QUEENSLAND COURTS

CORONERS COURT OF QUEENSLAND

FINDINGS OF INVESTIGATION

CITATION:	**Non-inquest findings into the death of Scott Anthony Bradshaw**
TITLE OF COURT:	Coroners Court
JURISDICTION:	Brisbane
DATE:	20 June 2016
FILE NO(s):	2013/1504
FINDINGS OF:	Christine Clements, Brisbane Coroner
CATCHWORDS:	CORONERS: Urgent general practitioner referral to hospital emergency department. Thirty eight year old man with presumed pneumonia. Assessed and differential diagnosis did not include pulmonary thromboembolism. Cause of death pulmonary thromboembolism due to deep vein thrombosis. Contributing factor dilated cardiomyopathy. Background antipsychotic medication.

Introduction

Scott Anthony Bradshaw lived at Logan Reserve Road, Logan Reserve in Queensland. He died on the 29th of April 2013 in the Queen Elizabeth II Jubilee Hospital at Coopers Plains in Queensland. Mr Bradshaw was 38 years of age at the time of his death. There were some questions regarding the cause of his death and there was concern expressed by family members with respect to health care. Therefore Mr Bradshaw's death was reported to the Coroner.

Presentation to general practitioner, Dr Cafferky

On Monday 29 April 2013 at approximately 14:15 hours, Mr Bradshaw consulted his regular General Practitioner, Dr Jonathan Cafferky at the Calamvale Medical Centre. He was in company with his fiancée, Lisa. Dr Cafferky recorded Mr Bradshaw was a vague historian. It was documented that Mr Bradshaw was already scheduled to see Dr Pertnikovs, a respiratory physician on the following day. He needed a referral letter.

Mr Bradshaw reported a six month history of shortness of breath but with no wheeze. His condition had worsened in the past week, with fever and sweating on the Saturday. No coughing was noted nor was there any runny nose or sore throat. He had pleuritic chest pain mostly on the left hand side. Dr Cafferky documented that Mr Bradshaw wanted a chest x-ray and had stopped smoking three days earlier on Friday. Previously he had smoked 10 cigarettes per day.

On examination Dr Cafferky recorded the following physiological observations:

- Blood pressure (sitting) 110/70;
- Pulse 130 beats per minute;
- Chest – reduced air entry with crepitation (crackling/rattling sounds) at the bases of his left lung;
- Oxygen saturation 96%;
- Temperature 39.2 (elevated); and
- Tachypnoeic (abnormally rapid breathing) at rest.

Pneumonia was considered by Dr Cafferky to be the most likely cause of his symptoms. He advised Mr Bradshaw that he needed to *immediately* attend the Emergency Department for a chest x-ray and intravenous antibiotics. He told Mr Bradshaw there was a risk to his life if he did not attend and that it could not wait until he saw Dr Pertnikovs the following day. Dr Cafferky recorded he advised Mr Bradshaw of the risk to his life if he leaves it. He also noted Mr Bradshaw's mother phoned him during the consultation and she too was aware of the doctor's advice.

Dr Cafferky also documented an alternative, but not preferred option, was to have the chest x-ray done and return straight back to Dr Cafferky. He provided a request form for the chest x-ray and a referral letter addressed to the Emergency Department doctor.

The referral note stated Mr Bradshaw was presumably suffering from pneumonia and had symptoms of fever, pleuritic chest pain and laboured and difficult breathing at rest (dyspnoea). It was also noted that there was reduced air entry.

Mr Bradshaw's past active medical history was documented by Dr Cafferky as follows:

- (2000) schizo-affective disorder;
- (2001) bipolar affective disorder;
- (2002) asthma; and

- (2005) neutrophilia.

Dr Cafferky noted the inactive condition of ITP (idiopathic thrombocytopenic purpura).

Dr Cafferky noted Mr Bradshaw's currently prescribed medications were:

- Hypnodorm 1mg two tablets to be taken in the evening;
- Lithium 450mg tablet modified release, two to be taken before bed;
- Rivotril 2mg tablet, four to be taken in the evening;
- Seretide Inhaler, two puffs twice a day;
- Seroquel 300mg tablet, five to be taken in the evening;
- Spiriva 18mcg to be taken one capsule daily;
- Ventolin inhaler to be taken as required; and
- Ventolin nebules 5mg, one to be taken every four hours as required.

Events at the emergency department of Queen Elizabeth II hospital

At 16:30 on 29 April 2013 (approximately two hours following his consultation with Dr Cafferky) Mr Bradshaw arrived at the QEII Emergency Department in company with his fiancée, Lisa. Other family members also attended.

Mr Bradshaw was triaged as a Category 3 patient in accordance with the Australasian Triage Scale. This meant he was to be seen by a doctor within 30 minutes.

At 16:40 the triage nurse recorded the following physiological observations:

- blood pressure was 111/84;
- temperature was 37.9 degrees (elevated);
- pulse was 132;
- respiration was 28, (elevated); and
- oxygen saturation was 96

The nurse who assessed Mr Bradshaw recorded that he was neurologically alert and had a pain scale reading of zero out of ten, meaning no pain.

