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 William Suckling

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

a) The identity of the deceased is James William Suckling;
b) Mr Suckling was born in Devonport on 6 June 1944 and was aged 72 years;
c) Mr Suckling died on 23 November 2016 at the North West Regional Hospital (NWRH); and
d) The cause of Mr Suckling’s death was hypovolemic shock due to haemoperitoneum following surgical repair of an abdominal wall hernia.

Background
Mr Suckling was a retired manager who resided with his wife Barbara Suckling at Melrose. His medical history included hypertrophic cardiomyophy (HOCM), aortic stenosis and hypertension.

Circumstances Surrounding the Death
On 22 November 2016 Mr Suckling was admitted to the North West Private Hospital (NWPH) for exploratory surgery of a mass in the left iliac fossa region of his abdomen. The surgeon was Mr James Roberts-Thomson. The mass was found to be a Spigelian hernia which is a defect in the interior lateral abdominal wall through which abdominal contents may protrude and become entrapped. This was successfully repaired. Postoperatively his condition was stable and he was returned to a ward with the expectation that he would be discharged home the following day. However, that evening his condition deteriorated. He began sweating excessively, was hypotensive, bradycardic (slow heart rate) and pale. At 10.13pm a Medical Emergency Team (MET) call was made and Mr Suckling was transferred next door to the Emergency Department (ED) of the NWRH.

On arrival Mr Suckling was alert, oriented and without pain. However, he was pale and sweating. Blood pressure was 74/75 mmHg, heart rate was 57bpm, respiratory rate was 15 bpm and oxygen saturation level was 98% on 2 litre oxygen supplementation. He was afebrile. It was noted by Consultant, Dr Michael King, that Mr Suckling’s ECG
taken earlier at the NWPH showed ST elevation in leads V1 and V2 with ST depression and T wave inversion in the infero-lateral leads. This was interpreted to be consistent with an ST elevation myocardial infarction and the decision was taken to contact the cardiology team at the Launceston General Hospital (LGH). A plan was then made to transfer Mr Suckling to the LGH but its implementation was delayed because of the "competing priorities of the retrieval team." In the meantime, Mr Suckling was given intravenous fluids and increasing amounts of metaraminol to support his blood pressure. Aspirin, clopidogrel and clexane were also administered.

At 1.26am on 23 November Mr Suckling’s blood pressure was less than 80 mmHg systolic. It was noted to fall on sitting up and to improve when lying down. At this time he vomited. At 1.30am further blood was taken for a high sensitivity troponin level. The result was normal. At about this time Dr King left the ED. His handover instructions to ED registrar, Dr Katherine Snow, were: “will keep bolusing (sic) saline to max of 3 L and increase Aramine infusion to 10 mg/hr to keep MAP ideally 65-70. If still not controlling BP then call cardiology for further advice.”

It was noted at 2.55am that Mr Suckling was hypotensive with a mean arterial pressure (MAP) of ~40. He was reviewed by Dr Snow and the metariminol infusion was increased. It was recorded that Mr Suckling was restless and complaining of chest tightness. His condition continued to deteriorate. Further advice was sought from the LGH after which a low dose adrenaline infusion was commenced. MAP continued to be consistently ~40. At 3.55am Dr King was contacted and asked to return to the hospital. He had arrived by 4.22am. By this time Mr Suckling was unresponsive, and required airway suctioning and airway support. His MAP was ~30. He was intubated. His condition continued to deteriorate despite active treatment. Mr Suckling died at 4.40am.

A note subsequently entered into the record by Dr King includes this comment: “Pre-mortem FAST scan showed free fluid in the abdomen. I note fall in haemoglobin on serial blood gas samples from 141 at 22:30 to 87 at 03:50, consistent with bleeding, likely intra-peritoneal. Impression: likely post-operative myocardial infarction progressing to cardiogenic shock; intra-peritoneal bleeding exacerbated by Enoxparin and anti-platelet agents administered for treatment of MI.”

Post-Mortem Examination
This was carried out by forensic pathologist, Dr Donald Ritchey. His report includes this comment: “The autopsy revealed a well developed, obese (obesity defined as a body mass index of greater than or equal to 30 kg/m²) elderly Caucasian man with approximately 1.5 litres partially clotted blood within the anterior peritoneum (haemoperitoneum). The source of the bleeding was the surgical site of the anterior abdominal hernia repair although sutures and mesh appeared secure and a focal source of bleeding within the surgical site could not be established at autopsy.”

In Dr Ritchey’s opinion the cause of Mr Suckling’s death was hypovolemic shock due to haemoperitoneum following surgical repair of an abdominal wall hernia. I accept this opinion.
Investigation

This was informed by:

2. An affidavit from Mrs Suckling.
3. A precis of Mr Suckling’s hospital records.
4. A report from Associate Professor (Adjunct) Robert Pegram of Tasmanian Health Service.
5. A report prepared by Dr A J Bell as medical adviser to the coroner.

