QUEENSLAND COURTS

CORONERS COURT OF QUEENSLAND

FINDINGS OF INVESTIGATION

CITATION:	**Non-inquest findings into the death of Mr P**
TITLE OF COURT:	Coroners Court
JURISDICTION:	CAIRNS
DATE:	24 July 2019
FILE NO(s):	2014/3687
FINDINGS OF:	A/Coroner Carmody
CATCHWORDS:	CORONERS: Health-care related death; severe hypoxic respiratory failure; pneumonia; pulmonary embolism; deep vein thrombosis; skin infection; morbid obesity; cholesterolosis; diverticular disease; cardiac arrest; computerised pulmonary angiogram; Ayr Hospital; The Townsville Hospital

Table of Contents

Background

1. Mr P, 47 years of age, lived in Ayr. Mr P had diabetes mellitus type 2, hypertension, osteoarthritis and obstructive sleep apnoea. He was medicated accordingly. He was also morbidly obese weighing 155 kilograms and 160 cm tall. He was known to have a tendency for blood clots in the arteries of the lung or pulmonary embolism (PE).

2. On 24 September 2014 he consulted his local General Practitioner (GP) after hurting himself lifting dumb-bells several weeks earlier and had a productive cough and dyspoena (laboured breathing). His right calf and foot had been swollen for some months.[1] Suspecting abdominal wall hernia and either/or both pulmonary embolism (PE) and pneumonia, Mr P's GP referred him to the Ayr Hospital (AH) for urgent diagnostic imaging and further investigation of oxygen levels and lung changes.

3. He was moved from the AH to The Townsville Hospital (TTH) later the same day with severe hypoxic respiratory failure, ongoing treatment of left-sided pneumonia and for investigation of suspected PE given he had a skin infection with pain and swelling of his right lower leg (on a background of obesity). He was assessed in Emergency Department (ED) and admitted to Intensive Care Unit (ICU) with a primary diagnosis of pneumonia.

4. The specialist ICU doctor did not believe that the swollen leg, which was noted to have been present for months, represented a deep vein thrombosis (DVT) and did not require ultrasound or other investigation. Neither DVT nor PE were included or excluded before he was transferred to a medical ward less than 24 hours later, given antibiotics and transferred back to AH a week later after marked improvement. The calf swelling had settled was not painful and now bilateral. Assuming the PE had been evaluated and excluded the Senior Medical Officer (SMO) at AH considered the residual symptoms consistent with community acquired pneumonia and did not give any follow up treatment for clotting. His slow recovery and continuing oxygen dependency were put down to restrictive lung disease due to his morbid obesity and obstructive sleep apnoea. On 5 October 2014, Mr P's blood pressure dramatically decreased, he went into shock and cardiac arrest before dying awaiting retransfer to TTH.

Autopsy

5. At post-mortem there were multiple emboli throughout the pulmonary vascular system and evidence of DVT in the pelvic veins and in the lower limbs as well as diverticular disease and cholesterolosis of the gallbladder.

[1] GP's report lists Mr P's history as (1)Hurt himself using dumb-bells several weeks ago- noted pain middle of abdomen the next day - ?lump, only hurts if lifting things, (2)Noted dyspnoea last few days, following cough with fever, Productive cough. Has had a swollen R calf and foot for several months. He observed Mr P's abdomen was "mildly tender L upper/mid abdo."

6. Death was certified by the pathologist as due to natural causes *viz*., pulmonary thromboemboli from obesity related DVT.

Clinical Narrative

7. Mr P had presented to his GP in Ayr with a productive cough, high blood pressure, a painful swollen right calf and foot with redness, reporting shortness of breath (dyspnoea) and abdominal wall soreness.

8. The GP's assessment of the airway problem on examination is recorded as "? pneumonia? consolidation (disease) secondary to PE". I take from that the GP formed a preliminary diagnosis of pneumonia and/or PE from a deep venous thrombosis (DVT) in the lower limbs as the likely explanation of Mr P's symptoms.

9. Mr P was referred to AH for investigation and recommended admission for the management of hypoxia (respiratory distress from deficient oxygen reaching the tissues and organs) and urgent imaging including a Computerised Pulmonary Angiogram (CTPA) and, if the chest x-ray is essentially normal, a Venous Doppler ultrasound for the right lower limbs to exclude PE.

10. On admission to AH, Mr P was assessed by the SMO since 2014 with 9 years post registration experience, who noted the swollen right calf, which was now tender, and oedematous right foot and identified respiratory failure secondary to bilateral pneumonia with oxygen saturations at 60% and likely underlying poor respiratory reserve, secondary to morbid obesity.

11. The chest x-rays showed consolidation changes confined to the left lung field which, given the state of hypoxia, needed further investigation. The Senior Medical Officer formed the opinion that the air-space disease on the chest X-ray was probably secondary to a PE because, as he explained in response to a Form 25 information requirement:

> "…he had a skin infection with pain and swelling of his right lower leg (on a background of morbid obesity). As such I commenced him on the treatment for PE (with a DVT) and discussed (CTPA for further evaluation) … with the accepting team in Townsville*."*

12. In his referral letter to the TTH the SMO said that Mr P, "**will need a CTPA tonight**" to exclude a pulmonary embolus and has been commenced 100mg of Clexane, a low weight heparin initiating anticoagulation, should the clinical suspicion of a PE be confirmed by CTPA at TTH.

13. He also mentioned that Mr P had type 1 respiratory failure with a normal carbon dioxide (45mm Mercury) and hypoxaemia (a condition where there is a low arterial oxygen supply within the body).

Townsville Hospital

14. Mr P was admitted to TTH just before 11.30 pm on 24 September 2014. He had a swollen right calf, sore abdominal wall from stress or trauma, breathlessness, was immobile, morbidly obese and high blood pressure.

