QUEENSLAND COURTS

CORONERS COURT OF QUEENSLAND

FINDINGS OF INVESTIGATION

CITATION:	**Non-inquest findings into the death of YC**
TITLE OF COURT:	Coroners Court
JURISDICTION:	BRISBANE
DATE:	29/08/2017
FILE NO(s):	2015/200
FINDINGS OF:	Ainslie Kirkegaard, Coronial Registrar
CATCHWORDS:	CORONERS: Elective bronchoscopy, bridging anticoagulation, patient history transcription error by admitting respiratory team, pulmonary haemorrhage, anthraco-silicotic lung disease

Table of Contents

Background

YC was an 87 year old man who died at a tertiary hospital on 10 January 2015. He ordinarily resided with his family.

Mr C's death was reported to the coroner as he died several days after an elective bronchoscopy and biopsy to investigate a recently found lung lesion.

Mr C's medical history

Review of Mr C's medical records shows he had significant cardiac history that included a previous aortic valve replacement, atrial fibrillation for which he was taking warfarin, myocardial infarction and congestive cardiac failure.

Mr C received a tissue aortic valve replacement in July 2010. This surgery was performed at the tertiary hospital. A 23mm Mosaic Medtonic Bioprosthesis was inserted. There is reference to the tissue valve throughout the hospital chart in 2010.

On 13 October 2010, consultant cardiologist, Dr W, wrote to Mr C's general practitioner noting that Mr C was prescribed warfarin for his atrial fibrillation. Dr W was happy for Mr C to revert to aspirin if he experienced any problems with bleeding tendencies.

On 23 December 2014, Mr C presented to the tertiary hospital emergency department on referral by his general practitioner with a presumptive diagnosis of a respiratory neoplasm. Mr C had undergone a chest x-ray and CT scan of the chest the previous day to investigate his non-resolving pneumonia. The CT scan revealed a 5cm x 6.2cm lesion in the upper lobe of the left lung at the mediastinal margin encasing the aorta, and enlarged lymph nodes. There were also multiple low-density liver lesions. Ultrasound scan of the abdomen performed privately on 23 December demonstrated the liver lesions were cystic rather than metastatic disease.

Mr C was referred to the respiratory team and reviewed by the on-call Respiratory Registrar, Dr E. I note that documentation of this review incorrectly refers to Mr C having a mechanical aortic valve replacement. The notes contain no explanation for Dr E's belief that the valve was mechanical. Dr E considered the radiological findings to be consistent with pulmonary malignancy and recommended a diagnostic bronchoscopy.

Mr C was discharged home from the emergency department and scheduled for admission on 5 January 2015. He was instructed to cease his warfarin on 2 January with the intention of commencing heparin bridging therapy on admission prior to an elective bronchoscopy scheduled for 8 January.

The Respiratory Booking Form completed on 24 December 2014 indicates that warfarin was to be ceased five days prior to the bronchoscopy.

Admission to the tertiary hospital for elective bronchoscopy

Mr C was admitted under the respiratory team on 5 January 2015 under the care of consultant respiratory physician, Dr H. The admission notes document a three month history of productive cough, right sided pleuritic pain, weight loss, decreased appetite and decreased exercise tolerance. There is subsequent mention of a three month history of haemoptysis in a further entry dated 6 January.

I note the medical review again incorrectly mentions "mechanical AVR (severe AS) warfarinised" as part of Mr C's background medical history. He noted to have ceased the warfarin on 2 January as planned.

On admission, his APTT was normal at 31 and INR was still elevated at 1.9 despite him having ceased the warfarin 2-3 days previously. His APTT was rechecked at 8:30pm with the result of 70 seconds and again at 2:55am with the result of 60 seconds, both slightly below the desirable range for heparin infusion.

