



MAGISTRATES COURT of TASMANIA
CORONIAL DIVISION



**IN THE MATTER OF THE
CORONERS ACT 1995**

AND

**IN THE MATTER OF AN
INQUEST TOUCHING THE
DEATH OF
NEVILLE ROBERT HOSKINSON**

**FINDINGS, RECOMMENDATIONS AND COMMENTS of
Coroner Rod Chandler following an inquest held in Launceston on
6, 7 and 8 February 2017.**

31 March 2017

Introduction

On 17 May 2012 Mr Neville Robert Hoskinson died at the Launceston General Hospital (LGH). The cause of his death was a cardiac tamponade resulting from a proximal aortic dissection due to hypertension. On 6, 7 and 8 February 2017 an inquest was held concerning the death. Its focus was upon Mr Hoskinson's medical care provided by the LGH. These are the findings arising from that inquest.

Background

Mr Hoskinson resided with his wife Leeanne and their son at Waterhouse in North-East Tasmania. He was aged 55 years and was employed as a farm manager. He enjoyed good health although he had a family history of ischaemic heart disease.

Circumstances Preceding Presentation to LGH

In the afternoon of Monday 15 May 2012 Mr Hoskinson telephoned his wife at her workplace to tell her that he had severe chest pain and headache. She arranged for a work colleague to collect her husband from their home and drive him to the Barnboughle Golf Course. There he was met by an ambulance and conveyed to the North East Soldier's Memorial Hospital at Scottsdale arriving at around 4.15pm. He was promptly seen by Dr R Skerka who recorded: *"Acute onset severe (10/10) chest pain, sub sternal with radiation to neck/head/jaw."* He was administered 2 mg of morphine and then returned to the ambulance to be transported to the LGH.

Events at the LGH Preceding Death

Mr Hoskinson arrived at the Emergency Department (ED) of the LGH at 6.05pm on 15 May. He was triaged as a Category 3 patient thereby requiring medical review within 30 minutes. At 6.30pm he was seen by ED consultant, Dr Erica Kreismann. She recorded that he had constant pain, increased with inspiration. He denied shortness of breath, palpitations, visual changes, abdominal pain, nausea, vomiting, diarrhoea or back pain. Her physical examination indicated that he was awake, alert and conversant. She noted no bruits on his neck, his abdomen was soft and non-tender and his heart and lung sounds were normal. Neurological examination was normal. The treatment plan was noted as; *"Pleuritic in nature, chest pain minimal at present, complaining of mild headache, unclear aetiology, will check x-ray, labs, D dimer, Troponin. ECG: normal sinus rhythm, 80, nil acute changes."*

The chest x-ray was reported to show a *"?thickened paratracheal stripe."* The troponin level was raised and a repeat test was ordered for the following morning. The D dimer test (used to rule out the presence of blood clots) was also elevated. Because of this result and the *"questionably abnormal x ray"* Dr Kreismann ordered a CT angiogram of the chest to rule out the possibility of an aortic dissection or pulmonary embolus.

The CT angiogram was carried out by radiologist, Dr Anil Gupta. He dictated a report but this was not typed until the following day. He also reported verbally to Dr Kreismann who then made this entry in the notes:

"CT brain normal."

CT.A chest: stranding in mediastinal fat. Infxn/inflamm/haemorrhage."

At 11.15pm Mr Hoskinson was seen by medical registrar, Dr Suneet Kohli. He made a very comprehensive record of the attendance in the medical notes. It included a history, symptom details and his examination findings. The symptoms, namely chest pain, jaw pain and headache were not, in his view, typical of aortic dissection. His examination findings including a slightly elevated blood pressure, mildly elevated D dimer test and an ejection systolic murmur were also not suggestive to him, either separately or in combination, of an aortic dissection. As to the CT angiogram of the chest he recorded; *“no dissection/PE. Mediastinal fat stranding? Inflammation?? Infection? Haemorrhage (verbal report by Dr Gupta).”* He noted that the *“issue”* was *“Pleuritic chest pain with headache? cause (no significant findings on imaging).”* Dr Kohli was unable to say whether his recording of the term *“no dissection”* in his notes was a reflection of the information on the aortogram conveyed to him by Dr Kreismann or whether it represented his own conclusion based upon the symptoms, his examination and the scan as verbally reported.

