IN THE CORONERS COURT
OF VICTORIA
AT MELBOURNE

Court Reference: COR 2020 001307

FINDING INTO DEATH WITHOUT INQUEST

Form 38 Rule 63(2)
*Section 67 of the **Coroners Act 2008***

Findings of:	Sarah Gebert, Coroner
Deceased:	Mrs K [1]
Date of birth:	[redacted] 1938
Date of death:	7 March 2020
Cause of death:	1(a) Cardiac tamponade 1(b) Dissection of ascending aorta Contributing factors Hypertension, hypercholesterolaemia
Place of death:	Dandenong Hospital, Monash Health, 135 David Street, Dandenong, Victoria
Keywords:	Aortic dissection, chest pain pathway, chest pain NEC

1. This finding has been de-identified by order of Coroner Sarah Gebert to replace the names of the deceased and her family members with pseudonyms to protect their identity and redact identifying information

INTRODUCTION

1. On 7 March 2020, Mrs K was 81 years old when she died in hospital following an aortic dissection. At the time of her death, Mrs K lived in Dandenong.

THE CORONIAL INVESTIGATION

2. Mrs K's death was reported to the coroner as it fell within the definition of a reportable death in the *Coroners Act 2008* (**the Act**). Reportable deaths include deaths that are unexpected, unnatural or violent or result from accident or injury.

3. The role of a coroner is to independently investigate reportable deaths to establish, if possible, identity, medical cause of death, and surrounding circumstances. Surrounding circumstances are limited to events which are sufficiently proximate and causally related to the death. The purpose of a coronial investigation is to establish the facts, not to cast blame or determine criminal or civil liability.

4. Under the Act, coroners also have the important functions of helping to prevent deaths and promoting public health and safety and the administration of justice through the making of comments or recommendations in appropriate cases about any matter connected to the death under investigation.

5. As part of my investigation and in light of concerns received from Mrs K's family, I asked the Coroners Prevention Unit (**CPU**)[1] to review the medical care Mrs K received at Dandenong Hospital.

6. This finding draws on the totality of the coronial investigation into Mrs K's death. Whilst I have reviewed all the material, I will only refer to that which is directly relevant to my findings or necessary for narrative clarity. In the coronial jurisdiction, facts must be established on the balance of probabilities.[2]

[1] The CPU was established in 2008 to strengthen the coroner's prevention role and to assist in formulating recommendations following a death. The CPU is comprised of health professionals with training in a range of areas including medicine, nursing, public health and mental health. The CPU may also review the medical care and treatment in cases referred by the coroner as well as assist with research into public health and safety.

[2] Subject to the principles enunciated in *Briginshaw v Briginshaw* (1938) 60 CLR 336. The effect of this and similar authorities is that coroners should not make adverse findings against, or comments about, individuals unless the evidence provides a comfortable level of satisfaction as to those matters taking into account the consequences of such findings or comments.

Background

7. Mrs K was a non-smoker. Her medical history included hypertension treated with perindopril, high cholesterol treated with atorvastatin, glaucoma treated with Combigan, prednisolone, and latanoprost eye drops, diverticular disease of the bowel, vertigo, ovarian cyst, and arthritis treated with meloxicam. Mrs K's family reported that she was fit and well, lived alone, and was still driving and attending to her own care.

8. At about 10.00am on 2 March 2020, Mrs K developed chest pain whilst at home. Ambulance Victoria paramedics subsequently attended and administered aspirin, GTN,[3] and morphine to treat Mrs K's pain. She was taken to Dandenong Hospital at approximately 12.00pm. Triage notes describe the pain as *"sudden onset central chest pain radiating to the jaw, stabbing in nature, worse on inspiration, tightness in throat area*".

9. Mrs K was seen by an Emergency Department (**ED**) doctor who recorded a detailed presenting history of *"Sudden onset of central chest pain ... crushing in nature. Not ripping/tearing ... radiating to jaw bilaterally, teeth, neck ... not pleuritic"*. The doctor also noted that Mrs K's initial blood pressure was recorded as 220/110. The chest pain was not reproducible by palpation (pressing) on the chest and the doctor specifically noted that there was no *"radial-radial delay"*[4] and *"no radio-femoral delay".* Observations were otherwise normal.