Through an interpreter, he reported daily haemoptysis for the past three months. He was consented for a bronchoscopy +/- biopsy. As part of the consent process, the complications of sore throat, infection, haemorrhage, pneumothorax and death (quoted as 1 in 10,000) were specifically noted and documented. The notes also document discussion about the heparin infusion as necessary for bridging anticoagulation while his warfarin was ceased. His APTT on 6 January was noted to be within the desired range of anticoagulation at 95 seconds.

In preparation for the bronchoscopy, Mr C underwent further CT imaging and respiratory function tests the next day, 7 January. His spirometry was within normal limits indicating normal lung function. A PET scan revealed findings considered to be consistent with metastatic malignancy.

On 7 January, the evening prior to the bronchoscopy, Mr C's INR was checked and noted to be 1.6 (acceptable prior to a procedure). It was planned to cease his heparin infusion early the next morning.

The bronchoscopy

A bronchoscopy was performed on the morning of 8 January, with Mr C first on the list. Bronchial washings, brushings and bronchial biopsy were performed in the left lung at the left lower lobe take off – the area where there was splaying noted of the structure where the airway divides between the left upper lobe and left lower lobe. This was thought to represent submucosal tumour infiltration and had friable overlying mucosa which bled easily. Mr C also had an endobronchial ultrasound guided biopsy of mediastinal and hilar nodes.

The procedural report for the endobronchial lymph node biopsy describes a quite difficult procedure with a thickened airway wall making it difficult to pass the needle and biopsy smaller nodes. Nodal biopsies were performed from the right hilar node (with two passes with difficulty), left para tracheal node (with four passes and difficult to access through the airway wall) and left lower lobe (a single pass of the needle was made into the left hilar region documented as "attempted to visualise left hilar region, though difficult to pass scope into LLL due to airway narrowing at this site. 1 pass only of tissue ?atelectic lung"). A cytologist was present and could not identify expected tissue from lymph nodes.

Mr C returned to the ward at 11:30am.

At 1:30pm he had a temperature of 37.4 degrees and recorded blood oxygen saturations of 88%, with an elevated heart rate of 120.

By 1:50pm, he had spiked a temperature of 38.2 degrees and his blood oxygen saturations were 92% on nasal prong oxygen.

He was recommenced on the heparin infusion at 2:30pm (and this continued while the warfarin was recommenced).

Mr C was medically reviewed by the Respiratory Resident, Dr H, at 2:50pm for a temperature of 39 degrees. Mr C's daughter was present during the review and helped interpret.

A chest x-ray performed at 4:30pm showed a new opacity in the right mid and lower zones of the chest (the other side to where the samples were taken during the procedure that morning). It was thought that this finding and the temperature could be related to residual fluid from the collection of bronchial washings taken during the procedure that morning. Mr C's antibiotics were withheld at this point unless there were further temperatures. His temperature subsequently came down and he was noted to improve.

An APTT taken at 5:20pm was still normal at 35 seconds.

Mr C's post-procedural course from 9 January

Mr C was reviewed by respiratory consultant, Dr G during the medical ward round the next day, 9 January, with an interpreter present. The treating team spoke to Mr C about the challenges experienced during the procedure and the likely diagnosis of a lung cancer with probable metastatic disease (in the lymph nodes and the adrenal gland), which would not be curable. It is documented that Mr C indicated he did not want treatment and asked about the dying process. This prompted a discussion about the availability of palliative care services.

Mr C reported ongoing haemoptysis since the bronchoscopy. He otherwise appeared quite well at that time. He was afebrile and his oxygen saturation was 100% on room air. It was decided to continue the heparin infusion until the warfarin was therapeutic. The plan was for Mr C to be discharged as soon as the heparin infusion could be discontinued.

Nursing notes made at 2:05pm that day document a low grade temperature of 37.4 degrees for which he was given paracetamol by a nurse.