15. The ED noted respiratory failure and morbid obesity with a proposal to treat for severe bilateral community acquired pneumonia.

16. The Locum Staff Specialist says Mr P was treated for both PE and pneumonia, but the ICU team were convinced that pneumonia was a very good reason for the respiratory failure and that DVT with subsequent PE was unlikely, a planned urgent venous Doppler of the leg was deferred or cancelled.

17. The Emergency Medicine Registrar asserts that the PE diagnosis is always considered as a differential in cases of severe hypoxic respiratory failure but in this case there was good evidence to support a diagnosis of severe bilateral pneumonia as the primary cause of respiratory failure, given Mr P's history of infective symptoms, clinical examination findings and progressive chest X-ray signs of bilateral pneumonia. However this diagnosis ignores the swelling and proneness to PE.

18. From the outset the admitting ICU Consultant adopted a working diagnosis of bilateral pneumonia. In the ICU plan a +/- Doppler ultrasound was not seen as being needed "at this stage" because the right leg swelling was longstanding. Even more significantly, the urgent CTPA requested by AH was not ordered and TTH has not explained why, even though it concedes ICU patients are high blood clot risks and a CTPA should always be done at some stage.

19. Mr P was administered heparin sodium (amongst other drugs) and transferred to the general ward for continuing antibiotics and oxygen therapy.

Return to Ayr Hospital

20. Mr P was discharged back to the AH on 1 October 2014.

21. The discharge summary from TTH notes the reasons for admission as:

> "47 year old male presenting with increasing shortness of breath, cough and general feeling unwell for a couple of weeks". Principal diagnosis as "severe bilateral pneumonia and respiratory failure - ICU admission" and other active problems as "Type 2 diabetes mellitus, Hypertension, Osteoarthritis, Morbid obesity".

22. In the inpatient clinical management section of the discharge summary, nonspecific venous thromboembolism (VTE) prophylaxis (preventative action) anticoagulation or blood thinning via herapin is listed. (PE and DVT

are VTEs). Recommendations to the GP included referring Mr P for sleep studies and to monitor Mr P's Hb levels and consider further investigation of possible iron deficient anaemia.

23. Mr P was to continue IV antibiotics for a further three days and then change to oral antibiotics. He had had a couple of hypertensive episodes and had become hypotensive. Thus, antihypertensive medication was to be temporarily withheld.

24. A chest X-ray was to occur in four weeks post discharge to ensure resolution of the bilateral lower respiratory tract infection.

25. On the day prior to transfer back to AH, Mr P's oxygen saturation on room air was 88% improving to 92% on 2 litres of oxygen. He had hyperkalaemia (an electrolyte imbalance with a high level of potassium in blood) and was treated accordingly.

26. On his return to AH Mr P was deemed to have symptoms consistent with the community acquired pneumonia diagnosis given by the TTH specialist team. No clinical review was conducted to ascertain one way or the other whether the recommended CTPA procedure had been carried out at TTH but the Senior Medical Officer at the AH "…assumed that …it had been considered and excluded…" on evaluation in Townsville and did not further consider the possibility of PE or DVT. He put down the bilateral swelling to both lower limbs to morbid obesity and obstructive sleep apnoea.

27. Mr P's oxygen levels continued to drop at night. This was put down to his obstructive sleep apnoea and poor respiratory reserve with resolving pneumonia. He was managed symptomatically with continuous positive airway pressure during the evenings.

28. On 5 October 2014 Mr P's blood pressure decreased after returning from a shower. He went into shock with peripheral shut down. He was managed with oxygen, fluids, vasopressors and it was decided to retrieve him back to TTH. Whilst awaiting transfer he went into cardiac arrest likely due to cardiogenic or obstructive shock. He was treated for both conditions but did not respond to resuscitation efforts and was sadly declared deceased 45 minutes later.

Townsville Hospital's reply to Ms P's concerns

29. On 22 October Mr P's sister, Ms P, wrote to TTH questioning the suitability of his health care as a patient there.

30. On 3 December 2014, TTH CEO, Ms S, relevantly responded as follows:

> " …. Mr P was transferred to the TTH with respiratory failure. The doctor in Ayr considered pneumonia and a clot in the lung as possible causes in his referral letter.

On assessment in Emergency Department at the Townsville Hospital an urgent chest x-ray revealed changes consistent with pneumonia involving more than one lobe of the lungs. This diagnosis was also supported by blood test results that pointed to an infection. Mr P commenced on antibiotics for infection, heparin for prevention against blood clots (deep vein thrombosis), high flow oxygen and intravenous fluids without delay.

Mr P was transferred to ICU for observation because he had respiratory failure from his pneumonia…. his condition progressively improved and he was therefore transferred to the medical ward after 18 hours. While in ICU the doctors reviewed the possible causes of his respiratory failure and felt that the degree of pneumonia was severe enough to explain his condition.

Mr P's treatment was continued in the medical ward where his condition progressively improved. Gradually his treatment was scaled down with intravenous fluid stopping and he required less additional oxygen to maintain a reasonable level of oxygen in his blood. He was still receiving antibiotics for the pneumonia and heparin to prevent deep vein thrombosis (blood clots).

The treating team saw Mr P on the morning of the day he was transferred to Ayr and he was doing reasonably well. He was up and about and walking independently. There was no worsening shortness of breath or chest pain which are symptoms of pulmonary embolus.

Mr P had a normal blood pressure and normal oxygen levels in his blood.

The plan on his transfer back to Ayr Hospital was for him to continue antibiotics for his severe pneumonia. He was to continue heparin, to prevent blood clots while in hospital. ... Unfortunately, whilst in Ayr Hospital, he deteriorated a few days later and died. The post-mortem showed extensive pulmonary embolus (clots in the lungs). There was also marked congestion and oedema of the lungs.