The treatment plan settled by Dr Kohli at this time was to admit Mr Hoskinson to the care of the Medical Team for observation with a repeat ECG and a repeat troponin test to be done the next morning. Also in the morning a transthoracic echocardiogram was to be undertaken as advised by the cardiology registrar.

The following day at 9.30am Mr Hoskinson was seen by Drs Griffin and Batta of the Medical Team. They noted that he had *“slight chest pain when breathing in worse when lying flat.”* Of the CT angiogram they noted; *“?mediastinal fat stranding. No dissection.”* It was their impression that Mr Hoskinson was suffering from pericarditis and the treatment plan was then noted as:

- “1. Echo*
- 2. Monitor*
- 3. Cardiology R/V.”*

Sometime on 16 May Dr Gupta’s written report upon the imaging became available. As to the CT angiogram of the chest it states:

“Examination performed for suspected dissection.

Examination shows good flow of contrast through the ascending, arch and ascending aorta. No evidence of dissection seen. No obvious abnormality in aorta or its major vessels noted. There is a good flow of contrast noted through the aorta and its major branches. There is some stranding and increased density of the mediastinal fat seen in prevascular space and aortopulmonary window region. Possible changes of inflammation/haematoma in the mediastinum cannot be excluded completely. Patient should be monitored clinically and followed up.

The remainder of the chest and abdomen included in this study demonstrate no obvious/acute abnormality except for the presence of gallbladder stones.

Impression:

No acute intracranial abnormality seen.

No evidence of dissection seen.

There is stranding of fat in the prevascular space and the aortopulmonary window region noted raising the possibility of inflammation/haemorrhage/haematoma in the mediastinum in the appropriate clinical context.

Note of GB Stones is made.

Clinical monitoring and follow up advised.”

Mr Hoskinson's case notes for 16 May do not contain any entries made by a doctor following the entry made following the review with Drs Griffin and Batta that morning. However, the nursing notes are more forthcoming. They show:

- The repeat troponin test was normal.
- That up to 2.00pm both Mr Hoskinson and the nursing staff were expecting him to undergo the transthoracic echocardiogram.
- That at 2.45pm Mr Hoskinson was reviewed by the Medical Team. This note was made; *“Patient for echo they were rang but don't know anything about it.”* The medical team then determined; *“Patient not for echo now”* and that he instead be observed overnight for pain with the possibility of him being able to be discharged the following morning.
- That Mr Hoskinson reported that he was pain free for the balance of the day.

At 9.30am on 17 May radiologist, Dr R Heng, at the request of the Medical Team, reviewed the CT angiogram carried out by Dr Gupta. They reported on that review:

“(Dr Heng) feels that there are some abnormal findings which were not reported:

- *Ascending aortic aneurysm;*
- *Fat stranding-?blood in the mediastinum and pericardial thickening +/- fluid/blood in pericardial sac suggestive of an aortic leak;*
- *Cannot rule out an ascending aortic dissection.”*

Following Dr Heng's review the Medical Team sought the assistance of cardiologist, Dr Bhuwan Singh. He saw Mr Hoskinson at 10.30 that morning. He arranged for a bedside transthoracic echocardiogram. It showed normal LV function, trivial to mild posterior pericardial effusion, mildly dilated ascending aorta with mild aortic regurgitation. Dr Singh did not consider that these findings enabled a firm diagnosis of aortic dissection to be made. It was then planned for Mr Hoskinson to undergo a transoesophageal echocardiogram (TOE) which he considered to be the most appropriate diagnostic tool to diagnose aortic dissection. However, this could not be done immediately as Mr Hoskinson had had breakfast that morning.

The TOE was carried out by Dr George Koshy and was given urgent priority at the request of Dr Singh. It was commenced at 12.35pm. It identified a dissection flap during sinus valsalva thus confirming the diagnosis of aortic dissection. However, during the course of the procedure Mr Hoskinson's aorta dissected further causing bleeding into the pericardial sac impacting on the heart's ability to beat. A Code Blue was called and resuscitation carried

out. However, Mr Hoskinson could not be revived and he was declared deceased at 12.57pm.

Post-Mortem Examination

This was carried out by State Forensic Pathologist, Dr Christopher Lawrence. In his report he comments:

“Autopsy revealed a proximal aortic dissection with 300 mls of blood in the pericardial sac. The tear is just above the aortic valve. The aortic valve is bicuspid and this may have something to do with the dissection. There is a known association between proximal aortic dissection and bicuspid aortic valve. The proximal aorta does appear slightly dilated.”