The presenting history from Dr Cafferky was repeated. In addition, the triage nurse recorded:

> 'Left sided worsening chest pain stated by patient. Unwell approx last month.'

A student enrolled nurse also added the following:

> 'Family state patient generally unwell over last three months, increasing over one month, worse over one week. States anorexia (asthma?), increasing shortness of breath, pleuritic chest pain. No bowel or urine complaints.
> '

An ECG was ordered and at 17:00, the ECG showed sinus tachycardia and left access deviation.

Very shortly after Mr Bradshaw was triaged, Mr Bradshaw was seen by medical resident Dr Muhammad Shah. This was between 16:34 and 16:40. Dr Shah immediately notified a more senior clinician Dr Terry Nash, who was the Senior Medical Officer in Emergency Medicine. Dr Nash attended upon Mr Bradshaw at 16:50.

Dr Nash was concerned with Mr Bradshaw's abnormal vital signs, and ordered that he be moved to the resuscitation unit. This occurred at 16:55.

Dr Nash ordered oxygen (via a Hudson mask), intravenous fluids, antibiotics and continuous monitoring. Dr Nash also reviewed the chest x-ray which suggested consolidation as well as pulmonary congestion. According to Dr Nash, the provision of saline and better posture, improved Mr Bradshaw's condition and he became more settled.

At approximately 17:30, Mr Bradshaw's condition deteriorated and he became distressed, confused and was sweating profusely. Dr Addae-Bosomprah, staff specialist in emergency medicine was called to review Mr Bradshaw. Mr Bradshaw was observed to be agitated, with increased work in breathing. His respiration rate was 30, oxygen saturation was down to 88 %, his temperature was 38, and heart rate 150 with sinus tachycardia and cold mottled skin. Swelling was noted in the extremities. Bibasal 'crackles' were heard in the lungs. Mr Bradshaw had removed the Hudson mask which had been provided to assist his respiration with positive pressure oxygen delivered via a mask over the mouth and nose.

Mr Bradshaw's condition rapidly declined with his heart rate increasing to 200 together with a weak pulse. He appeared pale, cyanosed and was sweating profusely.

The intensive care team was called to alert them of the likely need for an intensive care bed. The Intensive Care Specialist Dr Wright was informed of the general condition of the patient at about 1830 hours and the assembled team of doctors attending upon him. He did not have anything to add given that information and the seniority of the medical staff. Dr Wright checked in on progress on the Emergency department at about 1930. Resuscitation was continuing with an expert team assembled. He returned to ICU to ensure readiness for the patient upon transfer. When next he returned to the emergency department it was evidence that Mr Bradshaw was deceased.

Mr Bradshaw was initially placed on a non-invasive mechanical pressure support ventilation device (BiPAP) while set up for intubation occurred. Mr Bradshaw did not tolerate the BiPAP machine however.

Mr Bradshaw was then pre-oxygenated and given medication for rapid sequence induction (ketamine, fentanyl and suxamethonium).

Due to the presence of secretions and fluid in Mr Bradshaw's airway, intubation was difficult and two unsuccessful attempts were made by Dr Addae-Bosomprah. Suctioning was required twice during attempts to intubate Mr Bradshaw. His breathing was supported with bag and mask in the interim period.

The third attempt was successful and the correct placement was confirmed on x-ray.

Mr Bradshaw then became bradycardic and went into cardiac arrest with pulseless electrical activity at 18.34hours. Five cycles of full cardiopulmonary resuscitation) were performed including administration of adrenalin infusion, and metaraminol. Mr Bradshaw was also connected to a defibrillator.

It was at about this time that the intensive care specialist Dr Wright was present and confirmed a bed would be available upon transfer to the ICU. Mr Bradshaw's heart rate had dropped to 70bpm however so he was then palpated for carotid pulse. None was found. CPR was therefore continued for approximately 70 minutes to attempt to get a resumption of a perfusing rhythm. Multiple doses of Adrenaline were provided and ICU Registrar Claire Dunsdon inserted a right femoral venous line with 3 way access.

A bedside echo cardiograph was performed by cardiologist, Dr Franjic, which showed the heart had no contractility.

Dr Nash confirmed there was discussion between Doctors Wright, Addae-Bosomprah and Dr Nash what other interventions might assist, and what other possible diagnoses could be applicable. There was consideration of toxins, drugs, atypical infections, including myocarditis and pre-existing cardiomyopathy.

By 19.55 hours there was agreement from all of the attending doctors that further resuscitation was futile and Mr Bradshaw was declared deceased.

Family members were understandably shocked at his sudden and unexpected death. They raised many concerns with respect to his medical care. Their concerns were conveyed to the Coroner and to the pathologist. To the extent these matters were within the coronial jurisdiction, these matters were reviewed and considered.

Establishing the cause of death

Autopsy examination was conducted by the forensic pathologist, Dr Olumbe, on the 29th of May 2013.

The pathologist noted Scott's height and weight which correlated to a body mass index of 30.8.

The significant anatomical findings documented in the autopsy report were:

1. Pulmonary thromboemboli and lung infarcts (left lower lobe and possibly right lower lobe);
2. Bilateral calf muscle deep venous thrombi;
3. Dilated cardiomyopathy;
4. Mild obesity;
5. Pulmonary oedema.