In his helpful report Associate Professor Pegram advises that:

• He sought feedback from the NWRH clinicians involved in Mr Suckling’s care before compiling his report.
• THS accepts the cause of death as articulated by Dr Ritchey.
• It was communicated to the NWRH’s ED at the time of Mr Suckling’s transfer that his surgery had involved a simple inguinal hernia repair. This was incorrect as Mr Suckling’s operation was complex involving exploration of an inguinal mass. Further Mr Suckling’s clinical notes were not provided by the NWPH at the time of his transfer.
• The fact that Mr Suckling had significant aortic stenosis was not conveyed to the ED at any time and this created a misdirection on the assessment of his cardiovascular status.
• It is now acknowledged that a surgical review should have been provided for Mr Suckling in the ED given his earlier operation.
• The NWRH now requires patients being admitted from a private hospital to be accompanied by their clinical notes.
• This case has an important clinical education message for medical staff, namely: “Post op Patients with unrecognised significant haemorrhage can easily present as circulatory compromise due to haemorrhagic shock as this often produces ECG changes that can include ST depression. This may be interpreted as an acute coronary syndrome or as a ‘silent AMI’ post op rather than looking for a hypovolaemic cause. In this case it was compounded by the fact that the operation was thought to be simple (i.e. inguinal hernia repair) and one where you would not expect significant bleeding risk. Surgeons should be educated on hypovolaemia causing ST changes and that if they are called by someone and they are told that the patient has had a coronary event they should ask if the patient has been assessed for haemorrhage.”
• THS carried out its own Root Cause Analysis of the case which identified some system issues which if corrected would improve patient care. It led to these recommendations being made:
  ➢ “In the post operative patient always consider bleeding, even in the absence of tachycardia or in the presence of an ECG that suggests MI.
  ➢ All clinical handovers of deteriorating patients from NWPH to NWRH must include consultant to consultant clinical handover. All relevant notes
must accompany patient, including a summary of suspected complications.

- NWPH to review processes around referrals for anaesthetic assessment in the preadmission clinic for general surgical patients. These referrals can be initiated by the surgeon, or by a formal nurse assessment process, where the nurse has access to the full referral from the GP, including a full past medical history.”

In his report Dr Bell makes these observations:

- That at around 10.00pm on 22 November Mr Suckling suffered an acute haemorrhage. His underlying HOCM and aortic stenosis exposed him to the likelihood of rapid haemodynamic collapse. At this time his treatment should have focussed on re-establishing the blood volume and using medication (vasoconstrictors) to narrow the blood vessels thereby increasing arterial blood pressure.
- Clinicians at the NWRH wrongly diagnosed cardiogenic shock. The subsequent use of anticoagulation was inappropriate. The diagnosis also inhibited the administration of the required level of fluids.
- Standard treatment for a post-operative haemorrhage often involves surgery to tie off the bleeding vessel along with blood replacement and the maintenance of adequate fluid for circulatory stability. Most patients, if promptly diagnosed and appropriately treated, are likely to make a full recovery.
- The diagnosis of Mr Suckling’s condition was made more difficult in the ED because of the non-provision of his clinical record at the NWPH, the failure to arrange a surgical review and the ignorance of Mr Suckling’s pre-existing cardiac health.
- During the course of Mr Suckling’s time in the ED there was mounting evidence that the diagnosis of cardiogenic shock was wrong. Notably the ECGs recorded in the ED did not show sufficient changes to account for cardiogenic shock. Further, a high sensitivity troponin level test was normal indicating no myocardial damage. Despite this evidence the diagnosis of cardiogenic shock was not seriously reconsidered.
- The conclusions reached following the Root Cause Analysis and its recommendations were valid.

Findings, Comments and Recommendations

It is clear from the evidence that in the late evening of 22 November 2016 Mr Suckling suffered an acute haemorrhage related to his hernia surgery earlier in the day. This development was not recognised at the NWPH before his transfer to the NWRH. At the NWRH Mr Suckling was diagnosed with cardiogenic shock. This diagnosis was wrong and led to treatment which was inappropriate for Mr Suckling’s actual condition, namely a postoperative haemorrhage. I accept Dr Bell’s opinion that during the course of Mr Suckling’s time at the NWRH evidence accumulated which should have caused a
reconsideration of Mr Suckling’s diagnosis. Regrettably, this did not occur with the result that Mr Suckling continued to deteriorate and then died.

I further accept Dr Bell’s opinion that persons suffering from a postoperative haemorrhage are likely to make a full recovery if the condition is promptly diagnosed and appropriately treated. Sadly this did not occur for Mr Suckling.

I acknowledge that the NWRH has conducted its own Root Cause Analysis into the circumstances of Mr Suckling’s death and I accept its conclusions and support its recommendations. It is unnecessary for me to make any further recommendations.

I have decided not to hold a public inquest into this death because my investigation has been sufficient to disclose the identity of the deceased, the date, place and cause of death, relevant circumstances concerning how death occurred and the particulars needed to register the 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.

I convey my sincere condolences to Mr Suckling’s family and loved ones.

Dated: 5th day of December 2018 at Hobart in the State of Tasmania.

Rod Chandler
Coroner