CORONER'S COURT OF THE AUSTRALIAN CAPITAL TERRITORY

Case Title:	**Inquest into death of JEFFREY BROWN**
Citation:	**[2019] ACTCD 16**
Findings Date:	4 November 2019
Before:	**Coroner Boss**
Decision:	See [41]-[42].
Catchwords:	**CORONIAL LAW** – cause and manner of death – whether medical procedure contributed to death – whether appropriate investigations made – whether failure to diagnose complication of treatment – whether post-surgical treatment appropriate – conflicting expert reports – no matter of public safety – hearing unnecessary
Legislation applicable:	*Coroners Act 1997* (ACT), s 13(1)(a), 13(1)(f), 34, 34A, 52, 55
Cases cited:	*Briginshaw v Briginshaw* (1938) 60 CLR 336 *The Queen v Coroner Maria Doogan; ex parte Australian Capital Territory* [2005] ACTSC 74 *March v E & MH Stramare Pty Ltd* (1991) 171 CLR 506 *Harmsworth v The State Coroner* [1989] VR 989 *Taing & Buob v Territory Coroner* [2011] NTSC 58
File Number:	CD 81 of 2013

CORONER BOSS:

1. The death of Jeffrey Brown, a 72 year old man at the date of his death, was reported to the ACT Coroner as he was thought to have died unnaturally in unknown circumstances; alternatively, because no doctor was prepared to write a death certificate suggesting the most probable cause of death. Chief Coroner Walker initially had carriage of the inquest but she has become unavailable.

2. Mr Brown was ordinarily resident in NSW but died in the ACT. I, acting as the ACT Coroner, have primary jurisdiction given the location of death is in the ACT. However, the NSW Coroner also has concurrent jurisdiction to investigate Mr Brown's death should they choose to do so.

Facts surrounding death

3. Mr Brown started to complain of chest pain and shortness of breath in around April 2012. However, no cause of the pain was identified by doctors. In December 2012 Mr Brown suffered a Transient Ischaemic Attack (also known as a minor stroke). From that point Mr Brown was treated with medication for high cholesterol and to reduce his risk of further stroke.

4. On 25 February 2013 Mr Brown was admitted to The Canberra Hospital ("TCH") for a carotid endarterectomy, a procedure to remove plaque build-up and increase the diameter of the carotid artery. The operation was complicated by haematoma, but was considered successful. It was noted during the period of this admission however that Mr Brown had experienced a myocardial infarction at some time in the perioperative period, so he was recommended to undergo an urgent coronary angiogram. That occurred on 1 March 2013 and revealed a blockage in Mr Brown's right coronary artery. He was recommended to have a coronary artery bypass graft ("CABG") as an elective procedure, which was scheduled for 15 March 2013. Mr Brown was discharged to home on 3 March 2013.

5. Consequently, Mr Brown was admitted to TCH on 14 March 2013. The CABG operation was undertaken on 15 March 2013 and while technically successful, Mr Brown complained of ongoing pain and he required backup pacing possibly due to hypotension. Mr Brown was discharged from surgery initially to the Intensive Care Unit, but moved to the ward the following day when his condition improved. Mr Brown was discharged from TCH to home on 21 March 2013, and given discharge medications to lower the risk of further arterial blockages.

6. Mr Brown was reviewed at his residence by the Eden community nurse on 26 March 2013. During the visit Mr Brown complained of pain and the nurse observed that Mr Brown was very pale. An ambulance was called to take Mr Brown to the nearest hospital, which was Pambula District Hospital ("PDH").

7. While at PDH Mr Brown was diagnosed with thromboembolism involving a branch of the right pulmonary artery. Initially Mr Brown was given medication as treatment, but given his gradual deterioration despite treatment ultimately a decision was made to transfer Mr Brown to TCH where more specialist treatment was available. The transfer occurred on 31 March 2013.

8. On arrival at TCH, Mr Brown was found to have a highly elevated INR (measure of time for blood to clot). Mr Brown's dosage of anticoagulant medication was lowered so as to bring his INR down into the acceptable range. Tests undertaken on 1 April 2013 showed a decrease in INR and Mr Brown's condition appeared to be improving.

9. However, at 7:21am on 2 April 2013 Mr Brown was found in cardiac arrest by TCH staff. Resuscitation was attempted but was unsuccessful, and Mr Brown was declared life extinct at 7:42am.

