Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Rod Chandler, Coroner, having investigated the death of Stephen Guy Bruinger

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

a) The identity of the deceased is Stephen Guy Bruinger;

b) Mr Bruinger was born in Hobart on 14 July 1954 and was aged 62 years;

c) Mr Bruinger died on 16 January 2017 at the Royal Hobart Hospital in Hobart (RHH);

and

d) The cause of Mr Bruinger’s death was a cerebral haemorrhage.

Background

Mr Bruinger was married to Debra. They had three children, Rebecca, Rachael and Shianna. Rachael suffers from Down Syndrome and resided with her parents. Rebecca and Shianna lived independently. Mr Bruinger had worked as a painter and a brewery line manager. However, he injured his back in two separate work incidents and had not been employed for the last 20 years. He received a disability pension.

Mr Bruinger’s medical history includes chronic back pain, dyslipidaemia (elevated cholesterol or fats), microalbuminuria (increased level of albumin urine), hypertension and gastro-oesophageal reflux.

Circumstances Surrounding the Death

In her affidavit Mrs Bruinger describes the events of the early morning of 15 January 2017 in these terms: “Stephen got out of bed to go to the toilet and he started complaining about the cold. Whish (sic) was unusual for Stephen. Stephen went back to bed and started saying that he couldn’t breath (sic), and became very aggressive towards me. I called the ambulance and upon their arrival Stephen was very abusive (which is not like him)“.

Ambulance Tasmania records show that an ambulance arrived at the Bruinger home at 5.16am. It was noted that on arrival: “patient rolling around on the floor, sweaty RR 36, c/o SOB, sats 90%. Px obstructive at first but became more compliant as time passed”. The Initial Assessment form records: “acute pulmonary oedema; chucked a wicked hissy fit as wife suggested”. Mr Bruinger was transported to the Emergency Department (ED) of RHH arriving at 6.18am.
Mr Bruinger had a chest x-ray at 6.53am. It was reported: “No pulmonary oedema is seen”. He was assessed by nursing staff at 8.59am who recorded: “62 yo man TAS, post? Panic attack and episode of a cold shiver and shortness of breath. Patient stated only happened once before…Well perfused, alert and oriented…other vitals within acceptable parameters”. A CT pulmonary angiogram showed no evidence of a pulmonary embolism.

Later Mr Bruinger was seen by emergency registrar, Dr Elizabeth du Bois. Her notes include: “Thought was having panic attack. Was diaphoretic but denies chest pain or dizziness”. The electrocardiogram was considered to show an atrial fibrillation rate of 130bpm with no ischaemic changes. Her provisional diagnosis was new onset atrial fibrillation with the possibility of acute coronary syndrome or pulmonary embolus as the underlying cause. After blood tests showed an elevated troponin of 196, Dr du Bois discussed Mr Bruinger’s case with an emergency consultant and then made a referral to cardiology.

At 12.45pm Mr Bruinger was reviewed by a cardiology registrar. He considered that Mr Bruinger presented with “chest discomfort and shortness of breath”. A plan was made to treat Mr Bruinger for a non-ST segment elevation myocardial infarct (NSTEMI). Standard treatment was commenced including an anti-platelet agent ticagrelor, aspirin, beta-blocker and therapeutic clexane. Mr Bruinger was then formally admitted as a cardiology patient but remained in the ED.

At 2.20pm Mr Bruinger suddenly began sweating profusely and experiencing shortness of breath. He was very pale, hypertensive and tachycardic. He was moved to the ED’s resuscitation area for closer monitoring. Over the following two hours Mr Bruinger rapidly deteriorated. By 3.50pm clear neurological signs were presenting. At 4.23pm he had a CT scan of the brain. It demonstrated a large lobar cerebral haemorrhage within the left cerebral hemisphere. There was surrounding oedema and significant mass effect. Mr Bruinger was then reviewed by a neurosurgery team. It was decided that surgery was not feasible and he was transferred to the Intensive Care Unit for supportive care. He died at 4.08pm on 16 January 2017.

Report of Death

Mr Bruinger’s death was not reported to the coroner until 24 January 2017. The report was made by the Acting Manager of Strategy and Projects at the Department of Health and Human Services and was prompted by its receipt of an internal report made by Dr Emma Huckerby, the Director of the ED. Her report states: “Patient attended ED by ambulance Sunday, Jan 15 2016 at 0618. Triage notes on AT arrival at his home he was diaphoretic, tachpnoeic, SaO290% RA, SBP > 200, PR120 AF. Vital signs had normalised on arrival in ED. This patient should have been allocated a monitored cubicle in the main ED to observe his vital signs continuously over time in order to establish the pattern of his episodes of vital sign abnormalities which included episodes of severe hypertension. Due to extreme ED overcrowding there was no capacity for this to occur for more than 8h – including after he was discovered to have a NSTEMI. Patient experienced an intracerebral bleed, was admitted to ICU and has subsequently died. Hourly audits of the ED capacity over this time period show that at all times there was ambulance ramping; no availability of acute ED cubicles and for 7 hours there was access to 0 or 1 resuscitation cubicles.”
Before Mr Bruinger’s death had been reported to the coroner his body had been released to his family for burial or cremation. As a result the coronial investigation has not been aided by a post-mortem examination.