Mr P's symptoms were consistent with pneumonia causing respiratory failure and he responded well to antibiotics and other supportive care. It is unlikely that he had a pulmonary embolus (clot in the lungs) at initial presentation to Townsville Hospital because he would not have improved without specific treatment for that.

Unfortunately, any patient who is very unwell for another reason is always at greatly increased risk of a clot. In a small number of hospitalised patients, heparin fails to give them the protection against deep vein thrombosis (blood clots) and this is difficult to predict. During his admission there was nothing unusual about his

recovery to suspect pulmonary embolus (clot in the lungs).
The senior clinicians who advised on Mr P's care on transfer to the Emergency Department and to ICU all felt his symptoms related to his severe pneumonia. He had improved significantly after he received treatment for his pneumonia and did not show any signs of a blood clot (DVT or pulmonary embolus).

The Doctors did recognise that Mr P was at risk of a blood clot (DVT or pulmonary embolus) and he was given preventative treatment…."

Response of Ms P

31. Ms P did not accept that response. She raised the following concerns:

> "They say that my brother was up and about walking; he walked very little, just to the bathroom and would be out of breath, and that was with the oxygen and he would take a lot to get his breath back."
>
> "They state that he had normal oxygen levels in the blood the morning he was transferred back to Ayr. He was still on oxygen because I asked Mr P that night when I rang him, he had just been to the toilet and was still getting his breath back. He had to take the oxygen to the toilet with him as he couldn't do without it."
>
> "The Townsville Hospital even had the gall to enclose a form for me to fill out asking me if I was happy with their performance; talk about rubbing salt into the wound."

The Clinical Forensic Medicine Unit (Forensic and Scientific Services - FSS) Report 13 February 2015

32. Dr Griffiths, Forensic Medical Officer reported on the diagnosis and clinical assessment of PE and critiques Mr P's health care at AH and TTH based on a review of medical records, the TTH response to Ms P and autopsy report.

33. Noting that pulmonary embolism remains a complex area of medicine and diagnoses Dr Griffiths observes:

> "It has been long known that a fatal pulmonary embolus is often preceded by a number of "herald" emboli, occurring over time, and giving rise to a constellation of symptoms and signs including shortness of breath, hypoxaemia and cough."

34. He goes on:

> "Algorithms now exist, which claim to have predictive value, and are available to clinicians to assist in the determination of the most

> appropriate clinical pathway based on the probability of suspected pulmonary embolus.
>
> There is also a scoring system (the Wells Score) ... The presence of a deep venous thrombosis (DVT) plus the additional signs and symptoms of a pulmonary embolus, would attract a high Wells Score which is increased even more by a history of recent immobility of any cause. In these circumstances, the algorithm would indicate that a CTPA is the most appropriate investigation.
>
> A blood test for a "D-Dimer" which is a fibrin degradation product and a test ...is only of predictive value where the probability is low. If the D-Dimer is positive, this would add nothing to the pre-test probability where the Wells Score is high. In circumstances where the D-Dimer is positive, but a Wells Score is low, according to the current algorithm, an ultrasound investigation of the lower limbs for a possible DVT (to explain the positive D-Dimer) would normally be recommended*.* "

35. Dr Griffiths suggests that if a Wells Score had been employed, the pre-test probability for a pulmonary embolus would have been high, and at least, a Duplex Ultrasound of the lower limbs would have been ordered.

Management at Townsville Hospital

36. Of Mr P's treatment at TTH Dr Griffiths remarked:

> *"*From the outset (24 September 2014) until his eventual discharge on 1 October …. the working diagnosis… remained a bilateral pneumonia."
>
> *"*No other diagnostic possibility to explain Mr P's previous dyspnoea and hypoxia appears to have been considered despite the presence of a swollen right calf, which was noted on arrival, and the Senior Medical Officer's concern that his patient might have a pulmonary embolus and his request for an urgent CTPA."

37. The right calf swelling was therefore not investigated with a Duplex Venous Ultrasound.

38. Dr Griffiths also observes:

> "Beginning with the Emergency Medicine Registrar who performed the initial assessment at 1255 hours on the 24th September, the medical consultant, or possibly Locum Staff Specialist, and a later ICU Consultant, the Senior Medical Officer's request for a CTPA to be performed on arrival, seems to have been overlooked, or at least, not referred to in any of the documentation that I have read."

39. And adds:

> "[the Senior Medical Officer's] clinical concerns with a request for a CTPA, as well as those of [the GP], should have been addressed… They appear not to have been."

40. Dr Griffiths also reported that there was a possibility that the hypotensive episode on 29 September 2014 and postural dizziness when Mr P's blood pressure dropped below 90, was embolic in origin.

41. Dr Griffiths concluded that:

> "[I]t would be a brave clinician indeed who would choose to overlook a request from a senior colleague for an urgent Computerised Pulmonary Angiogram, or to dismiss the likelihood of a pulmonary embolus in a patient with a swollen tender right calf, who had severe hypoxia, and who was short of breath."

42. Dr Griffiths questioned why therapeutic anticoagulation, even empirically, was not commenced until a pulmonary embolus was excluded as there were no contraindications to using a Low Molecular Weight Heparin at an uncapped dose of 1.5mg/kg as Mr P had normal renal function and had not undergone recent surgery.

43. Prophylaxis against venous thromboembolism (VTE) was commenced at TTH (heparin 5000iu bd) which was enough to prevent a VTE from developing but insufficient to treat an established PE. It is not clear why this was given; perhaps it was because he was at risk of developing a clot given his weight and other co-morbidities unless it was a routine precaution against hospital acquired DVT.

Comments on Townsville Hospital's response to Ms P

44. Dr Griffiths also considered the matters raised in this response. He takes issue with many assertions.

45. *-the diagnosis of pneumonia was supported by blood test results that pointed to an infection*

Dr Griffiths notes, no mention of elevated troponin-1 normally a marker of myocardial cell damage, but may also be elevated in PE and that the increases in Mr P's white cell count and elevation of an Acute Phase Reactant known as C-Reactive Protein can indicate several non-infective disease states, including PE.