CT imaging confirmed there were no findings of concern in the head.

There was haemorrhage within the left neck, presumably reflecting an attempted line placement. No pneumothorax was evident.

There were large bilateral pleural effusions. The pathologist considered there was a component of haemorrhage. Calcified granuloma was evident at the base of right lung. There was an acute transverse fracture through the sternum and fractures of the left 3rd to 6th ribs and anteriorly which was consistent with resuscitation. The consultant radiologist who reviewed the CT imaging confirmed the opinion regarding the explanation for the fractured ribs.

Tissue examination under microscopic magnification showed thrombi (clots) of several days to several weeks duration.

The lungs showed moderate congestion and patchy oedema and extensive pulmonary infarcts associated with multiple thromboemboli of several days duration.

The heart was noted to be markedly dilated and showing evidence of dilated cardiomyopathy.

There was no gross evidence of any infective process. Samples of femoral blood, aortic blood, urine and vitreous humour were tested. These showed the presence of multiple drugs of prescription including therapeutic levels of clonazepam and flunitarazepam, lithium, and

quetiapine. There were also low concentrations of ketamine, which is an anaesthetic agent used by medical personnel.

Methanol was detected in the blood, urine and vitreous humour. Dr Olumbe discounted the presence of methanol as it was known that Mr Bradshaw's body had been embalmed prior to re-presentation to the Government mortuary for autopsy examination.

The pathologist concluded Scott Bradshaw died due to pulmonary thromboembolism. This is the formation of blood clots/thrombus that break loose and are carried by the blood stream to plug another vessel. This was complicated with lung infarcts (area of tissue death due to the obstruction of the blood supply, typically by a thrombus/blood clot).

The presence of pulmonary thromboemboli prevented blood flowing to the lungs where it becomes oxygenated prior to flowing back to the heart and on to the rest of the body. This accounted for Mr Bradshaw's sudden unexpected death. It also explained the symptoms of respiratory distress, shortness of breath and chest pain a few days prior to his death.

The pathologist noted the most common source of pulmonary thromboemboli is deep venous thrombi, usually in the calf muscles which was confirmed in Mr Bradshaw's case.

Pulmonary embolus can be associated with certain risk factors including- obesity, immobility, trauma, recent surgery, underlying clotting abnormality and other factors. The pathologist noted a possible factor of obesity in this case.

Dr Olumbe commented that dilated cardiomyopathy would have contributed to the death by potentially causing dysfunction of the heart rhythm. This can occur even in the absence of coronary artery disease or valvular heart disease. The cause of this cardiomyopathy could be genetic, non-genetic or acquired and predominantly confirmed to the heart muscle. Alternatively, secondary cardiomyopathy can result from systemic or multiple organ disease. The pathologist considered it wise for immediate family members to seek medical advice from their general practitioner with respect to their susceptibility to this condition.

It was concluded by the pathologist, and accepted by the Coroner that Scott Anthony Bradshaw died due to pulmonary thromboembolism caused by bilateral deep calf vein thrombi.

Other contributory factors in his death were noted to be dilated cardiomyopathy.

Discussion of autopsy findings

The autopsy conclusively established what had caused Mr Bradshaw's sudden deterioration and death.

The conclusion of the pathologist in reaching a clear cause of death has;

1. Helped to narrow the areas of concern initially expressed by the family concerning Mr Bradshaw's medical care;
2. Informed the treating team in retrospect of the nature of Mr Bradshaw's undiagnosed condition at the time of his presentation at the emergency department;
3. Focused Coronial review on the issues of:
    a. Whether the health care provided (or not provided) was likely to have caused or contributed to Mr Bradshaw's death; and
    b. If it did, then, immediately before receiving the health care would an independent person (appropriately qualified in the relevant areas of health)

not have reasonably expected that the health care (or failure to provide health care) would have caused or contributed to his death.

Mr Bradshaw died due to pulmonary thromboemboli and lung infarcts caused by deep venous thrombi in both calf muscles.

This condition was not diagnosed between 16.34 (when assessment by a doctor commenced in the emergency department) and 18.30 (when sedation was administered to enable intubation after Mr Bradshaw's rapid decline).

Cardiac arrest was called at 18.34 and all efforts from that time were concentrated on resuscitation until it was considered futile at 19.55 and Mr Bradshaw was declared deceased.

Consideration of medical treatment

Mr Bradshaw's family were concerned that his death might have been avoided.

Statements were also obtained from a number of the medical and nursing staff at the QEII that were involved with Mr Bradshaw's care including:

- Dr Terry Nash, Senior Medical Officer in Emergency Medicine who carried out initial assessments of Mr Bradshaw in the presence of Dr Shah and arranged for him to be moved to the resuscitation area. Dr Nash was also the Team Leader for the resuscitation;
- Dr Hansel Addae-Bosomprah, Staff Specialist in Emergency Medicine who assessed Mr Bradshaw while he was in the resuscitation area and was appointed as the airway doctor for the resuscitation;
- Dr Claire Dunsdon, Intensive Care Principal House Officer who also assessed Mr Bradshaw while he was in the resuscitation area and assisted Dr Addae-Bosomprah in the management of Mr Bradshaw's airway during the resuscitation;
- Dr David Wright, Senior Staff Specialist in General Medicine and Intensive Care who was consulted in respect of the availability of beds within the Intensive Care Unit when Mr Bradshaw started to deteriorate;
- Registered Nurse Vivien Triz who was rostered to work in the acute area of the Emergency Department and was appointed as the circulation nurse for the resuscitation;
- Registered Nurse Gracielle Torres who was rostered to work in the resuscitation area on the day and was appointed as the airway nurse for the resuscitation;
- Registered Nurse Sophie Sebilo was rostered to work in the acute care of the Emergency Department on the day and who was appointed as one of the drug nurses for the resuscitation.