10. A general and cardiovascular examination appear to have been unremarkable. Mrs K's blood pressure settled without treatment and her pain appears to have settled to a low level and did not require further strong analgesia. The initial plan, which was approved by the ED consultant, was to *"rule out cardiac cause of chest pain"* and the management plan consisted of:

    (a) initial blood tests including troponin;[5]

[3] GTN is glyceryl trinitrate or nitroglycerin. This is a non-analgesic medication that is specifically used to relive the pain associated with coronary artery disease.

[4] Radial-radial and radio-femoral delay relates to the simultaneous palpation of either both radial pulses at the wrist, or of a radial pulse and a femoral pulse in the groin to detect differences in the timing or 'strength' of the pulses. In cases where aortic dissection involves an artery to an upper limb there may be an appreciable difference in the pulses when palpated. Whilst this may be a 'classic' sign of aortic dissection, it is frequently not present and cannot be used to discount a diagnosis of aortic dissection.

[5] Troponin is a protein contained in heart muscle that is released into the blood when there is injury of any cause to the heart muscle. Elevation above a certain baseline is consistent with heart muscle injury, usually due to coronary artery disease. It may be normal in aortic dissection, particularly when the dissection does not involve the coronary arteries.

(b) electrocardiogram (**ECG**), which was normal;

(c) to measure blood pressure in both arms;

(d) repeat troponin tests; and

(e) admission to short-stay unit (**SSU**).

12. No medical imaging was performed. In the SSU, a further troponin test was normal and it was noted that the blood pressure in both arms was equal. Other blood tests were normal.

13. Mrs K was discharged home from SSU at approximately 4.30pm that afternoon with a diagnosis of *"chest pain NEC"*.[6] The discharge plan was for Mrs K to follow-u with her general practitioner (**GP**) in the next one to two weeks and a request to *"consider outpatient investigation if recurrent chest pains."*

MATTERS IN RELATION TO WHICH A FINDING MUST, IF POSSIBLE, BE MADE

Circumstances in which the death occurred

11. At 10.45am on 6 March 2020, Mrs K returned to the ED at Dandenong Hospital, having been referred back by her GP with ongoing retrosternal chest pain and a report of an abnormal ECG. Mrs K described feeling generally unwell since discharge, feeling tired and weak, with difficulty breathing, and ongoing mild chest pain that had never completely resolved from her previous attendance. Her observations revealed a moderately elevated heart rate.

12. A chest x-ray was performed, which did not report abnormality of the aorta or mediastinum.[7] Further investigations revealed troponin to be minimally elevated at 22 and the working diagnosis was *"?NSTEMI"*.[8]

13. Mrs K was admitted to the hospital that evening under the cardiology unit. She was commenced on dual anti-platelet medications,[9] aspirin, and ticagrelor. Notably, the cardiology

[6] 'Chest pain NEC' means Chest pain, not elsewhere classified. This is from a computer-generated list.
[7] The mediastinum is the area of the chest between the lungs and containing the heart, great blood vessels including the aorta, major airways, and the oesophagus.
[8] NSTEMI means non-ST elevation myocardial infarction. This is a myocardial event producing pain and troponin rise without the typical ECG changes of a significant myocardial infarction. It may be caused by small vessel disease in the heart.
[9] Anti-platelet medication inhibits the action of platelets which are involved in the formation of blood clots. In the setting of heart disease the intention is to reduce the formation of small blood clots within the coronary arteries. Antiplatelet agents will increase the risk of bleeding or the extent of bleeding already present.

medical officer's admission notes record a gradual onset of pain. She was also given enoxaparin for deep vein thrombosis prophylaxis.

