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**