Investigation

10. Associate Professor Sanjiv Jain conducted a post-mortem examination of Mr Brown at the direction of then Chief Coroner Walker. A/Professor Jain's opinion as to Mr Brown's cause of death was haemopericardium, due to anti-coagulation treatment for pulmonary thromboembolitic disease. A/Professor Jain found that Mr Brown's recent coronary artery bypass graft surgery with fibrinous pericarditis was a significant condition contributing to death, but not related to the disease or condition causing death. The CABG surgery was technically successful, and the grafts were clear. There was no evidence of pulmonary embolus, suggesting that the anticoagulant treatments had successfully dissolved the blood clot. A/Professor Jain stated that the capsules of Mr Brown's kidneys stripped with ease revealing normal parenchyma without evidence of pre-existing disease or scarring. While there were no mass lesions in Mr Brown's liver, the parenchyma showed acute centrivenous congestion, and necrosis involving acinar zones 2 and 3 were observed. Relevantly, A/Professor Jain stated:

> *It appears that haemopericardium was related to the anticoagulant treatment given for pulmonary thromboembolitic disease with the underlying fibrinous*

pericarditis following recent coronary artery bypass graft surgery contributing to the bleeding because of the presence of thin walled newly formed capillary blood vessels. The anti-coagulation treatment was considered necessary clinically because of the diagnosis of pulmonary thromboembolitic disease on CTPA and a positive D-dimer test.

11. Mrs Betty Brown, Mr Brown's wife, immediately expressed her concerns to Police in relation to the treatment Mr Brown received at Pambula Hospital, but also in Mr Brown's sudden deterioration at TCH over 1 and 2 April 2013. Additionally Mr Brown's son, Mr Kenneth Spearpoint, wrote a letter to the Court in which he requested answers as to why Mr Brown was kept at Pambula Hospital and questioned the events leading up to Mr Brown's cardiac arrest at TCH.

12. As a first step, former Chief Coroner Walker requested a written response from TCH as to its care of Mr Brown, and to respond as best it could to the questions raised by Mr Spearpoint. The former Chief Coroner also requested that Mr Brown's treating practitioners at PDH be requested to provide statements.

13. Dr Michael Pentin provided a written statement in which he stated that he admitted Mr Brown to PDH on 26 March 2013 after non-specific chest pain complaints. Investigations undertaken there revealed no new cardiac abnormality and no new ischaemic heart disease, but after a positive D-dimer test result he organised for Mr Brown to receive a CT pulmonary angiogram (CTPA). The CTPA revealed evidence of a small pulmonary embolism, and accordingly Mr Brown was commenced on an anticoagulation regime of medication. Dr Pentin said that Mr Brown's condition was discussed with the TCH cardiothoracic registrar who agreed that the treatment regime was appropriate. Mr Brown appeared in a stable condition until the morning of 30 March 2013 when he commenced vomiting. Blood tests taken that day revealed no major change from those tests undertaken on 28 March 2013. Dr Pentin ordered fluids and antibiotics to be administered in case the vomiting was due to an underlying infection, notwithstanding that Mr Brown did not display fever. A chest x-ray of Mr Brown revealed possible infection in his lower lobes. Dr Pentin arranged for Mr Brown to be transferred to TCH for further investigation and specialist treatment, and Mr Brown was transferred by ambulance to TCH later that day.

14. Dr Donald Reed provided a written statement in which he stated that he saw Mr Brown for the first time at PDH on 27 March 2013. At that time, Mr Brown's condition was stable and he had been commenced on anticoagulant medications. Dr Reed saw Mr Brown again on 28 March 2013, and again considered that Mr Brown was stable. Dr Reed spoke to Dr Bhattarai, who was the cardiothoracic surgeon who had conducted Mr Brown's CABG surgery, and Dr Bhattarai agreed that the anticoagulation treatment was appropriate. A Doppler ultrasound was ordered to try to find the presumed source of the embolism in Mr Brown's legs. Dr Reed saw Mr Brown a further time on 29 March 2013, and again Mr Brown's condition was stable. Dr Reed handed over Mr Brown's care to Dr Pentin in the early hours of 30 March 2013.