Investigation

This has been informed by:

1. An affidavit sworn by Mrs Bruinger.
2. Consideration of Mr Bruinger’s records at Knopwood Medical Centre.
3. Review of Mr Bruinger’s records at the RHH undertaken by research nurse, Ms L K Newman.
4. A report provided by Dr du Bois.
5. A report provided by Cardiologist, Jonathan Lipton.
6. A report written by Dr A J Bell as medical adviser to the coroner.

Dr Bell has expressed these opinions in his report:

a) In the context of the initial presenting features a diagnosis of neither a NSTEMI nor a cerebral haemorrhage was not possible.

b) The reporting of chest discomfort by the cardiology registrar appears to have been a factor leading to the diagnosis of NSTEMI when the overall evidence suggests that any chest pain or discomfort was minor, if present at all. In this regard it is noted that a complaint of chest pain or tightness was not recorded by the ambulance officers or nursing staff at the initial assessment. Also, it was noted by ED medical staff that Mr Bruinger denied chest discomfort or pain and that he had good exercise tolerance.

c) That rather than settle on a decision that Mr Bruinger’s presentation was cardiac caused his better management required close monitoring and a fully considered assessment of all the signs of symptoms, including those which contraindicated NSTEMI. Such monitoring and assessment were made particularly difficult by the environment existing in the ED at the time and described by Dr Huckerby in her report.

d) The diagnosis of NSTEMI led to the commencement of antiplatelet anticoagulation therapy. For a patient with a cerebral bleed already present this treatment likely encouraged further bleeding.

e) That even without the anticoagulation therapy there was a significant chance of a further cerebral bleed leading to the same outcome.

f) That it was not until the mid to late afternoon that sufficient clear cut neurological signs presented to enable the diagnosis of a cerebral haemorrhage to be considered.

Findings, Comments and Recommendations

Although there was not a post-mortem examination I can nevertheless be satisfied upon the evidence that the cause of Mr Bruinger’s death was a cerebral intracranial haemorrhage.

It is evident, in retrospect, that the catalyst for Mr Bruinger’s presentation to the ED in the early morning of 15 January 2017 was a small cerebral haemorrhage. Unfortunately a diagnosis of this condition was not made at the initial presentation and instead Mr Bruinger
was mis-diagnosed with a NSTEMI. This diagnosis was maintained until late in the afternoon when clear neurological signs began to present and a CT scan enabled the diagnosis of a cerebral haemorrhage to be made. I make no criticism of the failure to make this diagnosis at an earlier time, accepting the opinion of Dr Bell that it was not until around 4.00pm that it became apparent that Mr Bruinger’s presentation had a neurological cause.

In hindsight it is regrettable that the diagnosis of a NSTEMI was made as it had two consequences. First, it distracted Mr Bruinger’s clinicians from an ongoing and open-minded assessment of his condition although, for the reason stated, I do not believe this process would have led to the correct diagnosis being made at an earlier time. Secondly, the NSTEMI diagnosis led to the initiation of antiplatelet anticoagulation therapy which is appropriate for this condition. However, it is not an appropriate therapy for a person suffering from a cerebral haemorrhage because of its blood thinning consequences. However, in Mr Bruinger’s case I accept the opinion of Dr Bell that there was a real chance of Mr Bruinger suffering his second cerebral bleed even without the anticoagulation so that his tragic death would still not have been avoided.

A matter of real concern arising from this investigation is the report of Dr Huckerby and her opinion that the overcrowding in her ED prevented Mr Bruinger from receiving, for a period of eight hours, the appropriate level of monitoring which his condition required. Whilst the incapacity to better monitor Mr Bruinger was not, in the circumstances of his case, a factor causative of his death, the state of affairs as described by Dr Huckerby had the prospect of seriously compromising patient safety. It also must have been particularly difficult and stressful for the medical and ancillary staff. In this circumstance it behoves those persons responsible for the management of the RHH to investigate the situation and to adopt changes which prevents or at least significantly reduces the prospect of its repetition.

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 Bruinger’s family and loved ones.

**Dated: 17th day of August 2018 at Hobart in the State of Tasmania.**

**Rod Chandler**
**Coroner**