14. The following morning, 7 March 2020, the Monash Heart consultant ward round reviewed Mrs K at which time they noted ongoing rapid heart rate, ECG changes, and pleuritic[10] chest pain. It was considered that she required a CT pulmonary angiogram (**CTPA**)[11] to diagnose or rule out a pulmonary embolism as a cause for her presentation. The notes record *"if not pulmonary embolus, then coronary artery disease"* and reflect the working differential diagnoses being considered did not include aortic dissection. A D-dimer[12] blood test was performed and was elevated at 0.58 mg/L (normal range is 0 to 0.2). A CRP[13] was also measured at this time, which was significantly elevated at 178.

15. Whilst in the ward awaiting the CTPA, Mrs K collapsed, initially in ventricular fibrillation and then a PEA[14] arrest from which she could not be resuscitated. Despite a prolonged resuscitation attempt involving the administration of thrombolysis (clot dissolving) medication for what was a presumed massive pulmonary embolism, Mrs K passed away at 3.05pm.

Identity of the deceased

16. On 7 March 2020, Mrs K, born [redacted]1938, was visually identified by her daughter-in-law, [redacted].

17. Identity is not in dispute and requires no further investigation.

[10] Pleuritic is a character of chest pain ascribed to pain originating from the lining of the chest (pleura). This would be considered to be sharp, catching pain reproduced by breathing or coughing. It is associated with conditions affecting the pleura, particularly infection or inflammation in the chest, or pulmonary embolus affecting the lung beneath the pleura.
[11] is a CT scan involving the administration od radiological contrast to demonstrate the pulmonary arteries and any clots (emboli) within.
[12] D-dimer is a by-product of blood clotting that is abnormally elevated in the blood where there is any significant blood clot, including in deep vein thrombosis (**DVT**) and pulmonary embolism (**PE**), trauma, post-operative and in generalised abnormal blood clotting. It is commonly used to screen and assess risk in patients with possible DVT or PE. It would be anticipated to be elevated in aortic dissection and there are some clinical risk scoring systems that utilise D-dimer to assess aortic dissection risk.
[13] CRP means C-reactive protein. This is a non-specific marker of infection or inflammation. Normal range is less than 5.
[14] PEA means pulseless electrical activity. This means that there was still electrical activity in the heart, but there was no effective cardiac output. Causes of this would be shock i.e. massive loss of blood volume, massive pulmonary embolism where the outflow of blood from the heart to the lungs is obstructed so that blood flow through the heart and circulation ceases, and cardiac tamponade, where blood or fluid in the pericardial sac around the heart prevents blood returning to the heart from filling the ventricles and cardiac output drops to zero. In the short term the electrical activity of the heart continues and can be seen on a cardiac monitor. Aortic dissection is a cause of cardiac tamponade when it ruptures into the pericardial sac. The amount of blood required to cause this may be relatively small – of the order of a few hundred millilitres.

Medical cause of death

18. Forensic Pathologist Dr Yeliena Baber from the Victorian Institute of Forensic Medicine (**VIFM**) conducted an autopsy on 10 March 2020 and provided a written report of her findings dated 31 July 2020.

19. The post-mortem examination revealed 380 ml of haemopericardium (blood in the pericardial cavity), dissection of the proximal aorta, coronary artery and aortic atherosclerosis, granular renal cortices, and bilateral pleural effusions.

20. Dr Baber was of the opinion that Mrs K's death was due to compression of the heart by haemopericardium, which prevented effective filling of the ventricles during diastole and leading to a rapidly decreasing cardiac output. This was caused by dissection of the aorta at a point of atherosclerosis which has ruptured into the pericardial sac. Histology conformed the macroscopic findings and showed hypertensive changes in the kidneys.

21. Toxicological analysis of post-mortem samples identified the presence of oxazepam[15] and paracetamol.

22. Dr Baber provided an opinion that the medical cause of death was *"1(a) Cardiac tamponade"* secondary to *"1(b) Dissection of ascending aorta"* with contributing factors of hypertension and hypercholesterolaemia. Dr Baber was also of the opinion that Mrs K's death was due to natural causes.