In Dr Lawrence’s opinion the cause of Mr Hoskinson’s death was a cardiac tamponade due to a proximal aortic dissection due to hypertension. Cardiac tamponade occurs when blood or fluids collect in the space between the sac that encases the heart and the heart muscle. This exerts pressure on the heart preventing its ventricles from expanding fully thus limiting its capacity to pump blood to the rest of the body. This leads to organ failure, shock and death. In Mr Hoskinson’s case blood from his dissected aorta was the cause for the cardiac tamponade.

Issues Arising

The circumstances surrounding Mr Hoskinson’s death have given rise to multiple issues, most particularly centred upon the failure to make a timely diagnosis of Mr Hoskinson’s aortic dissection and to arrange for its treatment. Those issues concern:

1. The initial interpretation of the CT angiogram.
2. Communication of the results of the CT angiogram.
3. The failure to undertake a transthoracic echocardiogram in response to a direction of a cardiology registrar made on 15 May.

I will deal with each in turn.

Interpretation of the CT Angiogram

A matter closely examined at this inquest was the adequacy of Dr Gupta’s findings upon the CT angiogram performed on 15 May 2012 and in particular whether he should have made the diagnosis of an aortic dissection. Several radiologists gave evidence on this subject and it is necessary that I set out the body of their opinions.

Dr Michael Carr is a consultant radiologist and a former Director of Medical Imaging at the Royal Hobart Hospital. He was retained to provide an opinion upon the CT angiogram and Dr Gupta’s report upon it. Dr Carr provided three reports which were put into evidence. He also gave evidence in person. He opined that:

- The thin slice images show an abnormal dilation of the ascending aorta measuring 5.7cm in diameter (the ‘norm’ is around 4cm) and the descending

aorta is tortuous or elongated. These findings are suggestive of hypertension, a condition which commonly presents in persons who develop a dissection.

- The images show areas of moderately increased attenuation in the anterior mediastinum measuring 25 to 40 Hounsfield units. This is consistent with the presence of blood.
- The images show a Stanford Type A dissection of the antero-lateral aspect of the ascending aorta with leakage of blood into the dissection and leakage of blood into the mediastinum. However, Dr Carr did make this concession; *“I concede that the changes in the ascending aorta wall were difficult to see but were there.”*
- Dr Gupta was correct to report the presence of mediastinal fat stranding and his three possible explanations for this are reasonable.
- The crux of the matter to be in these terms; *“.....even if Dr Gupta had missed everything else in the report he did see the mediastinal abnormality and if he'd measured the density that would have upgraded the whole matter to a vascular emergency and the actual management of Mr Hoskinson would have been completely different.....”*

To summarise, it was the evidence of Dr Carr that Dr Gupta was wrong to report that there was *“no obvious abnormality in the aorta”* and *“no evidence of dissection seen”* because the ascending aorta is abnormally dilated, the descending aorta is tortuous and the diagnosis of an aortic dissection was not made. Too, he says that the conclusion should have been made that the changes in the mediastinum were due to haemorrhage.

Dr Darren Ault is a radiologist who was retained by the Australian Health Practitioners Regulation Agency to review Dr Gupta's report upon the CT angiogram. A written report from Dr Ault was put into evidence. However, Dr Ault did not make himself available for examination and I did not compel his attendance. In his report he states that he did not believe there was evidence of a dissection flap but agreed the images showed an abnormal increased density in the mediastinal flap as reported by Dr Gupta. He noted the dilated ascending aorta which measured at least 5cm. He considered that the finding of an aortic abnormality was subtle and that pulsation artefact significantly contributed to a lack of diagnostic confidence. He also commented; *“The final report of the CT examination is not incorrect but in retrospect there were secondary findings sufficient to raise the possibility of the presence of a dissection of the ascending aorta such that further investigations either with repeat cardiac gated CT or TOE could have been specifically recommended.”*

The other opinion evidence upon the radiology was provided by consultant radiologist, Dr Michael Jones at the request of the Tasmanian Health Service. Dr Jones' evidence can be summarised as follows:

1. He would not make a diagnosis of an acute aortic dissection unless an intimal flap is unequivocally present or, in its absence, there is unequivocal

hyperdense blood in the aortic wall and motion artefact can be excluded as an explanation.