Review of the autopsy findings and the medical records was arranged. There were reports provided by:

- Dr Adam Griffin, Director at the independent Clinical Forensic Medicine Unit;
- Associate Professor John Raftos, emergency medicine specialist, and an independent expert instructed by Mr Bradshaw's family lawyers;
- Dr Peter Tomlinson, consultant general and vascular surgeon, and an independent expert instructed by Mr Bradshaw's family lawyers;
- Dr Nai An Lai, Director of the Intensive Care Unit at the QEII Hospital; and
- Professor Anne-Maree Kelly, Professor and Academic Head of Emergency Medicine at Western Health in New South Wales.

An overview of these reports is provided below.

Report from Associate Professor John Raftos

An expert report from Associate Professor John Raftos was provided to the Coroner. Dr Raftos is an experienced and expert emergency medicine specialist practising in that specialty since 1983.

After review of the medical record, Dr Raftos formed the view that pulmonary embolism should have been among the differential diagnoses considered and that urgent echocardiography was indicated and was available within a short timeframe (15-60 minutes). He also stated that the ECG gave strong clues towards pulmonary embolism and that Mr Bradshaw was strong enough to undergo CT angiography until late in his clinical course.

Dr Raftos opined that anticoagulation and thrombolysis (clot dissolving treatment) should have been administered at an earlier time – but after confirmation of massive pulmonary embolism by CT angiography.

Dr Raftos also opined that it should have been possible to diagnose massive pulmonary embolism by 18:00 and that prompt treatment thereafter would, on balance of probability, have prevented Mr Bradshaw's death.

Review from Clinical Forensic Medicine Unit, Dr Adam Griffin

Dr Adam Griffin, the director of the independent Clinical Forensic Medicine Unit, also reviewed the medical records and provided an initial and subsequent report. He also had the opportunity to refer to Dr Raftos' report.

Dr Griffin noted the initial cause of death documented in the hospital record had concluded Mr Bradshaw died due to:

- pulseless electrical activity arrest, due to
- sepsis, due to
- left lower lobe pneumonia, due to
- chronic lung disease

Dr Griffin noted the subsequent autopsy established Mr Bradshaw had died due to pulmonary thromboembolism as a result of bilateral deep calf vein thrombi. Dilated cardiomyopathy also contributed to the death.

Dr Griffin had the advantage of being able to compare an earlier ECG of Mr Bradshaw, completed in 2007. This was accessed via general practitioner records. Bearing this in mind, Dr Griffin's opinion was that the ECG changes were non-specific and do not assist in favouring one diagnosis over another, as distinct from the opinion expressed by Dr Raftos.

Had the treating team considered and reached the diagnosis of pulmonary embolus, Dr Griffin was not as convinced as Dr Raftos that the provision of thrombolysis would have saved Mr Bradshaw had it occurred prior to cardiac arrest.

Dr Griffin referred to 19 studies which showed no improvement in end-point when thrombolysis was used and no improvement in survival. The studies did show the improvement in circulation but the risk of bleeding was shown to be higher.

Therefore, Dr Griffin considered there was inadequate evidence to support the use of thrombolytic therapy in adult patients with pulmonary embolus following a return of spontaneous circulation after cardiac arrest, which was the situation in Mr Bradshaw's case.

The other issue raised by Dr Griffin was the possible timeframes in which there was theoretically a window of opportunity to consider and reach a particular diagnosis and administer appropriate treatment.

In Dr Griffin's opinion, he noted there were three emergency doctors and two intensive care doctors and supporting cardiology expertise to assist in an investigation. The group of clinicians did not diagnose the presence of pulmonary embolus on this occasion. He thought this was understandable given the clinical picture and available investigations which supported the diagnosis of pneumonia as the cause of Mr Bradshaw's presentation.

Dr Griffin disagreed with ECG being determinative in assisting the diagnosis of one condition over another.

Dr Griffin did, however, consider that pulseless electrical activity cardiac arrest can be caused by pulmonary embolus. When this occurred, pulmonary embolus should have been considered as a possible cause for the arrest by the treating team. Dr Griffin considered that even had that diagnosis been made it would not have affected the outcome. The recommended approach in such a condition would be to follow the resuscitation flowchart. This is the treatment which Mr Bradshaw received. Dr Griffin concluded the use of thrombolytic therapy in such cases has not been demonstrated to increase survival rates beyond normal resuscitative measures.

Report from Dr Peter Tomlinson

Dr Tomlinson is a consultant general and vascular surgeon in private practice in New South Wales.