23. I accept Dr Baber's opinion.

FURTHER INVESTIGATION

Family concerns

24. Mrs K's family voiced concerns that a CTPA was not undertaken at her initial presentation to Dandenong Hospital on 2 March 2020 and she was discharged without further investigation despite ongoing chest pain.

[15] Oxazepam is a sedative/hypnotic drug of the benzodiazepine class.

Coroners Prevention Unit review

Aortic dissections generally

25. The CPU explained that aortic dissection is a relatively rare condition but one that that features regularly in this Court. Chest pain due to coronary artery disease is more common than aortic dissection. Despite this, the diagnosis continues to feature regularly and this case highlights some of the issues that are frequently seen, in particular:

    (a) the apparent discounting of the diagnosis of aortic dissection on the basis of the absence of classic features; and

    (b) the placement of the patient on a 'chest pain pathway' in the ED that is designed to stratify chest pain patients into risk categories for an acute coronary syndrome (that is, heart attack), but does not address ongoing symptoms or the cause of the chest pain (such as aortic dissection or pulmonary embolism) once the pathway is complete.

26. A 'classic' description of the onset of aortic dissection pain could be considered to be:

    (a) sudden onset;

    (b) severe at onset;

    (c) radiating to the back between the shoulders;

    (d) being of a tearing or ripping quality;

    (e) producing a significant difference (>20 mmHg) in the blood pressures between the upper limbs; and

    (f) a chest x-ray may reveal a wide mediastinum or abnormal aortic outline.

27. However, it is known that this 'classic' description is not the 'typical' description and that:

    (a) abrupt onset occurs in most dissections (85%);

    (b) anterior chest pain is common (approximately 70%) in Type A dissections, such as was found in Mrs K;

    (c) posterior chest pain occurs in about 32% of Type A dissections;

    (d) a pulse or blood pressure deficit is present in only 20% of Type A dissections;

(e) the quality of the pain is describes as tearing or ripping in 50% of type A dissections;

(f) the quality of the pain is described as sharp in more than 60% of Type A dissections; and

(g) a widened mediastinum is present in about 60% of patients with aortic dissection and abnormal aortic outline present in about 50%.

31. It can be appreciated that aortic dissection cannot be excluded by the absence of x-ray abnormalities, equal radial pulses and blood pressures, or the absence of a description of ripping or tearing posterior chest pain radiating into the back.

32. In the CPU's experience, these are common and persisting themes in cases of missed aortic dissection and is not confined to junior doctors.[16]

33. Similarly, in commonly used medical and emergency medicine textbooks and expert guidelines the descriptions of Type A aortic dissection pain are commonly of sudden onset, anterior chest pain radiating to the jaw, more often described as sharp, rather than ripping or tearing. The pain of acute myocardial infarction is usually not acute onset severe sharp pain.

Family concerns regarding Mrs K's presentation on 2 March 2020

34. The CPU explained that a CTPA was not undertaken at Mrs K's initial presentation because at this stage, pulmonary embolism was not considered as a differential diagnosis. Therefore, there was no requirement to do a CTPA in these circumstances. A CTPA would not have necessarily detected an aortic dissection. However, depending on a variety of circumstances, it might have been suspected.

35. The CPU considered that the description of the pain and other features described at this presentation were not suggestive of a pulmonary embolism and Mrs K did not have any risk factors for a pulmonary embolism. She did however have risk factors for coronary artery disease and aortic dissection, namely hypertension, high cholesterol, and her age.

36. The CPU agreed that it was concerning that Mrs K was sent home without further investigation despite experiencing ongoing chest pain. Unfortunately, this reflects the Court's

[16] See for example the *Finding into Death With Inquest of Constandia Petzierides,* dated 5 June 2014, which highlighted the issues often seen by the court and was the subject of a 'roundtable' held jointly by the court and the Australasian College for Emergency Medicine. The finding is available at https://coronerscourt.vic.gov.au/sites/default/files/2018-12/constandiapetzierides_157110.pdf and responses to recommendations from the Australasian College for Emergency Medicine, Ambulance Victoria, and Victorian Department of Health are published on the Court's website.

past experience of patients with chest pain – being placed on 'chest pain pathways' but then discharged once their risk of an acute coronary event is deemed to be low to medium. The CPU considered that it was reasonable for clinicians to consider coronary artery disease, which is far more common and likely than aortic dissection. However, once an acute coronary event was ruled out, there was no further investigation as to the cause of Mrs K's significant chest pain. She had not undergone simple investigations such as a chest x-ray.