15. TCH provided three written responses to me during the course of this matter. In the first letter, Mr Brown's recovery from the CABG surgery was described as uneventful. They described the medication regime that Mr Brown had received in PDH as "the standard treatment regime for a pulmonary embolus". However, they noted that when this treatment did not resolve Mr Brown's symptoms of pain, nausea and dehydration, that an alternative diagnosis was post-cardiac injury syndrome. The letter described this syndrome as an immune response to trauma to the heart muscle, including from surgery, and noted that interpericardial haemorrhage leading to tamponade is a rare but well documented complication of the syndrome. They suggested that the clinical course of Mr Brown's observations leading up to his cardiac arrest did not suggest a slow progression of intracardiac bleeding, but more likely an acute and sudden bleed, and noted that "*the requirement for anticoagulant medication to treat the pulmonary embolus did little to lessen the bleeding around Mr Brown's heart*". The letter also suggested that the evidence the resuscitation team were able to intermittently establish a ventricular rhythm may give some indication that an acute bleed had only occurred a short time before. In regards to a specific family question about delay in attending to Mr Brown on the morning he died, the letter noted that while there is no record of a medical practitioner attending to Mr Brown between 5:30am and 6:30am, there is a record that a member of the nursing staff was sitting with him intermittently during this period. TCH's Clinical Review Committee did not find any issues with the treatment that Mr Brown had received.

16. Former Chief Coroner Walker caused this information to be provided to Mr Brown's family. The Court then received another letter from Mr Spearpoint alleging that matters of public safety arose in respect to a number of alleged clinical decision-making errors, which he particularised as follows:

    a. On 31 March 2013 at Pambula Hospital, Mr Brown displayed evidence of having suffered an acute kidney injury, specifically an increasing serum potassium rate and creatinine levels with an accompanying reduction in his glomerular filtration rate. Prior to transfer to TCH Mr Brown also suffered a significant period of oxygen desaturation. Mr Spearpoint alleged that there was an absence of any evidence of appropriate medical intervention in this regard at Pambula Hospital.

    b. On 1 April 2013 at TCH, Mr Brown suffered three further desaturation periods, which were progressively worse, and by late that evening became anxious and hypotensive. Mr Spearpoint alleged that no additional medical interventions were enacted, although Mr Brown continued to receive oxygen via a face mask and intravenous fluids.

    c. On 2 April 2013, prior to his collapse Mr Brown was aware of his deteriorating condition and asked to be taken to the Intensive Care Unit (ICU). He continued to have 95% oxygen saturation despite receiving 4 litres of oxygen per minute via face mask. At 6:06am Mr Brown displayed an abnormal heart rhythm on the continuously monitoring ECG, but Mr Spearpoint alleges that there is no record of nurses raising concerns with the medical team.

    d. Mr Spearpoint alleges that the ongoing resuscitation care was deficient in that they failed to notice that Mr Brown was disastrously metabolically acidotic and hyperkalaemic, which is consistent with a long standing acute kidney injury. Additionally, defibrillation was not attempted, nor a diagnosis of cardiac tamponade considered.

    Mr Spearpoint requested that a hearing be held to investigate these issues which he considered amounted to matters of public safety.

17. Former Chief Coroner Walker caused Mr Spearpoint's letter to be sent to TCH and requested they respond in writing. TCH responded initially by suggesting that Mr Brown's case be reviewed by an independent interstate medical expert. In a follow-up letter, TCH advised that it had forwarded Mr Spearpoint's letter to

its Clinical Review Committee, who conducted a further review of Mr Brown's case. They commented in relation to Mr Spearpoint's concerns as follows:

a. PDH had observed a deterioration in Mr Brown's renal function, which was normal upon his admission to hospital on 26 March 2013. Mr Brown was treated on 31 March 2013 with continuing intravenous saline and an initial bolus. Mr Brown's renal function was tested on admission to TCH and found to be abnormal. While Mr Brown's oxygen saturation had dropped to 90% on room air at 11:15am that day, it had improved to at least 96% with supplemental oxygen.

b. On 1 April 2013, Mr Brown did suffer some periods of decreased oxygen saturation, and his oxygen was increased in response, having the desired effect of increasing his oxygen saturation to 95%. Mr Brown was reviewed by the cardiothoracic surgeon during the morning ward round and no respiratory issues were identified at that time. Following another period of decreased saturation later that morning Mr Brown was reviewed again by the cardiothoracic registrar, who arranged for a chest x-ray to be taken. That x-ray showed a left lower lobe collapse/consolidation with an associated small pleural effusion. The registrar formed the opinion, in discussion with the admitting consultant, that the peripheral symptoms were consistent with Mr Brown's Reynaud's disease and ordered warm blankets, intravenous fluid and chest physiotherapy. Oxygen continued to be administered resulting in oxygen saturations between 94-100%. Later that evening Mr Brown's oxygen saturations decreased again and he was given higher flow oxygen via a Hudson mask, which increased his saturation to 99% in the short term and 95% consistently after the flow rate of oxygen was reduced.