46. *-in the ICU opinion the extent of respiratory failure was explained by the severity of the bilateral pneumonia*

Dr Griffiths says the clinical grounds relied on to form the opinion, without imaging, are not adequately explained and contends that the "…degree of

consolidation in one segment of a lung field, in a patient with no known pre-existing chronic lung disease could not explain the finding on oximetry of an oxygen saturation of 60% on room air" on his arrival at AH on 24 September 2014.

47. *-normal blood pressure and oxygen levels*

Dr Griffiths does not accept that Mr P had normal blood pressure and oxygen levels in his blood. He says Mr P's O2 levels were never within a normal range during his TTH admission without supplemental oxygen. His blood pressure also dropped during the final 2 days in Townsville requiring an adjustment of his normal anti-hypertensive regime. He suggests this circulatory collapse and drop in pressure and associate presyncope could have been an embolic event.

48. *-it is unlikely that he had a pulmonary embolus at the initial presentation to TTH*

According to Dr Griffiths this "extraordinary assertion" is not supported by the autopsy finding of multiple thromboemboli in the smaller pulmonary vessels suggesting that propagation of probable 'herald' emboli had been occurring over time and asks rhetorically "How could the physicians at a teaching hospital form the view that PE was "unlikely" when they needed to be certain".

49. *-heraprin was used as a preventative of PE or DVT*

This statement is interpreted by Dr Griffiths as an attempt to (wrongly) imply that, despite prophylaxis, the genesis of the fatal PE was TTH or AH and did not propagate from pre-existing venous thrombosis in the lower limbs or pelvis.

Townsville Hospital Morbidity and Mortality Review 5 March 2015

50. Dr Griffiths' opinion prompted a discussion with the Clinical Director of the TTH resulting in the clinical management of Mr P being referred to the Morbidity and Review Committee.

51. The TTH Review Forum comprising the Chair and Locum Staff Specialist who had attended on Mr P, felt Mr P's presentation (symptoms, CXR, labs) were consistent with pneumonia and a CTPA was not required at any point during his Townsville admission but did not state why it was not necessary for TTH to investigate an alternative view of the symptoms including PE.

52. The forum noted that, "…the clinician felt the patient's swollen leg did not represent a DVT. That person probably felt another diagnosis was present, but they did not mention that in the notes". While accepting the need to revisit diagnoses if the pieces do not fit, the committee did not reach any conclusion as to whether the medical team should have revisited the PE issue before discharge, in the context of this case, but did note that the day prior to discharge the doctor's notes state there was no pain or swelling of

legs.

53. Other reported points of discussion mentioned were:

54. *Whether Mr P had DVT and when it developed?*

The committee could not determine the age of the PE found on autopsy based on available information. Therefore, it was not possible to know whether Mr P had DVT for months, or it may have developed during his hospitalisation or even on the day he died.

55. *Did the patient receive adequate DVT prophylaxis?*

In addressing this issue, the forum noted Mr P was 160 kg. No mechanical prophylaxis was ordered.

56. *Was the usual dose of anticoagulant adequate?*

Without answering this question, the committee mentioned that Mr P was given Heparin 5000 units BD at TTH and enoxaparin 40 mg on return transfer to AH.

57. The outcome of the analysis of the incident by the committee was a suggested review of DVT prophylaxis guidelines to see if there was evidence for different recommendations in the obese population and to improve TTH practices.

58. TTH later reported the Heads of Medical Departments and Medication Management Committee considered this issue and resolved to use the Australia and New Zealand Best Practice Guidelines 5th Edition. A link to this document was established on the TTH intranet, and hard copies were distributed to medical staff and across the wards.

Townville Hospital – Locum Staff Specialist

59. In his response to inquiries about his care of Mr P when he was a locum at TTH of 16 November 2016, the Locum Staff Specialist notes that on the day before his admission Mr P started coughing up blood stained sputum and was short of breath. Mr P was said to have noticed some pain, redness and swelling in the right leg for some months. At Ayr Hospital the doctor made two possible diagnoses – PE and pneumonia. The referral note states, “The patient will need CTPA tonight”.

60. Mr P was treated for both conditions, oxygen at 15 litres per minute, benzyl penicillin and azithoromycin intravenously and clexane 100mg subcutaneously twice daily (the dose of clexane would have been suboptimal as the patient weighed over 140kg).

61. Mr P was seen at the ED of TTH later that evening and following assessment,

an urgent chest X-ray and blood tests were organized. The ICU Registrar was called and determined he was unwell enough to be in ICU but did not need non-invasive ventilation yet. The X-ray showed changes consistent with multi-lobar pneumonia. Benzyl penicillin was ceased and Tazocin commenced for severe pneumonia.

> "In ICU the issue of pulmonary embolus was revisited. It was confirmed that the leg swelling had been there for over a year. With obvious severe pneumonia as revealed by the chest X-ray and raised white cells and C-reactive protein the ICU team were convinced that there was a very good reason for the respiratory failure and that deep vein thrombosis with subsequent pulmonary embolus was unlikely so a planned venous Doppler of the leg was cancelled."

62. After twenty hours Mr P was transferred out of ICU and on 26 September 2014 the Locum staff Specialist saw him on the ward round for the first time. He was "also convinced that severe multi-lobar pneumonia was the most likely cause for his respiratory distress."

63. The Locum Staff Specialist concludes:

> "…. although PE was considered initially by the doctor at AH, when combining the patient's symptoms, fever, and respiratory failure associated with abnormal chest X-rays and raised markers of infection/inflammation, pneumonia was most likely. The ICU team were convinced that severe pneumonia was the most likely cause for his respiratory failure. When I took over his care when the patient transferred to the ward I reviewed him and looked at his test results and agreed with the assessment, diagnosis, reasoning and management plan which was to treat with antibiotic for severe pneumonia…..I was satisfied that there was nothing else going on."