CPU review of Mrs K's medical care provided on 2 March 2020

37. When Mrs K first attended Dandenong Hospital ED there was a clear history obtained of *"sudden onset central chest pain radiating to the jaw, stabbing in nature".* There was also specific comment about the quality of the pain as not being *"ripping or tearing"* and the patient was specifically examined for pulse abnormalities in the upper limbs and had a specific request for blood pressure to be measured in both upper limbs. The CPU advised that this indicates that the attending ED doctor specifically thought about the possibility of the diagnosis of aortic dissection as there would be no other reason to enquire about a 'ripping or tearing' character of the pain or to measure the blood pressure in both arms.

38. The CPU advised that while the doctors who initially assessed Mrs K on 2 March 2020 probably considered the possibility of aortic dissection, the diagnosis was likely discounted on the basis of the absence of classical features or examination findings. There appears to have been a lack of appreciation of the inability of the absence of 'classic' features of aortic dissection to be able to clinically rule out the diagnosis.

39. Despite the presence of some atypical features for pain from coronary artery disease, and 'ruling out' an acute coronary syndrome in the 'chest pain pathway', no other cause for Mrs K's pain, such as a lung condition or pulmonary embolism, appear to have been sought by undertaking simple tests such as a chest x-ray or a D-dimer, and Mrs K was discharged with ongoing, albeit mild, undiagnosed chest pain. The CPU noted that the discharge diagnosis of 'Chest pain NEC' is not a diagnosis.

40. The CPU considered there were clues in Mrs K's presentation that, in other circumstances, might have led to a diagnosis of aortic dissection whilst she was in good condition. However,

CPU recognises that the surgery associated with repair of aortic dissection is extensive, complex, and high risk, particularly in an elderly patient.[17]

41. The CPU suggested that Monash Health:

(a) review their approach to patients with significant but undiagnosed chest pain after passing through the chest pain pathway; and

(b) review and improve the education of ED and other doctors with regard to this aspect of aortic dissection presentation and diagnosis.

CPU review of Mrs K's medical care provided from 6 March 2020

42. In Mrs K's subsequent admission on 6 March 2020, there is a specific admission note from a junior doctor that indicates the onset of the pain was gradual and acknowledging that her blood pressure had been recorded as equal in both arms. This may have steered treating doctors away from aortic dissection in the second admission. It is not known why the description of the acuity of the onset of Mrs K's pain would change so dramatically.

43. The CPU could find no record of a history of the acute onset of Mrs K's initial pain being acknowledged or considered by doctors, including cardiologists, caring for her on the second admission. It is not known if her history was retaken in detail by a consultant cardiologist.

44. The consideration of coronary artery disease and pulmonary embolism during the second admission was appropriate as there were features of these conditions. It is unfortunate that Mrs K passed away before the performance of the CTPA, as this *may* have noted the dissection.

45. The CPU suggested that Monash Health consider routinely using bedside ultrasound in similar circumstances of a PEA arrest where there may be a reversible cause. This could enable early, specific and directed treatment for specific conditions, described above, that in some circumstances could be lifesaving. Many critical care and cardiology doctors would already have the skills to undertake this.

[17] Surgery to repair an aortic dissection such as Mrs K's is extensive and associated with significant complications and has a relatively high mortality (20 to 40%) which would increase with the age and other comorbidities of the patient. Surgery requires cardiac bypass and, depending upon the individual anatomy and extent of the dissection, may involve removal and replacement of the aortic root, aortic valve, and variable parts of the arch of the aorta, from which arise the coronary arteries and major arteries to the brain etc. These may need to be re-attached into the graft. There is therefore high risk of stroke, myocardial infarction, bleeding, kidney failure, multi-organ failure, and sepsis as complications.