c. Although Mr Brown did request to go to ICU, his observations at that time did not identify a deterioration in his condition sufficient to trigger a medical review. At 5:30am Mr Brown's oxygen saturation was 95% and his respiratory rate was 24 breaths per minute, while still receiving supplemental oxygen; again, not sufficient to trigger a medical review. The ECG at 6:06am does not demonstrate evidence of hyperkaelemia. The nurse who was caring for Mr Brown documented

in the clinical notes that his condition appeared to improve at about 6:30am.

d. Mr Brown's cardiac arrest was unwitnessed but the Medical Emergency Team (MET) call was made at 7:20am. An arterial blood gas taken at the commencement of the resuscitation efforts identified hyperkaelemia, acidosis and an abnormal INR. It appears that the MET team did not administer medications to reduce Mr Brown's levels of serum potassium, or perform a cardiac ECG or needle thoracocentesis. Thrombolysis was discussed with Mr Brown's cardiothoracic consultant during the MET call but a decision was made not to administer this. Ventricular fibrillation was only intermittently established.

18. Former Chief Coroner Walker then caused an independent, interstate medical expert to review Mr Brown's treatment and care at both TCH and PDH. That review was undertaken by Dr Peter Slezak, a consultant physician in private practice in NSW with specific experience and interest in cardiology and haematology. Relevantly, Dr Slezak provided the following opinions:

    a. The treatment provided to Mr Brown at PDH was adequate. Mr Brown received prompt and appropriate investigation on admission, and the TCH cardiothoracic surgical service was consulted on the proposed treatment regime. It was appropriate following Mr Brown's progressive deterioration that a decision be made to transfer him to TCH. However, Mr Brown's dose of Warfarin should have been omitted or reduced on 30 March 2013 after his INR had increased on that day; this is a "minor criticism".

    b. The treatment provided to Mr Brown at TCH cannot be faulted. Mr Brown received appropriate treatment and therapy post-admission, and throughout the admission Mr Brown had been appropriately observed and increases in his INR promptly corrected. The slight fall in haemoglobin level had been noted without specific comment though repeated clinical examination did not suggest acute blood loss. The chest x-ray undertaken was compared to a prior x-ray of 19 March 2013 and no change in heart shadow was observed. Mr Brown remained afebrile, not complaining of abdominal pain, there was nil significant alteration in his ECG and the sole symptom was ongoing

nausea. Appropriate imaging was to be undertaken to further evaluate the ongoing nausea and deranged liver chemistries, but were sadly precluded by the fatal asystole cardiac arrest on the morning of 2 April 2013.

19. Dr Slezak also provided a supplementary report in response to the correspondence the Court had received from Mr Spearpoint. Dr Slezak commented as follows:

    a. It was the deterioration in Mr Brown's condition observed at PDH, specifically the ongoing nausea, vomiting, and possible chest infection, which prompted the request to transfer Mr Brown to TCH.

    b. It was only on 31 March 2013 that Mr Brown was observed to have had an elevated blood urea and serum creatinine and eGFR; prior to that point Mr Brown's serum potassium was within normal limits and his liver chemistry from admission was normal. The concern was that Mr Brown had become dehydrated, and this was appropriately treated with the commencement of intravenous fluid therapy. Further investigation was not conducted in light of Mr Brown's clinical deterioration and the decision to transfer him to TCH.

    c. It is not correct that no medical interventions were undertaken at TCH in relation to periods of desaturation: Mr Brown continued to receive oxygen, for periods of increased flow rate, to maintain an oxygen saturation equal to or above 95%. A further decline in renal function was noted on 1 April 2013 and a decision made to continue intravenous fluid therapy in view of his ongoing nausea. At that time, there would not have been any indication for further renal investigations.

    d. In relation to the allegedly abnormal ECG of 6:06am on 2 April 2013, the MEWS chart did not indicate cause for significant concern on the part of the nursing staff during the early morning hours.