Mr C was reviewed by the respiratory team, including Dr E, at around 5:00pm that day for decreased oxygen saturations and shortness of breath. The nursing notes document that Mr C's oxygen saturation was 87% on room air at 4:00pm. He was commenced on 2L per minute nasal oxygen but deteriorated over a 10-30 minute period with increasing hypoxia (his oxygen saturation remained at 87% on supplemental oxygen). He was breathless and sweaty. Auscultation of the chest detected both wheezes and crackles, concerning for the presence of pulmonary oedema. Mr C was tachycardic with a heart rate of 100-115 beats per minute with non-specific ST changes on ECG.

An urgent chest x-ray revealed an increase in the right sided opacification, increased hilar opacification and left lung infiltrates. The clinical impression was that Mr C was developing acute pulmonary oedema on the background of a new lower respiratory tract infection. He was transferred to the Respiratory High Dependency Unit and treated for both infection and pulmonary oedema with intravenous antibiotics and diuretic therapy. He required an inspired oxygen fraction of 80% to maintain oxygen saturations.

When reviewed again by Dr E at 6:45pm, Mr C's condition was noted to have improved. His oxygen saturation was 95% on an inspired oxygen fraction of 75% - this improvement was thought to be related to the diuresis of 400ml. Following discussion with Mrs C and the couple's daughter, an Acute Resuscitation Plan was completed which facilitated the provision of antibiotics, monitoring, intravenous fluids and supportive therapy but not non-invasive ventilation, intubation or CPR. This reflected Mr C's wishes, as expressed through his family.

When reviewed that afternoon, Mr C was noted to have haemoptysis described as “normal for pt -> daily small vol. Exacerbated now with heparin”. This was not entirely new – he had been coughing small amounts of blood in the last few months. When medically reviewed at around 10:00pm that evening, Mr C was noted to feel better and more comfortable. Small volume ongoing haemoptysis of fresh blood is noted at this time.

Nursing notes document the commencement of a haemoptysis chart overnight with three episodes, each one teaspoon in size, ranging from pink to dark red with a total volume of approximately 25mls. This prompted a request for medical review performed by the onsite Medical Registrar at around 6:30am – 7:10am the next morning, 10 January. By this time, the volume filled half a specimen container. Following discussion with the Respiratory Registrar, the Medical Registrar was not concerned about the haemoptysis at this stage and no further intervention was considered necessary.

When reviewed by the Respiratory Medical Registrar on the morning ward round at around 8:30am, Mr C’s inspired oxygen fraction was 70%. The clinical impression was that Mr C had developed right middle lobe pneumonia and had suffered from a myocardial infarction (in the setting of infection and post-bronchoscopy with associated pulmonary oedema). The plan was to cease the heparin infusion because the warfarin was therapeutic (INR 2.2), continue the intravenous antibiotics, aim for oxygen saturations above 90% and monitor the haemoptysis. A referral was made for cardiology review as there was a significant increase in troponin level (0.26 from a baseline of 0.041), concerning for an acute myocardial infarction (heart attack).

Mr C developed respiratory distress over the following 2-3 hours triggering a medical emergency team call at around 11:45am. His oxygen saturations had dropped to 84% on 100% supplemental oxygen. A chest x-ray showed a large left pleural effusion with increased collapse and consolidation of the left lung and increased airspace changes in the right lung. He was noted to be critically unwell with hypoxic respiratory failure thought secondary to a combination of infection and left lung collapse possibly caused by endobronchial obstruction post-bronchoscopy. His condition was discussed with Dr E and Dr G.

Mr C’s family were informed of his current condition and poor prognosis. They were talked through the options of active treatment versus palliation. During this discussion Mrs C asked whether the bleeding was caused by the bronchoscopy – she was told it was possible and that the bleeding was likely made worse by the heparin/warfarin but the anticoagulation could not be ceased because of the aortic valve replacement. The family advised they wanted to continue all active ward based treatments but Mr C was not for chemotherapy/radiation or aggressive measures as per the Acute Resuscitation Plan.