46. The CPU considered that conduct of Mrs K's resuscitation was appropriate. It was assumed that a massive pulmonary embolism was the cause of her PEA arrest, but this was never proven and treatment with thrombolytic medication was undertaken on the presumptive diagnosis of a pulmonary embolism. Whilst having no effect on Mrs K's outcome, the administration of thrombolytic therapy is contraindicated in aortic dissection where the lysis of blood clot may cause or worsen severe haemorrhage.

47. Resuscitation continued for over an hour and CPU considered that the performance of a bedside ultrasound early in the resuscitation cardiac arrest would most likely have revealed a hemopericardium and directed more specific therapy, although it would not have changed the outcome as by this point Mrs K's aorta had ruptured. In another patient this may have been lifesaving by enabling early and directed therapy for potentially reversible conditions such as cardiac tamponade, massive pulmonary embolism, tension pneumothorax, or abdominal or thoracic bleeding.

Submissions from Monash Health

48. In finalising my investigation into Mrs K's death, I informed Monash Health of the CPU's advice outlined above and invited submissions.

49. Peter Ryan, Chief Legal Officer at Monash Health, provided a detailed submission in which informed me that Monash Health has spent considerable effort in improving their diagnosis rate of aortic dissections and have developed an evidence-based guideline to assist the process. I was provided with a copy of the guideline.

50. Mr Ryan also informed me that in order to address the difficulty that ED medical staff encounter with regard to differentiating the conditions of pulmonary embolism from aortic dissection, Monash Health is currently reviewing the CTPA protocol to determine whether minor modification of the protocol can assist in detection of both conditions, whilst not exposing patients to needles increases in either radiation exposure or iodine contrast administration. The proposed new protocol will be termed 'Dual rule out' and be aimed at assessing patients with complex clinical presentation with a view to excluding both pulmonary embolism and aortic dissection.

51. Mr Ryan noted that Monash Health admitted 98 patients with aortic dissection between January 2017 and December 2019, about 33 per year. Monash Health is aware that we some diagnoses are missed – eight have been identified by the coroner but not originally by Monash

Health in a four-year period – amounting to a missed diagnosis of aortic dissection of just under 6% in that period.

52. Using 2019 data, Monash Health sees approximately 16,000 presentations a year with a triage diagnosis of chest pain, approximately 10,000 of which were believed to be cardiac in origin at triage. About 80% of these (8,000) presentations were discharged home with no significant cardiac pathology identified. That amounts to 22 patients each day. Mr Ryan noted that arguably, to detect an additional two cases per year, Monash Health would potentially need to scan an additional 8,000 patients, thereby significantly impacting on workload and unnecessarily exposing patients to radiation.

53. In regard to the CPU's suggestion that bedside ultrasound could be routinely used early in PEA, Mr Ryan noted that in this case the arrest occurred in a non-tertiary hospital at 2.00pm on a Sunday when such staff are unavailable. This is in contrast to some tertiary hospitals where doctors experienced in echocardiography or echocardiography technicians may be readily available 24/7. The use of echocardiography by non-credentialled staff is fraught with danger. In these circumstances, Mr Ryan advised that Monash Health would not be able to implement such a recommendation at the current time.

54. In regard to Mrs K's presentations, Mr Ryan noted that the diagnosis of aortic dissection was considered on both occasions but was not pursued. As the post-mortem revealed, this was erroneous, but the risk stratification algorithm was followed, the patient's score was zero and did not progress to a CT scan on her first admission. There was not sufficient concern at that time of a dissection or pulmonary embolism to progress to a D-Dimer test. Her blood pressure settled as did her chest pain. Troponin was 4 (0-10).

55. On the second admission Mrs K presented with numerous symptoms, including ongoing mild chest pain that had never completely resolved from her previous admission. Troponin was mildly elevated at 21 but did not rise, subsequent readings being 22 and 17. Again following the algorithm, her score was zero. On this occasion a D-Dimer was performed on 7 March and was elevated to 0.58 mg/L (0-0.2), so a CTPA was ordered to rule out a pulmonary embolism. The patient arrested whilst waiting for the scan.