Townsville Hospital - Emergency Medicine Registrar

64. The Emergency Medicine Registrar does not recall Mr P and reviewed the medical notes to give her response. She was involved with his care at the ED until his transfer to ICU at 1:15am.

65. The Emergency Medicine Registrar notes Mr P's background of morbid obesity, type 2 diabetes, mellitus, hypertension and osteoarthritis and that he had been unwell for 2 weeks with symptoms for a cold. He developed increasing dyspnoea, presented with a fever as well as brown and yellow sputum. The Emergency Medicine Registrar notes that Mr P's GP recorded his symptoms as, including temperature, pulse, blood pressure and oxygen saturations of 70%. A chest X-ray was, "interpreted as having significant opacification of left mid and lower lung fields."

66. The Registrar notes Mr P was referred to AH Senior Medical Officer who made differential diagnosis of respiratory failure due to pneumonia and pulmonary embolism. Swelling of the right calf was diagnosed as erysipelas.

67. There was no written handover note from QAS within the clinical records but she says, "The patient was accompanied by a referral letter from the Senior Medical Officer and his Ayr medical records."

68. The Emergency Medicine Registrar examined Mr P noting his respiratory distress and "there was marked swelling of the right leg which Mr P advised me was a longstanding issue."

69. She concludes:

> "My impression after assessment ……was that of severe bilateral pneumonia with respiratory failure complicated by morbid obesity. The diagnosis of pulmonary embolism is always considered as a differential in cases of severe hypoxic respiratory failure. In this case there was good evidence to support a diagnosis of severe bilateral pneumonia as the primary cause of respiratory failure, given his history of infective symptoms, clinical examination findings and progressive chest X-ray signs of bilateral pneumonia. The relevant negative for pulmonary embolism…. Were the absence of any tachycardia, hypotension, ECG feature of right heart strain, and most prominent, the presence of an alternative diagnosis that would completely explain his respiratory compromise."

Townville Hospital – ICU Consultant

70. The ICU Consultant is and was at the relevant time the Staff Specialist at TTH. He has no direct recollection of Mr P and his care and bases his comments on the notes made during Mr P's stay in ICU.

71. Mr P was admitted to ICU at approximately 2am on 25 September 2014 on referral from the ED Registrar for:

> "Type 1 respiratory failure deemed (based on their examination) to be due to bilateral lower respiratory tract infection. …. It was noted on admission that the patient had bilaterally swollen lower limbs below the knee, but that the right was larger than the left (although the exact size was not noted.)"
>
> "The notes reflect that the patient maintained that this swelling (and size difference) was chronic, with a time course of up to 1 year. The patient denied any associated pain and was noted to be ambulant and active in the period preceding admission."

72. The following morning, he was discharged after noting that Mr P's condition had not deteriorated. Based on clinical examination and investigation findings (blood tests and chest X-rays) the diagnosis of pneumonia was confirmed. Accordingly, he says:

> "…. there was no clinical reason to suspect pulmonary embolism other than an increased oxygen requirement. There were no findings usually associated with such a diagnosis (eg tachycardia, hypotension, ECG changes, chest pain)."
>
> "The right calf swelling was again noted, but in light of its chronic nature it was deemed that an urgent Doppler ultrasound scan of the deep veins of his legs was not indicated but rather this could be deferred to an elective investigation at a later point during the patient's admission."

73. The ICU Consultant makes the general comment that, "the diagnosis of DVT is largely based on clinical suspicion and various criteria have been developed to help guide relevant investigation surrounding such suspicion. He adds, "None of these criteria are infallible but assist in reducing the number of unnecessary tests on the basis of clinical probability." He also notes that Wells' Score for DVT and PE are validated and commonly used scoring systems, and PERC score is similarly often used to rule out the need to investigate for PE in low risk patients.

74. The ICU Consultant helpfully attached two review articles - *Meta-Analysis: The value of Clinical Assessment in the Diagnosis of DVT* and *PE: the Diagnosis, Risk-stratification, Treatment and Disposition of Emergency Department Patients"* presumably to support his approach and concludes:

75. Attachment 1: Review: *Meta Analysis: the value of Clinical Assessment in the Diagnosis of DVT*
The Review considers the literature to determine whether physician's empirical judgments, clinical findings, and risk scores affect the likelihood of detecting thrombosis with venography, ultrasonography, or plethysmography in adults with suspected DVT but not where there is an early alternative diagnosis.

76. Its purpose is stated as "…to determine whether [1] clinical findings, **[2] risk scores,** and [3] physicians' empirical judgments affect the likelihood of detecting DVT on definitive testing suggesting that clinical findings *alone* should not constitute the basis for assessing the likelihood of detecting DVT. It is increasingly being recognized that clinical diagnosis should be based on systematic evaluation of the scientific evidence."

77. The review evaluated 13 characteristics for making assessments including:
- Calf pain;
- Calf swelling;
- Prior DVT;

- Recent immobilization;
- Obesity;
- Difference in calf diameter;
- Calf tenderness;
- Erythema [redness of skin];
- Oedema

78. It concludes that the Wells Score seems to perform better in populations that exclude patients with previous thromboembolism and to perform worse in older cohorts. Mr P belonged to both categories.

79. In the absence of a history of calf swelling or no difference in calf diameter on examination slightly reduces the likelihood of DVT. According to Dr Griffiths, there was no, or only cursory consideration of this factor by TTH.