2. Based upon the CT angiogram he would make these diagnoses:
 - a) There is no evidence of aortic dissection.
 - b) There is calcific aortic stenosis of at least moderate severity.
 - c) There appears to be oedema in the superior mediastinum which may be due to infective or noninfective inflammatory disease or trauma.
 - d) There is no evidence to suggest it is due to arterial bleeding.
 - e) The changes in the aorta wall reported upon by Dr Carr as indicating a dissection were all attributable to motion artefact.
 - f) That the findings with respect to the mediastinum and the pericardial sac cannot be assumed to be attributable to blood as a definite pathway for such blood to reach these compartments is not apparent. These findings therefore cannot be said to support a diagnosis of an aortic dissection.
 - g) That Dr Gupta's report upon the angiogram was reasonable and in accord with competent professional practice.

Finally, I have already referred to Dr Heng's review of the aortogram made on 17 May which, in his opinion, identified some abnormal findings and did not enable the diagnosis of an ascending aortic dissection to be ruled out. Because of ill-health Dr Heng was unavailable to give evidence.

The factor common to these expert opinions is the absence of unanimity. They demonstrate that radiology is far from an exact science and in cases such as this, where a diagnosis is not plainly evident, experienced practitioners can make significantly different interpretations of the same images. This leads me to conclude that the evidence does not permit me to make a positive finding that Dr Gupta should have made a diagnosis of an aortic dissection following the CT angiogram of 15 May 2012. However, the weight of evidence is, in my view, sufficient to satisfy me that that the angiogram did demonstrate, to adopt Dr Ault's phrase, "*secondary findings*" which raised the possibility of a dissection and which required further investigation by the clinicians caring for Mr Hoskinson. This brings me to consider issues related to the communication of Dr Gupta's findings upon the angiogram and the actions taken.

Communication of the CT Angiogram Findings

The angiogram in question was carried out by Dr Gupta in the evening of 15 May and outside of normal working hours. This meant that there was not any administrative staff available to type up the report after it was dictated by Dr Gupta and to enter it into the

Picture Archiving and Communication System (PAC) so that it could be readily accessed and considered by those persons treating Mr Hoskinson. In the result all communication in the initial stages concerning the results of the scan had to be verbal. I need to consider the consequences which flowed from this state of affairs.

I have set out Dr Kreismann's note of her conversation with Dr Gupta. The first observation to make is that she has recorded infection/inflammation/haemorrhage to explain possible changes shown on the scan in the mediastinum when Dr Gupta's written report makes no reference to infection but instead attributes the possible changes to inflammation/haemorrhage/haematoma.

What of the communication around the possible presence of an aortic dissection? A stated purpose of the CT angiogram was to rule out this possibility and the note made by Dr Kreismann of her conversation with Dr Gupta does not show it to be ruled in or out. In fact it makes no reference to it at all. This is contradicted by subsequent entries in the records with Dr Kohli's notation indicating that Dr Gupta had verbally reported "*no dissection/PE*" and the replication by the Medical Team when they recorded the CT angiogram to show "*no dissection.*" There is no evidence to explain how the Medical Team came to make its entry. As I have already noted Dr Kohli explained his entry as perhaps being either a record of the information passed onto him by Dr Kreismann or a reflection of his own opinion of Mr Hoskinson's status based upon the symptoms, his examination and the radiology. In my view the former explanation is more likely given that it is written alongside the phrase 'CT of the chest' and is followed by a further description of Dr Gupta's interpretation of the scan as recorded by Dr Kreismann.

Finally, I need to make comment upon some specific evidence from Dr Kohli. He told the inquest that if he had had access to Dr Gupta's written report when he saw Mr Hoskinson he would have sought further information from Dr Gupta around the sentence; "*Possible changes of inflammation/haematoma in the mediastinum cannot be excluded completely.*" Particularly he would have been anxious to have an understanding of the likelihood of any blood being in the mediastinum as this would indicate a serious medical condition requiring an urgent response. If Dr Gupta was unable to be more informative then he would have enquired of other radiological investigations that he would recommend to assist with a diagnosis.