56. Mr Ryan also noted that thrombolytic therapy was appropriate given the provisional diagnosis of pulmonary embolism and given only when Mrs K failed to respond to initial resuscitation. While the CPU's advice that the administration of thrombolytic therapy is contraindicated in aortic dissection where the lysis of blood clot may cause or worsen severe

haemorrhage is accurate, in Mrs K's case, that can only be said with the benefit of hindsight. Given that the provisional diagnosis was pulmonary embolism, the scan ordered was designed to show the pulmonary trunk and its branches and would not have adequately showed an aortic dissection.

Review by the Coroners Prevention Unit

57. The CPU reviewed Mr Ryan's submission and considered the possibility of an enhanced CTPA protocol was promising.

58. The CPU also considered the new guideline, *Suspected Thoracic Aortic Dissection Assessment Emergency Department Procedure,* provided a safe and sensible approach utilising a clinical prediction risk score, the *'Aortic Dissection Detection Risk Score'.* The CPU noted that it relies on the taking of an adequate history of the pain and other risk factors and of aortic dissection being 'suspected'. In the Court's experience, a common theme in missed diagnosis is the quality of history taking. The timing, nature and severity of the pain is central to suspecting the diagnosis and a careful clinical history to clarify the characteristics of the presenting symptoms is essential.

59. The CPU commented that the guideline could also be subtitled *An approach to a patient with severe, undiagnosed chest pain*. EDs are generally very good at identifying more common causes of chest pain such as thoracic/lung conditions, ischaemic chest pain, and pulmonary emboli, but in the CPU's experience also demonstrates the shortcomings of ceasing investigations or further diagnostic consideration once these common conditions have been excluded, sometimes using a 'chest pain pathway'. The discharge of a patient with a 'diagnosis' of Chest Pain NEC (not elsewhere classified) encapsulates this issue.

60. The CPU acknowledged the difficulties that diagnosing aortic dissection can present and recognises that there are numbers of unusual, atypical, or rarer presentations of aortic dissection. However, the CPU's experience is that the majority of missed aortic dissections have relatively typical presentations and risk factors and they are 'missed' because of shortcomings in obtaining basic clinical information or interpreting this information.

61. The CPU considers that senior ED clinician review of patients who have experienced 'severe, acute onset' chest pain and are being discharged from a short stay unit or 'chest pain pathway' without a clear alternative cause could be a beneficial addition to the new guideline.

62. Given the changes Monash Health has implemented or intend to implement, I do not intend to make any recommendations. However, I will distribute this finding to Monash Health for consideration of further possible changes that may be implemented and for education purposes.

FINDINGS AND CONCLUSION

63. Pursuant to section 67(1) of the Act I make the following findings:

(a) the identity of the deceased was Mrs K, born [redacted]1938;

(b) the death occurred on 7 March 2020 at Dandenong Hospital, Monash Health, 135 David Street, Dandenong, Victoria, from cardiac tamponade secondary to dissection of ascending aorta with contributing factors of hypertension and hypercholesterolaemia; and

(c) the death occurred in the circumstances described above.

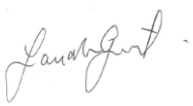
I convey my sincere condolences to Mrs K's family for their loss.

Pursuant to section 73(1A) of the Act, I order that this finding be published on the Coroners Court of Victoria website in accordance with the rules.

I direct that a copy of this finding be provided to the following:

Mr K, senior next of kin
Monash Health
Senior Constable Ganesh Naidu, Victoria Police, reporting member

Signature:



Coroner Sarah Gebert

Date: 28 February 2023

NOTE: Under section 83 of the ***Coroners Act 2008*** ('the Act'), a person with sufficient interest in an investigation may appeal to the Trial Division of the Supreme Court against the findings of a coroner in respect of a death after an investigation. An appeal must be made within 6 months after the day on which the determination is made, unless the Supreme Court grants leave to appeal out of time under section 86 of the Act.