    e. In the acute setting of profound hypotension and hypoxia, a severe lactic acidosis can develop within 15-20 minutes (as was seen in Mr Brown's case), notwithstanding Mr Spearpoint's assertion that metabolic acidaemia takes hours to develop. The profound hyperkaelemia is an accompaniment to Mr Brown's profound

metabolic acidosis, not (as suggested by Mr Spearpoint) an acute kidney injury. It seems likely that the failure to restore any viable cardiac rhythm would have influenced any management directed towards correction of the profound acidosis and hyperkaelemia.

f. It is true that the possibility of a cardiac tamponade was not diagnosed during Mr Brown's deterioration. However, there was very little to raise a clinical suspicion of an underlying pericardial effusion. There had not been any alteration in the appearance of the cardiac silhouette following the introduction of anticoagulant therapy, despite the significantly elevated INR noted at the time of Mr Brown's transfer from PDH to TCH. Serial ECGs did not reveal any evidence of QRS alternans, a cardiographic sign that may be seen with a significant pericardial effusion. Bleeding complications are well recognised in association with combination anticoagulant therapy. As there was little to suggest, on clinical grounds, the possibility of an underlying pericardial effusion, within the acute timeframe, no criticism should be levelled at medical staff for not performing an ECG (as time simply did not allow for this to be performed) or attempted pericardiocentesis.

20. Former Chief Coroner Walker caused Dr Slezak's reports to be forwarded to Mr Brown's family. They indicated an intention to engage their own expert to review Mr Brown's treatment and forwarded me a report of Assistant Professor Sam Parnia, a colleague of Mr Spearpoint and an Assistant Professor of Medicine and Director of Resuscitation. A/Professor Parnia considered that Mr Brown's demise could have largely been prevented and that Mr Brown had demonstrated signs of haemodynamic compromise over the two days prior to his cardiac arrest. He opined:

    a. Mr Brown's condition met the criteria for clinical shock and/or severe septic shock, which if not treated will inevitably deteriorate into a cardiac arrest. Specifically, Mr Brown displayed on many occasions between 31 March and 1 April 2013 a low systolic blood pressure against a normal baseline which should have, on TCH's own MEWS guidelines, led to an escalation of medical care most likely to ICU.

    b. Mr Brown's blood test results from 31 March 2013, and specifically the reduction in serum bicarbonate, reveal signs of progressive acute renal failure or acute kidney injury, and severe metabolic acidosis. In

light of recent CABG surgery Mr Brown was at risk from the development of post-operative complications leading to pericardial effusion, which if untreated would lead to cardiac tamponade and cardiac arrest. If the appropriate investigations had been undertaken they would have discovered the cause of Mr Brown's deterioration and his cardiac arrest was preventable. The alternative possibilities included severe infection (septic shock) or progression of pulmonary embolism, the latter of which could have easily been tested for using a CT scan of the chest or bedside echocardiography.

c. The resuscitation of Mr Brown did not meet the current Australian Resuscitation Council guidelines, in particular the failure to defibrillate and the failure to treat the hyperkaelemia.

21. Former Chief Coroner Walker then asked A/Professor Jain whether he could give an indication on the balance of probabilities as to when the pericardial effusion/haemopericardium suffered by Mr Brown occurred, and whether it is correct to infer that this was the cause of his cardiac arrest. A/Professor Jain responded that he could not indicate the likely timing of the haemopericardium, but in view of the fact that there was acute centrivenous necrosis with congestion in the liver it would have been of at least some hours' duration. The A/Professor also noted that haemopericardium was the only significant abnormality found on post mortem examination to explain death, and this was secondarily associated with bilateral pulmonary oedema and liver congestion and necrosis indicating cardiac failure.

Scope of Inquest

22. I am required by section 52(1) of the *Coroners Act 1997* (the Act) to make findings as to the identity of the deceased person, when and where they died, and the manner and cause of their death. I am also required by section 52(4)(a) of the Act to state whether a matter of public safety is found to arise in connection with the inquest, and if I find such a matter, may comment upon it.

23. Coronial proceedings are civil proceedings and so the civil standard of proof - that is, on the balance of probabilities - applies in coronial matters. However, the Coroner is also to have regard to the principle laid down in *Briginshaw v Briginshaw* (1938) 60 CLR 336 as stated by Dixon J at 361-2:

"The truth is that, when the law requires the proof of any fact, the tribunal must feel an actual persuasion of its occurrence or existence before it can be found. ... The seriousness of an allegation made, the inherent unlikelihood of an occurrence of a given description, or the gravity of the consequences flowing from a particular finding are considerations which must affect the answer to the question whether the issue has been proved to the reasonable satisfaction of the tribunal."