Mr C was reviewed by a Cardiology Registrar at around 4:00pm that day. This doctor was the first clinician to correctly note the presence of a tissue (bioprosthetic) aortic valve (as supported by reference to an echocardiogram from 2012) and that the indication for Mr C’s prior anticoagulation was atrial fibrillation (rather than a mechanical valve). A haemoptysis volume overnight from 9-10 January of up to 100mls was documented at the time of this review.

The Cardiology Registrar considered that Mr C was in atrial fibrillation in the context of being critically unwell with infection and decompensated heart failure in the context of acute on chronic kidney disease (this explaining this troponin rise). The dyspnoea was considered to be multifactorial but more likely respiratory related though heart failure was contributing. The Cardiology Registrar made a suite of medical management recommendations including that

in view of the haemoptysis, anticoagulation be withheld and aspirin be considered when safe from a respiratory perspective.

This was discussed with the Respiratory Registrar who noted that given Mr C's aortic valve replacement was tissue and not mechanical, the risk of bleeding outweighed the clinical indication for anticoagulation. The respiratory team then attempted to reverse the anticoagulation with intravenous vitamin K.

Mr C continued to deteriorate. He was reviewed by the onsite Medical Registrar at 7:30pm who discussed his condition with Dr E. Following extensive discussions with the treating team that evening, the family chose to continue with active management. Mr C died at 10:00pm on 10 January 2015. His death was attributed to hospital acquired pneumonia.

Preliminary independent clinical review

Preliminary review of the hospital records by an independent doctor from the Department of Health Clinical Forensic Medicine Unit identified concerns regarding the management of Mr C's anticoagulation which may have unnecessarily exposed him to a greater risk of pulmonary haemorrhage following the bronchoscopy. The final pathology of the lung lesion was not available at this time. Further, in the context of Mr C being an anticoagulated patient who had a sudden respiratory decline, increasing haemoptysis, bilateral chest infiltrates, left lung 'white-out' on chest x-ray and death within two days of a bronchial biopsy of a lesion, the possibility of pulmonary haemorrhage as the cause of death could not be excluded. For these reasons, I declined to authorise the issue of the proposed cause of death certificate and proceeded to autopsy and further coronial investigation.

Autopsy findings

An external examination and full internal autopsy were performed at the John Tonge Centre on 20 January 2015. The final autopsy report was issued on 24 February 2016.

The pathologist noted that pathology from the left hilar endobronchial biopsy and brushing of the left lower lobe washing was reported as having no evidence of malignancy in the biopsy material. The biopsy showed superficial fragments of bronchial mucosa only. Microbiology testing of specimens of left lower lobe washing revealed no concerning infecting organisms and cytology indicated there was inadequate material and no evidence of lymph node sampling in the biopsy.

The autopsy revealed evidence of haemorrhage into the airways (bronchi and bronchioles) and lung tissue (parenchyma) with chronic anthraco-silicotic lung disease, complicated by pneumonia with:

- a 6cm x 2cm x 2xm haemorrhage within the left anterosuperior mediastinum
- haemorrhage without infection was present within the left lower lobe, right lower lobe bronchus, right lower lobe, anterior left upper lobe, posterior right middle lobe and posterior right lower lobe
- diffuse pneumonia within the anterior right middle lobe
- organising pneumonia within the left lower lobe
- a firm fibrotic lesion measuring approximately 10cm x 4cm x 4cm in the upper section of the left lower lobe of the lung
- a lymph node adjacent to the left adrenal gland contained a silicotic nodule with anthracotic pigment
- extensive silicotic sclerotic change in carinal lymph nodes

There was no macroscopic or microscopic evidence of lung malignancy or metastatic lesions. There was also severe coronary atherosclerosis but no evidence of acute myocardial infarction. Microbiology testing grew two bacterial species (Citrobactor freundii and Acinetobactor complex) in the blood. No microorganisms were identified in lung cultures. No respiratory virus nucleic acids were detected.