Dr Tomlinson's review of the information provided to him led to his conclusion Mr Bradshaw was treated as if he had a diagnosis of pneumonia, and no other consideration was given to exclude other causes for his clinical state. He postulated possibilities of cardiac failure and pulmonary embolism with pneumothorax. He considered it a failure that the treating team did not consider the possibility of pulmonary embolism as a cause of the increasing decline in the patient's condition.

In particular, Dr Tomlinson noted ECG changes of pulmonary hypertension and right atrial dilatation were classical signs associated with pulmonary embolism.

Dr Tomlinson also raised Mr Bradshaw's known history of idiopathic thrombocytopenia purpura, which he described as a bleeding condition with low platelet disorder. He observed, 'current literature over ten years has revealed an increased episode of thromboembolic disease in this patient group.'

(It is noted the record appears to state Mr Bradshaw went into cardiac arrest approximately two hours after arrival at the emergency department, not two and a-half hours referred to by Dr Tomlinson.)

Dr Tomlinson considered the treating doctors should have considered thrombo pulmonary embolism, given his idiopathic thrombocytopenia purpura, symptoms on presentation, and recent history of reported symptoms.

(It is noted that there were some equivocal matters, for example, the initial recording by the triage nurse that Mr Bradshaw had no pain when first seen).

As well, Dr Tomlinson stated differential diagnoses of myocardial infarction, pneumonia, cardiac tamponade, cardiac failure and pneumothorax should have been considered. Analysis of tests performed would have excluded these.

In Dr Tomlinson's view, pulmonary embolism should have immediately been one of the possible diagnoses upon Mr Bradshaw's presentation to the emergency department. His view was this condition should have been diagnosed within 30 minutes of presentation.

With the suspicion of thromboembolic disease, Dr Tomlinson stated heparin should have been administered prior to confirmation of the diagnosis.

Confirmation would have required nuclear ventilation perfusion scanning or pulmonary angiography.

Dr Tomlinson considered there was sufficient information to justify the administration of 5000u heparin intravenously as a bolus dose and a heparin infusion should have been given at the time he deteriorated and was taken to resuscitation unit.

Had this occurred, in Dr Tomlinson's opinion Mr Bradshaw would have survived on the balance of probabilities. This opinion was based on the assumption the pulmonary embolism which caused his demise most likely came just prior to his cardiac arrest.

Dr Lai

Dr Nai An Lai is the Director of the Intensive Care Unit at the QEII Hospital was invited to respond to a number of issues and clinical points raised by Dr Raftos.

Dr Lai indicated that while portable echocardiography can be a useful test for pulmonary embolism, the test cannot definitively identify pulmonary embolism as it lacks both sensitivity (accuracy identifying the condition) and specificity. Dr Lai also pointed out that this was particularly the case given Mr Bradshaw's underlying heart condition. Dr Lai opined that pulmonary embolism has a range of severities and prognoses dependant in part on the co-morbidities and that accurate estimates for individual patients can be difficult. It was also identified by Dr Lai that persistent hypotension is the only widely accepted indication for thrombolysis.

Dr Lai considered that the differential diagnoses for Mr Bradshaw were broad and that the clinical probability of pulmonary embolism based on information available upon his arrival at the ED (calculated using a validated clinical prediction rule) was not high.

Finally, Dr Lai considered that the ECG features were not entirely typical of pulmonary embolism.

Report from Professor Anne-Maree Kelly

Professor Kelly was engaged by the Office of the Stater Coroner to independently review the material and provide a report. Professor Kelly is the Professor and Academic Head of Emergency Medicine at Western Health and has over 25 years of experience in emergency departments.

Cause of death

Professor Kelly was of the opinion that pulmonary emboli was a major factor in Mr Bradshaw's death. However in her opinion cardiac failure related to the previously undiagnosed dilated cardiomyopathy found at autopsy and this condition also played a significant role.

Professor Kelly indicated that this would also fit with the progressive shortness of breath that had been developing over a period of months as the heart started to fail. The pulmonary emboli and consequent hypoxia (low blood oxygen levels) and pulmonary hypotension would have added to a 'weak' heart causing it to progressively fail. Professor Kelly indicated that it was more likely than not that the failing heart was the final pathway leading to Mr. Bradshaw's death which would fit with Dr Olumbe's findings that the pulmonary emboli were of several days' duration rather than fresh.

She stated that unfortunately the clinical staff's attempts to support what they thought was septic shock with IV fluids (the correct guideline-based treatment for this condition) may have inadvertently added pressure to a failing heart by increasing the amount of fluid in the vascular system.

Differential diagnosis of chest pain, fever and shortness of breath

Professor Kelly was of the opinion that while pulmonary embolism was a potential differential diagnosis, in light of the physical findings, the high white cell count and chest x-ray, pneumonia was "*an appropriate working diagnosis*". Other differential diagnosis that should have been considered were pneumothorax, heart disease, conditions in the upper abdomen causing inflammation of the diaphragm and viral pleurisy (inflammation of the lining around the lungs).

Investigation for pulmonary embolism

Professor Kelly noted that in Australia the recommended process for identifying pulmonary embolism is initial risk stratification using a validated clinical risk score such as the Wells' pulmonary embolism score. This identifies a low risk population and a non-low risk population. The non-low risk population is referred for pulmonary imaging. The low risk group is recommended to have a d-dimer test. If this is negative, pulmonary embolism is ruled out. If it is positive, pulmonary imaging is undertaken.