24. In *R v Doogan; Ex Parte Lucas Smith & Ors* [2005] ACTSC 74 (5 August 2005) the Full Court of the Supreme Court comprising Higgins CJ, Crispin and Bennett JJ stated in relation to the nature of the Coroner's inquiry:

    "The task of a coroner is not to determine whether anyone is entitled to some legal remedy, is liable to another or is guilty of an offence. The Coroner's task is to inquire into the matters specified in the relevant section of the Coroners Act 1997 and make, if possible, the required findings and any comments that may be appropriate." at [12]

    "The [Coroners] Act is generally concerned with the resolution of relatively straightforward questions such as "what was the cause of this death?" or "what caused this fire?". It does not provide a general mechanism for an open ended inquiry into the merits of government policy, the performance of government agencies or private institutions, or the conduct of individuals, even if apparently related in some way to the circumstances in which the death or fire occurred." at [15]

25. Their Honours go on at [28] to warn coroners against the conduct of 'a wide-ranging inquiry akin to that of a Royal Commission' and provide an example at [31] of the limits of enquiry:

    "... a coroner might well hear evidence suggesting that a cyclist's death had been caused not merely by a collision with a motor vehicle, but also by the antecedent conduct of the driver of that vehicle in failing to stop at a stop sign adjacent to an intersection. However, the limited jurisdiction conferred ... would not authorise the coroner to inquire into any perceived failures in relation to general policy relating to the siting of stop signs or the enforcement of traffic regulations. The particular siting and design of the relevant intersection may be a different matter. The application of the common sense

test of causation will normally exclude a quest to apportion blame or a wide-ranging investigation into antecedent policies and practices."

26. Their Honours endorsed, at [29], the 'common sense' test of causation laid down by the High Court in *March v E & MH Stramare Pty Ltd* (1991) 171 CLR 506, where it was said:

"A line must be drawn at some point beyond which, even if relevant, factors which come to light will be considered too remote from the event to be regarded as causative ... in the context of a coronial inquiry, [the common sense test of causation] may be influenced by the limited scope of the inquiry which, as we have mentioned, does not extend to the resolution of collateral issues relating to compensation or the attribution of blame."

27. Further, in *Harmsworth v The State Coroner* [1989] VR 989 at 997, Nathan J discussed the ambit of the Coroner's power to comment as follows:

"The power to comment arises as a consequence of the obligation to make findings … It is not free ranging. It must be comment 'on any matter connected with the death.' The powers to comment and also to make recommendations … are inextricably connected with, but not independent of the power to enquire into a death or fire for the purposes of making findings. They are not separate or distinct sources of power enabling a coroner to enquire for the sole or dominant reason of making comment or recommendation. It arises as a consequence of the exercise of a coroner's prime function that is to make 'findings.' "

Consideration

28. I have before me a multitude of medical evidence and conflicting opinions, which I am required to resolve as best I can.

29. The first matter I deal with is A/Professor Parnia's comments about the possibility of septic shock. A/Professor Parnia may well have meant those comments as illustrative or explanatory of the medical concept of shock, but I am satisfied that septic shock played no part in Mr Brown's death. There was no evidence at autopsy of infection or sepsis, Mr Brown was afebrile throughout both hospital admissions, and Mr Brown was in any case receiving prophylactic antibiotics.

30. The autopsy evidence suggests that the CABG surgery was conducted appropriately, notwithstanding that Mr Brown developed fibrinous pericarditis (a known complication) as a result; additionally, the anticoagulant treatment succeeded in treating the post-surgery pulmonary embolus. I make no further enquiry or comment in respect of those matters.

31. On balance, I consider the evidence points away from Mr Brown suffering an acute kidney injury as far back as Pambula. I am largely guided in this by the autopsy findings of no abnormalities in Mr Brown's kidneys, noting that Dr Slezak has provided a reasonable alternative explanation that severe lactic acidosis can develop quickly in a setting of profound hypotension and hypoxia.

32. The autopsy evidence suggests that Mr Brown had a deranged liver, which was most likely the cause of his abnormal blood test results from Pambula and early on in his admission at TCH. The fact that A/Professor Jain observed necrosis suggests that the liver injury did not occur in the perimortem period, but was present for some period of time prior to death. However, this does not appear to have been contributory to Mr Brown's death in the legal sense, in that A/Professor Jain has not specified this as a contributor or cause of death. Indeed, A/Professor Jain appears to suggest that this liver damage could be a result of post-cardiac injury syndrome, an immune response by Mr Brown's system to the CABG surgery. While undoubtedly Mr Brown's family will have questions about the cause and effect of this liver damage, and I accept that this would have been a factor bearing on his condition, as the case excerpts I have referred to earlier make clear, further enquiry is beyond the scope of my inquest.