There are, in my view, several conclusions to be drawn from the foregoing. The first is the increased risk of misunderstanding, misinterpretation or wrongful emphasis which can arise from a verbal report upon radiology which in this case is evidenced by:

- Dr Kreismann's apparent understanding that infection was a possible explanation for changes shown in the mediastinum when this was not shown to be so by Dr Gupta's written report. It's noted that Dr Kohli, in his written record, repeated the reference to 'infection.'
- Dr Kreismann's failure to record haematoma as a possible explanation for changes shown in the mediastinum contrary to the written scan report.

- The inconsistency between Dr Kreismann's note which makes no reference to dissection and the written report which stated; "*No evidence of dissection seen.*"
- The inconsistency between Dr Kreismann's note of the scan results and the subsequent notes made by Dr Kohli and the Medical Team which record "*no dissection.*"
- The contrast between the positive assertion recorded by Dr Kohli and the Medical Team of "*no dissection*" and the less definitive statement made by Dr Gupta in his report of "*No evidence of dissection seen.*"
- The absence in Dr Kreismann's report of any reference to the possibility of blood in the mediastinum as described in the written report.

The second conclusion concerns the consequences which can follow when a clinician does not have access to a full written report upon the radiology. Here, as I have noted, it was the evidence of Dr Kohli that if he had had access to Dr Gupta's written report and its reference to the possibility of blood in the mediastinum he would have sought further information of Dr Gupta. A likely outcome of this enquiry would have been Dr Gupta's reinforcement of the decision, already taken by the cardiology registrar, for Mr Hoskinson to undergo a transthoracic echocardiogram.

Dr Kohli impressed me as careful, conscientious and able physician who made meticulous patient notes. I have no doubt that the outcome of any discussion he had with Dr Gupta would have been fully recorded in the notes including any advice concerning an echocardiogram. It may be speculative to say it but if such an entry was made it is likely in my view that it would have militated against the decision later taken by the Medical Team to abandon the request for a transthoracic echocardiogram.

The Delayed Transthoracic Echocardiogram

When Dr Kohli saw Mr Hoskinson at 11.15pm on 15 May he recorded that part of Mr Hoskinson's treatment plan was for a transthoracic echocardiogram to be carried out in accord with the advice of a cardiology registrar. That advice was acted upon the following day by the Medical Team via a referral document which is on Mr Hoskinson's file. It is dated 16 May 2012 and signed by a Dr Jackson as the requesting doctor and by Dr Griffin on behalf of the Team consultant. The time that this document was created is not apparent but the relevant nursing notes which I have already referred to strongly suggest it to have occurred that morning.

It is clear that despite the referral document the echocardiogram had not been done by 2.30pm on 16 May and it has not been possible to establish how this came about. All that is evident is that at that time the Medical Team reviewed Mr Hoskinson, learned that the echocardiogram had not been done and then cancelled the referral.

Dr A J Bell is a specialist intensivist and a former Chief Medical Officer at the Royal Hobart Hospital. He provides advice to the coroner's office upon medical issues. In this instance it was his evidence that the factors warranting an echocardiogram, as identified by Dr

Kreismann and confirmed by Dr Kohli, had not changed and the decision to cancel the procedure was unwise and flew in the face of standard medical practice. In his further view the echocardiogram should have been undertaken immediately. I accept this evidence from Dr Bell.

It was the next day, as I have recorded, that Dr Singh promptly organised a transthoracic echocardiogram following Dr Heng's review of the CT angiogram. When its results did not, in Dr Singh's opinion, permit a diagnosis of aortic aneurysm to be made he arranged for an urgent TOE. However, it was not until 12.35pm that this procedure commenced.

In the result a procedure which began just beyond noon on 17 May would, in all likelihood, have been undertaken a full day earlier if the request completed by Dr Jackson had been acted upon. This raises the question whether the outcome for Mr Hoskinson would have been different if the TOE had been undertaken a day earlier and the aortic dissection diagnosed.

Some Findings upon the LGH's Care and Treatment

I am satisfied that Mr Hoskinson received proper and appropriate care and treatment when he presented in the ED on 15 May and no criticism is made of those practitioners who oversaw him at this time, namely Dr Kreismann and Dr Kohli. Of the latter I commend him for the medical notes made of his attendance upon Mr Hoskinson. They are of a standard rarely seen by me and would make a suitable benchmark for all other practitioners. I have previously set out the competing interpretations made of the CT angiogram. For the reason stated I cannot be satisfied that Dr Gupta's failure to make a diagnosis from the scan of an aortic dissection mandates criticism. However, some comment is necessary concerning the reporting of the scan.