Professor Kelly says that Mr Bradshaw's calculated risk using this score was 1.3% which was low risk. Professor Kelly noted that a d-dimer test was not undertaken, however an average time to undertake this test is about one hour and therefore the results would only have been available when Mr Bradshaw was "deteriorating rapidly and unsuitable for transfer to the imaging department".

ECG and pulmonary embolism

In Professor Kelly' opinion, ECG changes cannot confirm or exclude pulmonary embolism.

Treatment of pulmonary embolism

Professor Kelly noted that respected guidelines detailed the treatment for a pulmonary embolism is therapeutic anticoagulation with low molecular weight heparin or similar in proven pulmonary embolism and in non-low risk patients during investigation for suspected pulmonary embolism. Mr Bradshaw did not have proven pulmonary embolism and according to the Wells score would have been classified as low risk if PE had been considered.

Professor Kelly reported that the guidelines were clear that thrombolysis is not recommended in undifferentiated cardiac arrest. She also noted that one of the guidelines indicated that thrombolysis is 'reasonable' for patients with massive acute pulmonary embolism in whom the risk of bleeding is acceptable and that it may be considered in submissive pulmonary embolism in patients with new haemodynamic instability (low blood pressure or shock), worsening respiratory insufficiency, severe right ventricular dysfunction or major myocardial damage.

Professor Kelly was of the opinion that at no time before the sudden deterioration during endotracheal intubation did Mr. Bradshaw demonstrate hypotension or shock, the only widely accepted indications for thrombolysis in (identified) PE.

Idiopathic thrombocytopenic purpura (ITP) and risk of pulmonary embolism
Professor Kelly noted that Mr Bradshaw's diagnosis of ITP was more than 20 years previously and this was not an active clinical issue for at least the 10 years before these events. ITP was unlikely to be a risk factor in this case.

Time and availability of tests in emergency departments

Professor Kelly reported that sometimes clinicians who do not work in emergency departments underestimate the time it takes to undertake tests and the availability of tests especially after hours.

Professor Kelly noted that initial assessment, performance of an ECG and blood tests will take of the order of 15-30 minutes, with blood test results often available 45-60 minutes after this. To get a chest x-ray performed and processed will take a minimum of 10-15 minutes for a critically ill patient – often more like 30-60 minutes in a patient who initially appears stable. Echocardiography is not available in most hospitals after hours – remembering that when Mr. Bradshaw deteriorated it was about 1730 hours. Similarly access to other imaging such as CT scans can take a variable period but usually more than 30 minutes. It is important to remember that pathology and imaging services are not just there for one patient so their ability to respond depends on what else is 'in the queue.

Professor Kelly was of the opinion that when Mr Bradshaw deteriorated, efforts were rightly focused on ensuring adequate ventilation (breathing and oxygenation) which is life-saving rather than diagnostic processes. While parallel processes for resuscitation and diagnosis are preferable, at times resuscitation needs to take priority. In Dr Kelly's opinion, this was the case here.

Conclusions/opinions provided to the specific questions raised

Professor Kelly was of the opinion that the assessment of Mr Bradshaw was appropriate however pulmonary embolism was a potential differential diagnosis that should have been considered. Professor Kelly indicated that ideally, while steps to rule out pulmonary embolism (using a d-dimer test) and other diagnoses such as coronary heart disease might have been initiated, the results are unlikely to have been available in a time-frame that would have changed events.

Professor Kelly was of the opinion that it was very unlikely that pulmonary embolism could have been diagnosed safely before Mr Bradshaw's deterioration and need for resuscitation. If Mr. Bradshaw's pulmonary embolism had been diagnosed, the appropriate treatment was anticoagulation, risk stratification and monitoring. In the absence of shock or hypotension, thrombolysis was not recommended evidence-based treatment according to accepted guidelines. Anticoagulation would not have altered the outcome as it would not have reduced the clot burden in a relevant time frame.

Professor Kelly commented that given the clinical and radiological information available it was reasonable to have made a provisional diagnosis of pneumonia and begun treatment accordingly.

Supplementary advice sought from Dr Olumbe

Following receipt of Professor Kelly's report, the pathologist Dr Olumbe was given the opportunity to review his conclusion of the cause of death, particularly the potential relevance of cardiomyopathy in Mr Bradshaw's death.

Dr Olumbe confirmed his opinion that the cause of death was due to pulmonary thromboembolism due to bilateral deep calf vein thrombi. He considered dilated cardiomyopathy was a contributory factor in Mr Bradshaw's death.

Clinical review carried out by QEII Hospital

In response to Mr Bradshaw's death, the QEII carried out a clinical review which made a number of recommendations including:

1. Education and awareness campaign be established to assist clinicians in broadening their differential diagnoses and to raise clinicians' suspicion of pulmonary embolism in patients presenting with respiratory distress;
2. Implement a diagnostic algorithm in the Emergency Department for stable and unstable patients suspected of having pulmonary embolism;
3. Increased access to bedside ECG;
4. Strengthen the resuscitation team's ability and equipment to deal with a difficult airway; and
5. Consideration of having a CT scanner in the Emergency Department with appropriate resuscitation equipment to improve access and mitigate risk of transferring potentially unstable patients to the radiology department for an urgent CT.