33. I do not consider that there is any deficiency in the treatment that Mr Brown received at PDH at a level where I could be comfortably satisfied that it was causative or contributory of Mr Brown's death. The evidence is clear that the Pambula doctors regularly, and appropriately, consulted cardiac specialists at TCH for advice. The pulmonary embolus was appropriately identified, and treated with the accepted standard treatment regime. When Mr Brown failed to respond to treatment and his condition deteriorated, he was appropriately transferred to TCH. There were no objective symptoms prior to 31 March 2013 which indicated that different treatment was warranted, or that Mr Brown should have been transferred to TCH earlier. I note that should the NSW Coroner take a different view to me and consider that the treatment at PDH warrants further

investigation, then the NSW Coroner could elect to exercise their concurrent jurisdiction.

34. Dr Slezak did note in relation to PDH a potential delay in reducing Mr Brown's anticoagulant medication dosage in the face of a rising INR result. I am however satisfied that this potential delay does not materially bear on Mr Brown's death, given the significant events and treatment which came afterwards. I will in any case direct that a copy of my findings and Dr Slezak's report be sent to PDH for its information and any appropriate action.

35. I also consider, on the balance of probabilities, that the pericardial bleed that caused the tamponade which caused Mr Brown's cardiac arrest was likely an acute, sudden bleed rather than a long, slow bleeding process. I place particular weight on the evidence, as noted by Dr Slezak, that there was no alteration in the cardiac silhouette as between chest x-rays taken on 19 March and 1 April 2013. I also note that by and large, and except for complaints of nausea and slowly rising blood test results, Mr Brown's condition was largely stable.

36. It is in that context I turn to consider whether there was a failure to diagnose cardiac tamponade in Mr Brown's case. As A/Professor Parnia states, cardiac tamponade can be diagnosed by way of ECG or CT scan of the chest. A/Professor Parnia is also correct to note that Mr Brown should have been known to have been at risk of this complication. A/Professor Parnia does not deal with Dr Slezak's comment that serial ECGs of Mr Brown while in TCH did not reveal any evidence of QRS alternans, and I accept Dr Slezak's interpretation of the evidence. Although Mr Spearpoint considers that the 6:06am ECG on 2 April 2013 displayed an abnormal rhythm, A/Professor Parnia does not address this point at all, and both TCH and Dr Slezak suggest that there were no abnormal ECGs. I am satisfied, to the requisite standard, that it was not until Mr Brown's asystole collapse that his objective medical condition should have triggered consideration of this diagnosis. I am also satisfied that it would not have been possible to have diagnosed cardiac tamponade until the period just before Mr Brown's collapse, when it would have been evident, and there were no triggers that should have warranted further investigation prior to collapse.

37. It may have been possible to have diagnosed Mr Brown's fibrinous pericarditis, a form of post-cardiac injury syndrome, prior to his death. If this diagnosis had been made, then perhaps Mr Brown might have been transferred to ICU and an

increased risk of cardiac tamponade might have been recognised. In retrospect, Mr Brown's episodes of poor oxygen saturation were most likely due to this developing pericarditis. However, poor oxygen saturation is a non-specific symptom, and each of those episodes were promptly and appropriately treated and on each occasion his oxygen saturation returned to normal limits for a man of his age including after active treatment was reduced. It would be speculation on my part to make any inference as to whether there would have been a different outcome for Mr Brown had he been taken to ICU at a point prior to his collapse; I am inclined to think not, given my earlier finding that Mr Brown most probably died as the result of an acute bleed. In the circumstances the evidence does not rise where I can properly make an adverse comment or finding against any clinician for failing to diagnose Mr Brown's fibrinous pericarditis.