As I have noted the LGH did not in 2015 have the capacity to provide contemporaneous written reports upon radiology performed out-of-hours. In the result Dr Gupta had to provide Dr Kreismann with a verbal report on Mr Hoskinson's CT angiogram. I have set out the difficulties that arose in this case which, in my view were directly attributable to the unavailability of a written report. Ordinarily this circumstance would have led me to recommend that the LGH implement a system change to ensure the immediate availability of written radiology reports. (Such a recommendation was in fact made by me in March 2015 following an inquest into the death of Donald John Clarke). However, at this inquest evidence of the current situation upon this subject was provided by Mr Garth Faulkner, the LGH's Chief Radiographer. It was his evidence that in October 2016 the hospital's radiology services were outsourced to a private provider. He said that it was a term of the agreement with the provider that all CT and MRI scans taken of patients in ED, whether during working hours or outside of those hours, required a written report to be provided within one hour of the procedure being completed. This makes otiose any repeat recommendation by me concerning radiology reports.

I now return to the difficulties attributable to the absence of a written report on the scan. I accept Dr Kohli's evidence that if Dr Gupta's written report had been available to him he would have consulted with him concerning the possibility of blood being in the mediastinum. As I have already noted this would, in all likelihood have led to him recording in the medical notes detail of his conversation including advice upon the further radiology required. Such

an entry should have helped to dissuade the Medical Team from its decision, made in the afternoon of 16 May, to abandon the echocardiogram.

Mr Hoskinson had been scheduled to undergo a transthoracic echocardiogram, at least by late morning on 16 May. It did not occur because of an unexplained administrative bungle. The Medical Team, in my view, was in error for the reasons stated by Dr Bell, in cancelling the request for the echocardiogram. It was only after Dr Heng's review of the CT scan the following morning that an echocardiogram was re-ordered. It was then done promptly. However, it was in my view approximately 24 hours later than a proper level of care required.

I accept the evidence of Dr Singh that the results of the echocardiogram were not sufficiently conclusive to make the diagnosis of an aortic dissection and thus commit Mr Hoskinson to major cardio-thoracic surgery. I accept too the evidence that it was appropriate to proceed to a TOE. However, it was delayed by several hours because Mr Hoskinson had been permitted to have breakfast. When it did take place his aorta further dissected leading to his cardiac tamponade and death. I need to record that it was the evidence of Dr Singh, which I accept, that the cardiac tamponade was not attributable to the procedure but was co-incident to it.

The foregoing raises the question whether Mr Hoskinson's death would have been avoided if the diagnostic procedures had been undertaken a day previously and the diagnosis made of an aortic dissection. It was the evidence of both Dr Bell and Dr Singh that had the diagnosis been made one day earlier Mr Hoskinson would have required urgent and major surgery but that he had a 60% prospect of surviving and resuming an ordinary life. I accept this evidence. It leads me to conclude that because of those shortcomings in Mr Hoskinson's hospital management which I have set out, the diagnosis of aortic dissection was delayed by approximately 24 hours and that as a result of that delay he was denied that 60% prospect of survival.

Findings Required by s28 of the Coroners Act 1995

In compliance with this s28 (1) I formally find:

- a) The identity of the deceased is Neville Robert Hoskinson.
- b) The death occurred in the manner and circumstances detailed in these findings.
- c) The cause of death was a cardiac tamponade due to a proximal aortic dissection due to hypertension.
- d) Death occurred at the LGH in Launceston on 17 May 2012.

The circumstances of the death do not, in my opinion, require me to make any comment or any recommendation as permitted by s28 (2) and (3).

Concluding Comments

I extend my sincere condolences to Mr Hoskinson's wife, family members and loved ones. Particularly, it is hoped that this inquest and the participation in the process by Mrs Hoskinson has been of some assistance in helping her to cope with this tragedy.

Finally I acknowledge the excellent work of counsel-assisting, Mr C N Dockray and coroner's associate, Sgt Hamish Woodgate. Their efforts in preparing and presenting the evidence enabled the hearing to proceed smoothly and efficiently and was of considerable assistance to me.

Dated: 31st day of March 2017 at Hobart in the State of Tasmania.

Rod Chandler
Coroner