80. Attachment 2: Review: *Pulmonary embolism: the diagnosis, risk stratification, treatment and disposition of emergency department patients.*

This review observes that the diagnosis or exclusion of pulmonary embolism remains challenging for emergency physicians as symptoms can be vague or share feature with other common diagnoses. Testing can be complicated with some like the D-Dimer giving false positive, and imaging carrying the risk of radiation and contrast dye expose. It says it is therefore incumbent on emergency physicians to be both vigilant and thoughtful about this diagnosis.

a) The article cautions that emergency physicians must be able to risk-stratify patients to ensure the appropriate disposition.

b) Obesity is a known and common acquired risk factor for venous thromboembolism (VTE) and data has found that among the most obese subjects (body mass index >35), there was a 6 fold increase in risk when compared to normal weight subjects. Provoking risk factors such as limb immobility significantly increase VTE risk. This is usually associated with prolonged limb immobility and typically occurs after about 72 hours of acute limb immobility.

c) The review also advocates for the pulmonary embolism rule-out criteria (PERC) to identify patients in whom the risk of testing outweighs the benefits. Mr P had two of the eight rule out criteria - unilateral leg swelling and haemoptysis. It concludes that a low pre-test probability and a negative PERC effectively rules out PE with a false negative rate lower than the test-threshold.

d) **DVT diagnosis** - The review states the diagnostic test of choice for detection of proximal DVT is venous ultrasonography – it is always readily available in Emergency Departments and poses little to no risk to the patient. But since half of patients with PE have no imaging evidence of DVT, a negative venous ultrasound is not enough to rule it out. Analyses suggest that a non-invasive strategy **combining**

ultrasound and D-Dimer testing as the first diagnostic test for PE may be the most cost-effective approach.

e) **PE diagnosis** – The standard imaging modality for diagnosing PE is contrast computed tomography pulmonary angiography (CTPA). This was found to have specificity of >95%. Studies confirmed that CTPA can safely rule out PE in patients with a high pre-test probability of PE or a positive D-Dimer.

f) **Treatment** – Treatment of patients with PE will depend on multiple factors including the acuteness of the PE but anticoagulation usage is essential in halting the propagation of thromboembolism. High risk patients may require additional treatment measures to reduce the clot burden.

Supplementary FSS Report 31 December 2018

81. Following on from responses from the Specialist, the Registrar and the Consultant, the Forensic Medical Officer Dr Griffiths gave a supplementary opinion to the effect that:

> "From the time of Mr P's acute admission to TTH ICU via the Department of Emergency Medicine, the working diagnosis remained that of a severe bilateral pneumonia, and he was treated accordingly."
>
> "Despite the request for a CTPA from the referring SMO at AH, an imaging procedure considered to be the 'gold standard' diagnostic imaging for suspected PE, all of the clinicians involved in the care of Mr P were of the view that an alternative diagnosis satisfactorily explained his symptoms."

82. Dr Griffiths remarks it is uncertain whether the three treating clinicians were aware of Senior Medical Officer's request for a CTPA – "It is possible that they may not have been, although The Emergency Medicine Registrar reported that all of the referral letters had been forwarded to the ICU on his admission and specifically mentions the CTPA request in his statement."

83. Even after continuous intravenous antibiotic regime and high flow and then reducing oxygen at the time of discharge on 1 October 2014, Mr P required supplemental oxygen at a flow rate of 2 litres per minute. Oxygen saturation on room air fell to just 88% despite five or six days of intravenous antibiotics. "No red flags were raised by this finding, although body habitus might have been a contributing factor."

84. In comments on the *Review: Meta–Analysis: the value of Clinical Assessment in the diagnosis of DVT,* attached to ICU consultant's response in Dr Griffiths notes:

- even on the ICU Consultant's reasoning there should have been clinical suspicion held by the specialist team at TTH based on the presenting

symptoms and Senior Medical Officer's referral,

- clinical findings *alone* should not have constituted the basis for definitively assessing the likelihood of DVT. Risk scores and empirical judgments also affect the chances of detecting DVT. Wells scoring has a performance advantage over empirical assessment because it is standardized, reproducible and based on more studies,
- many technologies can be used to diagnose DVT from the cheap and simple but inaccurate (D-Dimer test) to the accurate but expensive and technically challenging (venography),
- it is increasingly being accepted that clinical diagnosis should be based on systematic evaluation of scientific evidence,
- the review has limited relevance where, as here, there DVT was not suspected and there was an early alternative diagnosis,
- a low Wells Score substantially decreases the likelihood of DVT and indicates that a simple non- invasive test such as D-Dimer assay may suffice to rule out DVT,
- Mr P was in both cohorts where Wells Scoring performs better and worse,
- the absence of calf swelling only slightly lowers the chance of DVT and TTH gave no or only cursory consideration to this factor,
- the ICU Consultant's assertion that diagnosis of DVT is based largely on clinical suspicion is at odds with the review's suggestion that a Wells Score assessment, and if low (which substantially degreases the likelihood) a simple non-invasive test such as D-Dimer may be enough to rule out DVT,
- CPTA was found to have a specificity of .95% and can safely rule out PE in patients with high pre-test probability of PE or a positive D-Dimer.

Townsville Hospital - comments on draft report

85. The TTH responded in detail to proposed findings on 27 June 2019.

86. The executive director-clinical governance relies heavily on the contrary opinions of the Director of ICU, and on the compelling data supporting the working pneumonia diagnosis, the high acuity treatment of that condition, daily clinical re-evaluation of the diagnosis and signs of recovery to counter Dr Griffiths criticism of the ICU intervention. She, in fact, reaffirms that if Mr P presented today with the same body mass index, longstanding swollen leg and x-rays, he would not have been treated any differently and treated for pneumonia which unlike the alternative DVT/PE diagnosis satisfactorily explained all his acute symptoms.