Taking these findings into account, the pathologist considered the death resulted from endobronchial and pulmonary haemorrhage secondary to pneumonia complicating chronic anthraco-silicotic lung disease. I note the pathologist's opinion that the effects of anticoagulation may also have played a role in causing the haemorrhage.

The autopsy finding of chronic anthraco-silicotic lung disease has been notified under the reporting requirements of the *Coal Mining Safety and Health Act 1999* and Coal Mining Safety and Health Regulation 2001.

Tertiary Hospital clinical review outcomes

Mr C's clinical management was reviewed by Dr CH in his capacity as Director of Respiratory Medicine at the tertiary hospital. Dr H clarified that he was not directly involved in Mr C's care. Dr H's report was provided to me on 12 March 2015. The final autopsy report was not available at the time of Dr H's review.

Dr CH observed that:

- Mr C's APTT prior to the procedure performed on 8 January 2015 was normal at 32 seconds, indicating that the heparin infusion had been discontinued long enough to reverse the anticoagulation
- while the mucosa was said to be friable and "bled easily", there was no significant bleeding during the procedure
- there were no immediate complications from the endobronchial ultrasound guided transbronchial biopsies, in particular, no immediate bleeding
- subsequent to the procedure, all biopsy samples failed to confirm the presence of malignancy despite the clear presence of pathology on the radiological imaging
- the left lung radiological appearance on the chest x-ray performed on the afternoon of 8 January 2015 was identical to that on the chest x-ray performed on 20 December 2014
- Mr C's APTT taken nearly three hours after the heparin infusion was recommenced after the procedure on 8 January was still normal at 35 seconds
- the APTT taken on the morning of 9 January was within the desirable range of 88 seconds
- the APTT taken later that day after Mr C's respiratory status deteriorated was 85 seconds, again within the desired range for intravenous anticoagulation.

Dr CH's review squarely acknowledged the failure to recognise Mr C had a bioprosthetic valve rather than a mechanical valve led to unnecessary bridging with intravenous heparin before and after the procedure. He attributed this error to an incorrect assumption by the treating team that the fact of Mr C having had an aortic valve replacement and was on warfarin meant the valve was mechanical. In reality, the indication for the warfarin was Mr C's atrial fibrillation rather than the valve replacement.

However, Dr H did not go further to make any recommendations or report any actions taken by his department to reduce the risk of future error leading to unnecessary anticoagulation.

Dr H took the opportunity to review the radiology series before and after the procedure. He noted that Mr C did have a temperature and pulmonary infiltrate shortly after the procedure;

however, Mr C subsequently improved to the point that he had absolutely normal gas exchange. He then deteriorated again approximately 30-36 hours post-procedure. Dr H considered there were definite clinical and radiological features of pulmonary oedema at this time and Mr C did show some clinical response to diuretic therapy. The most likely diagnosis was pulmonary oedema complicating pneumonia which in turn may have been related to the procedure. Dr H identified a slight delay in commencing antibiotics (not started on the day of procedure) but did not consider this to be clinically inappropriate as the antibiotics were commenced on 9 January following "mild" clinical deterioration and the radiological progression.

Dr CH considered the potential role of unnecessary anticoagulation in Mr C's post procedural deterioration and death. Without the benefit of the autopsy findings at this time, he did not consider Mr C's death to have been caused by excessive intrapulmonary bleeding because:

1. the pulmonary infiltrates initially appeared in the right chest (whereas the bronchoscopy samples were all collected from the left lung) and the radiological appearance of the left lung on chest x-ray performed on the day of the procedure was completely unchanged from before the procedure making the possibility of intrapulmonary bleeding very unlikely;

2. while Mr C did have haemoptysis post-procedure, he had reported daily haemoptysis for three months prior to the procedure; and

3. the subsequent radiological changes did involve the left lung but did not occur until more than 24 hours after the procedure. The final chest x-ray showed significant collapse and consolidation in the left lung but Dr H considered this unlikely to be related to endobronchial obstruction from bleeding. This is because firstly, Mr C was anticoagulated making the formation of an endobronchial blood clot less likely and secondly, a far greater volume of haemoptysis than was documented in the chart would be expected in the presence of significant pulmonary haemorrhage due to anticoagulation.