On 4 March 2016, correspondence was received from Dr Dale Seirup, Acting Director of Medical Services at the QEII Hospital.

Dr Seirup noted that in April 2013, Mr Bradshaw was treated in the former Emergency Department and that since that time, a number of significant changes had been made to the Emergency Department that will ensure they can better meet the health demands of their community.

He also indicated that there have been no clinical incidents relating to pulmonary embolism in the last 12 months.

Dr Seirup highlighted that in late 2013, a new Emergency Department was built including increased staffing and general resources including:

- An increase in senior clinical staff including medical officers and clinical nurse consultants;
- Allied health in the ED including a social work team; and
- A family room to ensure that family members are adequately supported.

Education and training

Dr Seirup advised that education sessions that specifically relate to pulmonary embolism have since been delivered as part of the ongoing educational requirements for junior medical staff. Attendance records were provided for two education sessions on pulmonary embolism that were held on:

- 2 September 2015 (by Clare Kao, Specialist in Emergency Medicine) and;
- 16 December 2015 (by Tim Haina, Specialist in Emergency Medicine).

These sessions were scenario based and focussed on ensuring that consideration be given to:

- Presenting symptoms;
- Whether those presenting symptoms indicate pulmonary embolism;
- Whether there is any other information that would assist in a diagnosis;
- Diagnosing pulmonary embolism using the Wells criteria for assessment of pre-test probability;

- What the treatment would be if the lung scan came back with a high probability for pulmonary embolism.

Dr Seirup also advised that a Simulation Registrar has been employed to coordinate ongoing education programs in the ED for both medical and nursing staff.

Implementation of a diagnostic algorithm

Dr Seirup advised that an algorithm for the diagnosis of pulmonary embolism for stable and unstable patients has now been developed and is now available to staff on the hospital intranet. It includes the validated pulmonary embolus rule criteria (PERC) and Wells pulmonary embolism scores. It also highlights the need to involve senior staff immediately in the case of vascular collapse with suspected pulmonary embolism.

Bedside echocardiography

Dr Seirup advised that a new bedside echocardiography (ECG) is available and provided by a private medical imaging provider during business hours between Monday to Friday. After hours, the Emergency Department's ultrasound is available to carry out ECG and whilst it was acknowledged that senior radiology or cardiology staff wold be considered more qualified to interpret an ECG, a number of the Emergency Department medical staff have been undertaking additional training to improve their ECG skills.

Education and Simulation Sessions in Airway Management

Dr Seirup advised that a number of education and simulation sessions regarding airway management have been established to strengthen the resuscitation team's ability and equipment to deal with a difficult airway.

He confirmed a new video laryngoscope has been purchased and training has been provided to staff in relation to its use.

New CT Scanner in the ED

In relation to the recommendation that consideration be given to the purchase of a CT scanner for the Emergency Department, Dr Seirup advised that the medical imaging department is now located adjacent to the new Emergency Department and that access to these facilities is available to patients. The QEII hospital management did therefore not consider that a further CT scanner was necessary.

Conclusion

The primary condition which caused Mr Bradshaw's death (pulmonary thromboembolism) was not diagnosed and therefore not specifically treated when Mr Bradshaw attended the Queen Elizabeth II Hospital on 29 of April 2013.

The possibility of this condition does not appear to have been considered in the differential diagnoses. Expert review obtained by Mr Bradshaw's' family considered the differential diagnosis of this condition should have been considered. Once considered, the treatment of anticoagulation and or thrombolysis should have been implemented pending confirmation of the diagnosis.

Further expert review agreed that pulmonary embolism was a potential differential diagnosis. Had the pre-test probability of pulmonary embolism been assessed by using the Well's score it would have given the likelihood as low. Ideally, a d-dimer test and other diagnostic investigation of possibility of coronary heart disease would have been pursued.

However, in the time frame available expert review considered this would not have changed the outcome.

The working diagnosis of pneumonia was reasonable in the circumstances, as was the care provided to Mr Bradshaw in the Emergency Department. Even if the pulmonary embolism had been diagnosed in the short time before Mr Bradshaw's sudden deterioration, there was differing expert opinion on whether or not Mr Bradshaw's death could have been prevented.

The hospital has thoroughly clinically investigated and reviewed the events that occurred from the time of Mr Bradshaw's presentation to the emergency department until the time he was declared deceased. The hospital has co-operated with the coronial investigation and responded to underlying issues.

The final expert report of Professor Kelly, which incorporated consideration of all of the material available to the coroner's investigation, provided further development of the contributory role of cardiomyopathy in Mr Bradshaw's death.

Professor Kelly summarised her report as follows-

> While Mr Bradshaw's death is tragic, I do not believe it was preventable at the time of his emergency department presentation. In my opinion, his death was due to the combination of pulmonary embolism and cardiomyopathy, potentially due to antipsychotic therapy, both of which were unsuspected. There may have been a prevention opportunity in primary care if his treating practitioners had known about his developing shortness of breath, investigated for a cardiac cause (identifying the cardiomyopathy) and changed his medications. From what I can see his GP was not aware of this. I did not have access to his treating psychiatrist's records.