87. Notably, The TTH does not use the Wells Scoring system to risk stratify for DVT in ICU because:

    a) it is an emergency tool and not validated in a critically ill setting;

b) all ICU patients are at high risk for DVT anyway and Mr P was effectively managed as such with heparin thromboprophylaxis;

c) the risks associated with ordering involved testing of critically unwell ICU patients and potential compilations of PE treatment are more complex and difficult to manage than PE itself;

d) based on the information in the draft findings and medical records Mr P's pre-test probability score would have been moderate (27.8%) not high as Dr Griffiths suggests ,

e) D-Dimer in the Director's view is inadequate for ruling PE in or out and the degree of hypoaemia was not disproportionate to putative pneumonia and consistent with the clinical and radiological picture.

88. All these comments have been carefully considered as part of finalising the following findings.

Forensic issues

89. Mr P died of cardiac arrest on 5 October 2014 when he was an inpatient of AH.

90. The direct cause of Mr P's death was found at autopsy to be extensive PE, and the likely source of these, was DVT in the lower limbs secondary to morbid obesity.

91. The TTH CEO described the death to Mr P's sister as, "… an unexpected but known complication of severe illness even when anticoagulant is provided to prevent blood clotting".

92. However, Dr Griffiths says reading the clinical narrative and looking at the results of the autopsy it is hard to escape the conclusion, that the "… fatal PE (was probably)… preceded by a number of cascading "herald" emboli giving rise to a constellation of symptoms and signs including shortness of breath, hypoxaemia and cough and more than likely shedding … from a thrombus in the iliofemoral vein of the pelvis over time until the 5th October 2014."

93. Clearly, issues arise about the adequacy of his clinical treatment for severe pneumonia symptoms at TTH and AH in the context of the fatal PE.

94. The next of kin understandably feels that the death should, could and would have be avoided if the PE had been suspected and detected at TTH so a care plan for removing or at least reducing its lethal potential could have been developed and actioned between 29 September and 1 October 2014.

95. Based on the available literature and clinical information, PE is a common cause of cardiovascular death with mortality varying widely depending on factors such as age, comorbidities and degree of stability. It occurs when blood clots are formed in the deep venous system, dislodge and migrate

through the heart to the pulmonary structures and embolize (congest). The blockage causes a sudden rise in pulmonary artery pressure and decreases cardiac output. They overwhelm the body's natural anticoagulant mechanisms and can lead to circulatory collapse. DVT tends to form in the lower extremities and causes associated swelling and thrombosis propagation.

96. Multiple risk factors increase the risk of DVT and PE. Some are inherited while others, like age, venous insufficiency, obesity, previous VTE and hypertension are acquired over time. Common non-disease related triggers are acute and prolonged limb immobility and recent trauma. These can overlap and aggravate other factors. Specifically, morbidly obese patients are six times more likely to have DVT or PE than their normal weighted counterparts.

97. Risk stratification is important to prognosis, management and chances of recovery.

98. The patient's pre-test probability of the diagnosis determines whether and what testing is indicated. Relevantly, Wells scoring indicates where PE or an alternative is the most likely diagnosis based on points awarded for each positive answer to six questions. The categories of pre-test probability of PE are low, intermediate and high. A low Wells Score substantially decreases the likelihood of DVT and indicates that a simple non-invasive test, such as D-Dimer assay, may be enough to rule out DVT. Wells scoring has advantages over empirical assessment because it is standardised and reproducible and its estimated performance is based on more studies.

99. The preferred diagnostic modality for DVT is venous ultrasonography with loss of vein compressibility being diagnostic. It is standard in Emergency Departments. However, as 50% of patients with PE have no imaging evidence a negative ultrasound is insufficient to rule it out while ultrasound evidence and other non-invasive blood test results or symptoms indicate treatment.

100. The D-Dimer blood test is an inexpensive, simple yet highly sensitive test for DVT but unless the result is negative, it is otherwise not accurate enough as a standalone exclusion because other conditions such as pneumonia can elevate the score. A negative CTPA, by contrast, can safely rule out PE in high pre-test probability of risk patients or a positive D-Dimer.

101. The severity of PE varies from asymptomatic to chronic or acute. Low risk patients have a one-year survival rate of 95% with oral anticoagulation medication according to US studies while for high risk patients intensive care may be necessary.

102. Anticoagulation with medication such as heparin is essential for stopping propagation of thromboembolism in all PE patients and other interventions such as systemic intravenous or catheter thrombolysis may be needed to improve mortality in higher risk patients.

103. Admittedly as Attachment 2 to the ICU Consultant's statement points out, clinically diagnosing or excluding DVT is challenging for emergency physicians due to its vague and shared symptoms and there is a risk of false positives with unsophisticated tests like D-Dimer and imaging carries radiation dangers. Also the value of a D-Dimer level as a marker in this case would have been limited because it is also elevated in pneumonia but this is precisely why the threshold for further investigations of a patient with symptoms or signs associated with it should always be low and the need for vigilance and mindfulness when risk stratifying to ensure proper disposition of patients is high.

104. Even Dr Griffiths concedes that when PE or DVT are suspected the choice of treatment is still an area of debate and high risk for PE patients presenting with hypoxia can deteriorate and die suddenly even with appropriate treatment.

105. Notably, despite initially being judged high risk for both DVT and PE based on the combination of symptoms and empirical assessments prior to this TTH admission the Senior Medical Officer at AH says, "Mr P would probably have scored as a low risk on the Wells Criteria (+1 for bedridden >3 day, -2 for alternative diagnosis (of pneumonia) as more likely than DVT) if it had been applied on 01/10/14 and would not have changed his approach to his health care given his clinical improvement after treatment in Townsville."

Conclusion

106. In my opinion, there is no forensic basis for characterising this death as a complication of severe pneumonia despite preventive anticoagulation medication. Rather the documentation compels a conclusion that it was due to PE propagating over time from a pre-existing DVT in the lower limbs or pelvis correctly suspected by both Ayr doctors because of the calf swelling in a morbidly obese man and a degree of hypoxia.