38. I turn now to consider the appropriateness of the anticoagulant treatment that Mr Brown received and which A/Professor Jain has said was causative of Mr Brown's death. This treatment was started at PDH, on the advice of clinicians from TCH and continued at TCH after Mr Brown was transferred there. It is apparent that when it was commenced Mr Brown needed the treatment for a pulmonary embolus, and that he was given the accepted standard treatment regime. Although by the time of his death Mr Brown's pulmonary embolus had resolved, that is not a matter that was evident to clinicians, or would have been evident without a CTPA. Additionally, while at the time Mr Brown arrested he had an abnormal INR, the previous day's INR result was within appropriate limits. Lifelong anticoagulant therapy is a consequence of CABG surgery in order to prevent further blockages of arteries or the grafts; Mr Brown would never have been completely taken off anti-coagulant medication. I find that it was not unreasonable for clinicians not to have explored whether the pulmonary embolus had dissolved prior to Mr Brown's collapse and subsequent death. I also find that the anticoagulant treatment Mr Brown received was appropriate, even though it unfortunately caused his death. Bleeding disorders are a known and not uncommon complication of long-term anticoagulant treatment. There was no objective evidence given the prior INR was within appropriate bounds that alterations to the medication regime should have been undertaken.

39. In regards to the resuscitation efforts on 2 April 2013, it does appear that what was done and the clinical decisions that were made could have been better documented. There is sufficient detail in the clinical record as to what was done

for me to find that there was no active treatment of Mr Brown's profound acidosis and hyperkalaemia, no defibrillation, nor any attempt at pericardiocentesis. I however agree with Dr Slezak that no adverse criticism can be made in this respect: undoubtedly the clinicians at this time were working to return spontaneous circulation to Mr Brown – which we now know was likely futile without pericardiocentesis, and pericardiocentesis would only have been indicated in the event of a probable diagnosis of cardiac tamponade, which had not been made. The lack of defibrillation and reasons for such again could also have been better detailed in the clinical record, but I cannot speculate that if defibrillation had occurred it would have altered the outcome for Mr Brown. However, I direct that a copy of my findings and A/Professor Parnia's report be sent to TCH for its information and any appropriate action.

40. It follows then from what I have said above that in the circumstances I make no adverse finding or comment against TCH or PDH, or any clinician at those institutions. I am satisfied that no matter of public safety arises in relation to Mr Brown's death. I am not convinced to the requisite degree that Mr Brown's death was preventable in terms of Mr Brown's death being due to failings in medical care or treatment, in the sense that, but for those failures, Mr Brown would not have died. The suddenness of his collapse in conjunction with little overt symptomology suggests that it would not have mattered if Mr Brown was in ICU at the time of his collapse. It would be speculation on my part to say that if pericardiocentesis and/or defibrillation had been attempted during resuscitation Mr Brown would have lived, and to say so would be disregarding Mr Brown's fibrinous pericarditis and his liver dysfunction.

41. I find that:

    Jeffrey Brown died on 2 April 2013 at The Canberra Hospital, Dann Close, Garran in the Australian Capital Territory;

    The manner and cause of Mr Brown's death is haemopericardium, due to anti-coagulation treatment for pulmonary thromboembolitic disease; Mr Brown's recent coronary artery bypass graft surgery with fibrinous pericarditis were significant conditions contributing to death, but not related to the disease or condition causing death; and

    Pursuant to s 52(4)(a)(i) of the *Coroners Act 1997*, no matter of public safety is found to arise in connection with this inquest.

42. Notwithstanding the family submissions, in all the circumstances, in my view there is no utility in or necessity for holding a public hearing in relation to Mr Brown's death. The decision about whether to hold an inquest hearing is a balancing exercise and *"the discretion needs to be approached assessing the strength of available evidence and determining after consideration, whether there would be any benefit in the holding of an inquest and whether it would be expected to yield further information that thus far has not come to light. Any benefit that can be ascertained to flow to the next of kin needs to be considered, particularly if the holding of an inquest will contribute to an important finding. … I do not consider eligibility to obtain victim's compensation to be a proper basis for the exercise of the discretion … an inquest should not be held where it would clearly be a futile exercise"*: see *Taing & Buob v Territory Coroner* [2011] NTSC 58 at [53]. I note here the manner and cause of death of Mr Brown are sufficiently disclosed.

43. I make no recommendations.

44. I direct that these findings be published in due course on the Coroner's Court website. I direct that a copy of my findings and Dr Slezak's report be sent to PDH for its information and any appropriate action. I also direct that a copy of my findings and A/Professor Parnia's report be sent to TCH for its information and any appropriate action.

45. I extend my condolences to Mr Brown's family and friends. I note the amount of time taken to conduct the investigation in relation to Mr Brown's death, although as these findings demonstrate, there was a significant amount of complex evidence to be obtained and considered.

B C BOSS
CORONER