In the circumstances working diagnosis of pneumonia was reasonable as was the care received in the emergency department. Even if the contributing pulmonary embolism had been diagnosed in the short time before Mr Bradshaw's sudden deterioration, in my opinion it is highly unlikely that treatment for this would have changed the outcome. "

The hospital and Mr Bradshaw's treating general practitioner have been provided with Professor Kelly's report.

The investigation and review process has been greatly assisted by the disclosure of expert opinion provided from a variety of sources. As is often the case, expert opinions can differ but all have contributed to a greater understanding of the background and the sequence of events prior to Mr Bradshaw's death.

These reports have;

- assisted in reaching the factual conclusions required to be determined by the coroner;
- provided guidance in clinical review to optimise appropriate diagnosis and treatment of pulmonary embolus and
- focused attention on the possibility of interplay of antipsychotic medication with the development of cardiomyopathy .

The focus of the Coroner's jurisdiction is to reach findings required in section 45 of the *Coroners Act 2003* if at all possible. There is sufficient information to do so and the findings are as follows:

a) The identity of the deceased is Scott Anthony Bradshaw;

b) He presented to the Queen Elizabeth II Jubilee Hospital emergency department at 1630 on 29 April 2013 following a consultation with his general practitioner at 14:30

> He told his general practitioner he had been short of breath over last 6 months, but worse over the last week. He was advised to go to the hospital immediately with a referral letter from his general practitioner stating, 'for your assessment and management of presumed pneumonia. He has dyspnoea at rest [shortness of breath], fever and pleuritic chest pain.'

He was seen promptly and triaged and medically assessed within the triage required time. It was immediately identified by the doctor that he was seriously ill and he was reviewed by more senior staff. An ECG was performed and reviewed and bloods were collected at 16.50. At 16.55 he was taken into a resuscitation unit due to worsening observations requiring intensive monitoring. A history of shortness of breath with pleuritic chest pain over a week was given. His family said he had been short of breath for a couple of weeks and reduced appetite in the last couple of weeks. Mr Bradshaw complained of pain and fatigue all over his body. Initial treatment was provided by ventilation support via a Hudson mask with oxygen under pressure and provision of intra venous fluids. He became distressed and a chest x ray and antibiotics were initiated as well as blood gases taken.

Additional expertise from ICU, and more senior emergency doctors including a cardiologist were involved as Mr Bradshaw's condition continued to decline.

At 1830 sedation was given to enable intubation. This was successful on the third attempt due to the need for suctioning. Within five minutes of intubation Mr Bradshaw went into cardiac arrest.

Full resuscitation efforts were immediately commenced from 18.34 and he was ventilated. He was connected to a defibrillator but no shockable rhythm was detected. Resuscitation efforts continued including medication and a bedside echocardiogram. At 19.55 it was determined by the treating team there was no sign of life and no return of spontaneous circulation. Mr Bradshaw was declared deceased.

c) Mr Bradshaw died at 19.55 on the 29th of April 2013;

d) He died in the emergency department of the Queen Elizabeth II Jubilee Hospital at Coopers Plains in Queensland;

e) He died due to pulmonary thromboembolism as a consequence of bilateral deep vein thrombi. Dilated cardiomyopathy was identified as a contributory factor in his death.

A copy of the findings is distributed to;

Mr Bradshaw's family,
the general practice Calamvale Medical Centre,
Mr Bradshaw's general practitioner Dr Cafferky,
the Queen Elizabeth II Hospital,
Patient Safety and
College of Emergency Medicine Physicians.

The findings are also published on the Queensland Coronial website. The dissemination of information in this way is the most appropriate and likely means to raise awareness of such an unexpected death and thus help to prevent a similar death occurring in the future.

Request for inquest

I do not consider the differences of opinion by reviewing medical specialists are matters which could lead to any useful recommendation or comment to prevent deaths occurring in similar circumstances.

The autopsy has clearly identified the cause of death and independent reviews of the treating team have identified the failure to include pulmonary embolus in the possible diagnoses causing Mr Bradshaw's symptoms upon presentation, rapid decline and death.

While there was no bronchopneumonia, there was moderate congestion and patchy oedema in the lungs leading the emergency doctors into overlooking other possibilities.

There are differences of expert opinion about whether or not the correct diagnosis should have been established. Expert opinion includes the view that the working diagnosis of pneumonia was reasonable in the circumstances as was the care Mr Bradshaw received in the Emergency Department.

There are also different opinions on the efficacy of thrombolysis and whether its administration could have prevented death, particularly in the time prior to his deterioration requiring sole focus on emergency resuscitation.

The hospital has received the autopsy report and considered the findings. The hospital has co-operated with the coronial investigation and clinically reviewed the events and circumstances surrounding Mr Bradshaw's attendance, assessment and treatment. The factual sequence of events has been reviewed and considered by the treating team.

The hospital has responded by implementing a number of the recommendations contained in the clinical review.

In all these circumstances it is not considered in the public interest that an inquest should be convened.

Chris Clements
Coroner
20 June 2016