Further independent clinical review

Having considered the autopsy findings and Dr H's review, the reviewing doctor provided further advice and opinion about the role of unnecessary anticoagulation in Mr C's death. This report issued on 17 February 2017.

The reviewing doctor noted Dr CH's confirmation that an incorrect assumption of a mechanical aortic valve was made by the respiratory team. The anticoagulation pathway that followed would be entirely appropriate if this was indeed the case.

The reviewing doctor explained that a patient with atrial fibrillation is often placed on warfarin to thin the blood and reduce the risk of stroke due to clots originating in the fibrillating atrial chambers. A warfarinised patient with atrial fibrillation will typically have their warfarin ceased five days prior to an elective procedure. The warfarin is then recommenced after the procedure when adequate haemostasis is achieved.

Bridging anticoagulation is the prescription of a shorter-acting anticoagulant (usually heparin) during the period of interruption of the longer-acting agent (usually warfarin) in an attempt to prevent the formation of clots (thromboemboli).

The reviewing doctor advised that current clinical guidelines for stroke prevention in non-valvular atrial fibrillation recommend bridging anticoagulation with heparin only in patients at high risk of thromboembolism. This would include those with severe mitral valve stenosis, mechanical mitral prosthesis or moderate-severe left ventricular valve dysfunction. Bridging anticoagulation (usually in the form of heparin) may be given during this period of interrupted warfarin treatment but this is associated with nearly triple the risk of major bleeding and no significant benefit with regards to preventing stroke.

The reviewing doctor explained that atrial fibrillation patients at very high risk of thromboembolism in the perioperative period may be identified by a scoring system based on the presence of congestive heart failure, hypertension, age greater than 75 years (2 points); diabetes mellitus, prior stroke or transient ischaemic attack or thromboembolism (2 points); vascular disease (peripheral artery disease, myocardial infarction or aortic plaque), age 65-74, sex category female.

Applying this to Mr C, the reviewing doctor calculated his score as 5, compromised as follows:

- congestive cardiac failure (score 1)
- hypertension (score 1)
- age >75 (score 2)
- diabetes mellitus absent (score 0)
- prior stroke/transient ischaemic attack/pulmonary embolism absent (score 0)
- vascular disease (score 1)
- male (score 0)

While this score would place Mr C into a high perioperative thrombotic risk category, bridging anticoagulation is usually recommended only in those with a very high thromboembolic risk, for example, mechanical heart valve or recent stroke, because of the increased bleeding risk with no reduction in rate in thromboembolism.

With reference to Medical Journal of Australia (MJA) consensus guidelines for warfarin therapy, the reviewing doctor advised that the absolute daily risk of a serious thromboembolic event is small in most people with atrial fibrillation, meaning it is safe to stop warfarin for several days before and after surgery, with high dose heparin cover rarely indicated due to the increased risk of bleeding.

Having regard to Mr C's higher calculated risk of thromboembolism, the reviewing doctor acknowledged that bridging anticoagulation could potentially have been justified on the basis of numbers alone. However, it is clear from the medical record that no risk assessment was performed by the respiratory team to deem him to be a very high risk atrial fibrillation patient who required bridging anticoagulation. Rather, the decision to prescribe bridging anticoagulation was based on an incorrect assumption.

It is trite to say that accurate identification of the nature of a prosthetic valve is absolutely crucial to avoid introducing the unnecessary risk of life-threatening peri-procedural haemorrhage. This was a basic and potentially outcome changing error for Mr C who went on to succumb to a catastrophic inflammatory pulmonary haemorrhage.

