bell’s criticism of the NWRH with respect to the calcium infusion. It has been accepted by the NWRH which I am informed has, as a result of Mrs Russell’s experience, adopted an alternate means of administering calcium.

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 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 Russell’s family and loved ones.

Dated: 26 day of March 2018 at Hobart in the State of Tasmania.

Rod Chandler
Coroner