107. The firmly fixed TTH position that Mr P did not already have PE secondary to DVT in ICU because of his chronic leg swelling and improved oxygen levels without specific treatment for that, is at odds with the multiple emboli throughout the pulmonary vascular system and evident of DVT in the pelvic veins and lower limbs post-mortem.

108. Both the fatal PE and its DVT source were there to be suspected (if not expected), fully investigated, detected and treated when Mr P was in TTH care but opportunities for doing so were missed because the ICU team simply did not share the DVT and PE hypothesis. They found no condition usually associated with a PE diagnosis (e.g. tachycardia, hypotension, ECG changes, chest pain) and other than an increased oxygen requirement, did not see any clinical reason to suspect a VTE explanation of the symptoms.

109. Despite their availability, standard modalities for assessing suspected PE

like Wells Score and simple non-invasive blood +/- tests such as d-dimer for predicting risk levels and informing the appropriate clinical pathways were not availed of in ICU. If the working diagnosis of a bilateral pneumonia was revisited it was not altered. There were compelling reasons to treat prudentially based on a high probability of the DVT/PE diagnosis unless and until testing positively ruled it out. Pneumonia alone may have been the most likely explanation of the respiratory failure but there was not enough unequivocal empirical information to ignore or overlook signs of DVT especially considering Mr P's reported historical tendency to PE, underlying obesity, breathlessness, leg swelling and a recent injury. Despite the TTH assertions to the contrary, fuller investigations were to be reasonably expected in the circumstances.

110. The right calf swelling could have been due to a DVT but was overconfidently dismissed as an indicator because of its age. Venous ultra-sounding was deferred indefinitely, and, inexplicably, the specifically requested urgent angiogram to rule PE out was not actioned at all and the rationale for not doing so is not clearly explained or documented even though TTH itself concedes that all ICU patients are high risks of blood clots by definition and at some point receive CTPA and effective prophylaxis as standard procedures.

111. Consequently, as Dr Griffiths points out, there was no clinical suspicion documented that Mr P's breathlessness and hypoxia could have been due to propagating PE. The failure to exclude PE with a CTPA and allow for the possibility of DVT and PE being an alternative explanation for Mr P's symptoms as other practitioners had posited, took an unacceptable risk with someone else's health and may not have optimised his chances of recovery because it meant that routine anticoagulation was used in the ICU preventatively but no curative plan was put in place. In the uncertain realm, it is always better to be sure and safe than sorry.

112. Mr P may not have died when and in the way he did, if more advanced therapies against VTE were instigated sooner or at all. He was treated with antibiotics orally and given the oral anti-coagulant heparin 5000iu as a routine precaution against hospital DVT developing but insufficient to treat an established PE.

113. DVT was indicated by obesity and leg swelling. Mr P had been partially immobilized because of a recent trauma, an incident with dumb bells. Even allowing for a wide margin in which honest and informed clinical minds can reasonable differ about complex and contestable matters of degree and professional judgement without either being demonstrably right or wrong, the inescapable conclusion is that opportunities to confirm the DVT that probably pre-existed Mr P's admission to TTH ICU were missed and the chances of survival were not optimized by the failure to consider the cost-benefit of a more therapeutic over routine precautionary anti-coagulation approach. PE should have been considered as a differential if not probable diagnosis because of the severe hypoxic respiratory failure. If it had been, therapeutic blood thinning may have been prioritised and more effective in stopping the

spread of thromboembolism.

114. We will never know now whether the missed opportunity to confirm the probable DVT by diagnostic imaging or testing and treating Mr P as a PE as well as a pneumonia patient necessarily contributed to or accelerated the cause of death. It is hard to fairly say with certainty at this distance that the treatment provided at TTH or later at AH would have been any different or more intense involving stronger oral blood thinning medication or advanced intrusive anticoagulation therapies for stopping propagation of thromboembolism or possibly other interventions such as systemic intravenous or catheter thrombolysis to improve his mortality or produce better overall outcomes. What can be said with confidence however is that clinical pathway decisions could have been better informed and targeted.

115. Risk is chance plus the consequence of an adverse result. An unacceptable risk is one that is not worth taking because the potential harm is too high even if the chance of it happening is assessed as low. As this case shows, comorbidities can coexist, and one can be effectively managed to improvement while the failure to treat the other lower, but equally or more, dangerous risk, can be fatal. Where there is more than one explanation for the symptoms the cautious approach is to err on the side of including, rather than excluding reasonable possibilities, especially based on contested emergency examinations and findings.

116. The lesson is that even if there are other preferred or even more likely alternative diagnosis, best practice requires emergency and intensive care physicians to apply low level threshold standards for suspecting, and testing for DVTs and PE in high risk patients so an opportunity for a confirmed diagnosis plus customised, as opposed to generalised treatment is not missed and unexpected (but not unpredictable) adverse health care outcomes are avoided.

Identity of the deceased: Mr P

How he died: Mr P presented to AH for diagnostic imaging, investigation of oxygen levels and suspected pulmonary embolism. He was transferred to TTH where he was diagnosed with pneumonia and admitted into the Intensive Care Unit. Suspected pulmonary embolism and possible deep vein thrombosis were not further investigated by the specialist ICU doctor.

Mr P improved and was transferred to the medical ward where he was given antibiotics to treat the pneumonia. After a week, he was transferred back to AH and that night his oxygen levels decreased. The following day, Mr P went into shock with peripheral shut down. During the wait for transfer back to TTH,

	Mr P had a cardiac arrest. Mr P was not able to be revived notwithstanding resuscitation efforts.
Place of death:	Ayr Hospital, AUSTRALIA
Date of death:	05 October 2014
Cause of death:	1(a) Pulmonary thrombo-embolism 1(b) Deep vein thrombosis 1(c) Morbid obesity 2 Cholesterolosis, diverticular disease

I close the investigation.

Coroner Carmody
A/Coroner
CORONERS COURT OF QUEENSLAND - NORTHERN REGION
24 July 2019