The reviewing doctor noted the autopsy finding of infection in Mr C's lungs as well as bacteria cultured from his blood. Blood within the lungs may result in inflammation, collapse and infection; conversely, infection within the lungs may result in haemorrhage. It is also possible that bleeding from the friable, recently biopsied mucosa could have been aspirated into the right lung, prompting an inflammatory process with subsequent collapse and intervening infection.

While I accept that it is not possible to determine whether the pneumonia came before, after or at the same time as the haemorrhage, I cannot be satisfied that the unnecessary anticoagulation played no role in Mr C's death.

Knowledge of the actual clinical indication for anticoagulation is imperative in planning peri-procedural anticoagulation. In Mr C's case, this information was readily available to the respiratory team given he had been admitted to the tertiary hospital on multiple previous occasions, had undergone cardiothoracic surgery there (during which the valve in question was inserted) and had imaging performed there. Indeed, this information was easily located upon perusal of the medical record for this investigation.

The reviewing doctor observed that Mr C and his family were presented with information from the treating team on 9 January 2015 that he had an incurable disease. In the absence of a tissue diagnosis at this time, this remained an assumption only and there is no indication in the medical record that this was made clear to Mr C in discussions about his clinical management options. That said I accept the reviewing doctor's advice that active and more aggressive treatment may not have altered the outcome for Mr C at this stage.

Anthracosilicosis

Exposure to airborne mineral dusts such as silica, coal and asbestos may result in a lung disease known as Pneumoconiosis. Pneumoconiosis due to the inhalation of coal dust is known as anthracosis; of silica, silicosis and of asbestos, asbestosis. Depending on the exposure, a combination of pneumoconises may result from occupational exposure to these dusts or from exposure to significant air pollution.

Exposure to these mineral dusts results in irreversible deposition of fibrous tissue within the lungs. It may progress, usually over years, to progressive massive fibrosis.

After dust is inhaled, the smaller particles reach the terminal small airways and air sacs. The particles are engulfed by inflammatory cells. These inflammatory cells may die, resulting in the release of substances from the dead cells which incite inflammation and fibrosis; or the inflammatory cells may migrate through the lymphatic systems and are eventually eliminated.

In silicosis, the initial lesions are often located in the upper zones of the lungs. It starts as very small nodules of fibrous tissue and as the disease progresses, so too does the fibrous tissue in the lungs. Often this results in large coalesced nodules that have the appearance of hard collagenous scars. These fibrotic lesions also appear in the lymph nodes at the hilum of the lungs and the pleural surface of the lungs. This disease usually starts in the upper posterior lungs (especially the right lung) because the lymphatic drainage is slowest in this area.

Coal dust often contains silica as a contaminant so there is often evidence of fibrosis associated with both carbon and silica. Inhaled carbon pigment appears black on inspection of affected lung tissue – the term 'anthracosis' describes these darkly pigmented carbon deposits within lung macrophages, connective tissue, lymphatics and lymph nodes.

Silicosis presents with cough, shortness of breath and decreased exercise tolerance.

I am advised that bronchoscopy is not usually helpful in the diagnosis. This is because the disease starts in the smaller peripheral airways and fibrotic narrowing of large bronchi may not develop until there is advanced disease. For similar reasons, biopsies taken at bronchoscopy rarely yield sufficient tissue to make a diagnosis.

The reviewing doctor suggested that the autopsy diagnosis of pneumoconiosis was a timely reminder to clinicians to consider this disease in the differential diagnosis of thoracic lymphadenopathy.

Further clinical review undertaken by the tertiary hospital

Noting that there are some distinctive features of silicosis which may be seen on chest imaging, I asked Dr H to review the imaging performed on Mr C to see whether any of these features were identifiable.

Mr C's clinical history and radiology (specifically the CT chest scan from December 2014 and the PET/CT scan from January 2015) were reviewed at the Respiratory Radiology Meeting on 9 May 2017 with input from a pulmonary radiologist and a number of respiratory physicians. The group agreed that the radiological features were still most consistent with pulmonary malignancy and there were no particular features suggestive of anthracosilicosis. In particular, the PET FVD avid left lower lobe collapse and the mediastinal lymph nodes were not in keeping with this diagnosis. There were a number of lymph nodes demonstrating calcification which although can be a feature of occupational lung disease, in this context were considered far more likely to represent previous infection with tuberculosis. Even with the benefit of the autopsy findings, the interpretation of the radiology was still strongly favouring pulmonary malignancy. I accept the specialist clinical consensus in this regard.

Dr CH clarified the impression given by the medical records about the focus of Mr C's clinical management after the bronchoscopy. He clarified that Dr G discussed the bronchoscopy results with Mr C with the assistance of an interpreter on 9 January 2015. He indicated at the time that he felt the diagnosis still represented underlying metastatic lung cancer, particularly based on the radiological appearances. Notwithstanding that advice, Mr C remained in the Respiratory High Dependency Unit for a further 36 hours where he continued to be reviewed by medical staff and continued to receive intravenous antibiotics, high flow oxygen therapy and diuretic therapy. The Acute Resuscitation Plan was completed after Mr C's acute deterioration late that afternoon indicating it was clinically appropriate to provide antibiotics, monitoring, intravenous fluids and supportive therapy. As such Mr C continued to receive active treatment – the only aggressive treatment that was withheld was mechanical ventilation and/or CPR.

Dr CH outlined two systemic changes implemented at the tertiary hospital since Mr C's death which he considered would assist in reducing the risk of the error which occurred in relation to the management of Mr C's anticoagulation during the January 2015 admission:

- the tertiary hospital now uses integrated Electronic Medical Records (iEMR), a key function of which is a common "Problem List" which is shared by all clinicians – the documentation of a patient's medical problems is specific (that is, it must be selected from a restricted list rather than free text) and is used by different treating teams to summarise the patient's ongoing active issues. This function would identify the type of cardiac valve replacement and as such could have ensured continuity of understanding by the admitting respiratory team in January 2015 of the type of Mr C's heart valve and the correct reason for his Warfarin; and
- a different Respiratory Procedure Booking Form is now in use – this form includes a specific reference to anticoagulation and includes the need to document a plan for anticoagulation before the respiratory procedure. Dr H considered that although this would not directly prevent the misinformation about the type of heart valve that occurred in relation to Mr C, it does guide the ordering clinician to give specific thought to the patient's anticoagulation requirements.

Findings required by s.45 of the *Coroners Act 2003*

Identity of the deceased:	YC
How he died:	Mr C died from endobronchial and pulmonary haemorrhage secondary to pneumonia complicating chronic anthraco-silicotic lung disease. The admitting respiratory team failed to identify the correct type of Mr C's cardiac valve replacement and incorrectly assumed he was on Warfarin for a mechanical heart valve; he was in fact on Warfarin for his atrial fibrillation. This error occurred despite Mr C's medical records containing information about his previous valve replacement surgery performed at the same hospital and his ongoing cardiac issues. This incorrect assumption led to Mr C being inappropriately placed on bridging heparin for the bronchoscopy, which therapy likely played a role in causing the haemorrhage. I am reassured that the functions of the integrated Electronic Medical Record implemented at the tertiary hospital since Mr C's death will improve the accuracy of clinical communication between different treating teams about a patient's previous surgeries and active issues, and help reduce the risk of the transcription error that occurred in relation to an important aspect of Mr C's medical history occurring in future.
Place of death:	A tertiary hospital
Date of death:	10 January 2015
Cause of death:	1(a) Endobronchial and pulmonary haemorrhage 1(b) Pneumonia complicating anthraco-silicotic lung disease 2 Coronary atherosclerosis, atrial fibrillation, bronchoscopy, anticoagulant therapy

I close the investigation.

Ainslie Kirkegaard
Coronial Registrar
Coroners Court of Queensland

29 August 2017