CORONERS COURT OF THE AUSTRALIAN CAPITAL TERRITORY

Case Title:	**AN INQUEST INTO THE DEATH OF ROBERT DOUGLAS**
Citation:	**[2018] ACTCD 2**
Hearing Dates:	21 to 25 November 2016 & 26 to 28 April 2017
Final Submissions:	27 November 2017
Date of Findings:	30 January 2018
Before:	Coroner Theakston
Findings and Recommendations	See Annexure A
Legislation Cited:	*Coroners Act 1997* (ACT)
Cases Cited:	*Briginshaw v Briginshaw* [1938] HCA 34 (30 June 1938)
Appearances and Representation:	Mr G Blank as Counsel Assisting the Coroner, instructed by Ms S Baker-Goldsmith of the Australian Capital Territory Coroners Court. Mr I Bradfield for Mr Douglas' family, instructed by Elringtons Lawyers. Mr H Chiu for the Australian Capital Territory, instructed by ACT Government Solicitor. Mr C O'Neill and Ms McPhee for Dr Sayed Ali, instructed by HWL Ebsworth. Mr W Sharwood for Assoc Prof Sanjiv Jain, instructed by KJB Law. Mr D Shillington for the Peter McCallum Cancer Centre and Assoc Prof David Ball, instructed by Clayton Utz. Mr J Johnson and Mr H McKay for Assoc Prof Brian McCaughan, instructed by Minter Ellison. Ms K Richardson SC for Dr Lavinia Hallam, instructed by HWL Ebsworth.
File Number:	CD 107 of 2013

CORONER THEAKSTON

Introduction

1. I would like to commence by offering my condolences to Mr Douglas' wife, daughter and extended family. The death of a loved one is difficult at the best of times, let alone when it is accompanied by elements of uncertainty.

2. There is uncertainty about the manner and cause of Mr Robert Douglas' death due to a pathologist erroneously identifying cancer within a biopsy, Mr Douglas subsequently receiving chemo-radiotherapy and Mr Douglas later succumbing to respiratory failure. There are also a number of associated matters of public safety.

Circumstances

3. On 11 December 2012, Mr Douglas died at The Canberra Hospital (TCH). He was 67 years old and married with an adult daughter. He had smoked cigarettes for 40 years, but had quit smoking the year before. He had worked on farms, the new Parliament House and at Fyshwick Markets. He had also spent a considerable period of his working life training and supporting disabled people, for the purpose of their entry into the workforce. I understand it was this latter work Mr Douglas found most rewarding.

4. In February 2012, Mr Douglas attended his general practitioner complaining of a cough, fever and rigors. His GP organised an X-ray and then a CT scan, the latter of which revealed a mass in his left upper lung. His GP referred him to TCH that led to further investigations, including a core biopsy of the mass and a PET scan. He was diagnosed with lung cancer.

5. From May to June Mr Douglas underwent chemo-radiotherapy treatment. During that period another mass developed in his chest muscle. It caused considerable pain and, in early October, finally ruptured through two skin openings.

6. Mr Douglas sought second opinions, including from Assoc Prof Brian McCaughan, a cardiothoracic surgeon in Sydney, and from the Peter MacCallum Cancer Institute (Peter Mac Centre) in Melbourne.

7. In late October he presented to TCH with a respiratory infection. He was treated with a range of medications, but ultimately without success. He was transferred to the intensive care unit. On 11 December 2012, Mr Douglas passed away.

8. His family, after consulting a treating clinician, was offered a partial autopsy of the lungs and chest wall. In the course of that autopsy samples were taken from the area of the mass in his lung and no cancer cells were identified. The autopsy pathologist then reviewed the original core biopsy sample and was unable to identify any cancer cells within that sample. The original pathologist reviewed the original sample and agreed he had made a mistake and there were no cancer cells identifiable within the sample.

9. The matter was reported internally within ACT Health, investigated and, on 23 April 2013, referred to the ACT Chief Coroner.

The coronial function and the structure of these reasons

10. As the death may be partly attributable to an invasive medical procedure, I have exercised the coronial jurisdiction pursuant to s 13(1)(e)(ii) of the *Coroners Act 1997* (ACT), as it was in early 2013, and pursuant to s 13(1)(c), as the Act is now. Section 52 of that Act requires me to make findings about Mr Douglas' identity and the date, location, manner and cause of his death. I may also comment on any matter of public safety that arises in connection with the inquest. While the date and location of Mr Douglas' death is not controversial, the manner and cause of death involves some complexity and uncertainty.

11. Mr Douglas' history included mycobacterial infection. He also suffered from emphysematic lungs. Shortly before his death he was observed to be suffering from a serious respiratory infection, with staphylococcus aureus being detected. Clinicians believed from his history and symptoms that he initially had lung cancer. However, there is no histological evidence to corroborate that. The mass that developed after his cancer treatment was unusual and its diagnosis was uncertain. Senior clinicians in Canberra, Sydney and Melbourne examined Mr Douglas and for the purpose of this inquest I engaged specialist medical practitioners to provide expert evidence. Consequently, there are a number of opinions about Mr Douglas' manner and cause of death.

12. A number of issues need to be discussed before reaching a conclusion about the manner and cause of death. Those issues are:

    (a) did Mr Douglas initially have cancer;

    (b) if so, was the treatment appropriate; and

    (c) what was Mr Douglas' condition following the cancer treatment?

13. A number of matters of public safety have arisen from the evidence. Of those concerns most have already been addressed and rectified by administrative action. Those matters can be listed as follows:

    (a) The initial biopsy:

        (i) how did the false positive for the biopsy occur;

        (ii) why did the ordinary review of the biopsy not occur; and

        (iii) how was the report from the Peter Mac Centre misunderstood?

    (b) The limited records of the April 2012 Lung Multidisciplinary Meeting.

    (c) The autopsy process:

        (i) the scope of the autopsy and the communication between clinicians and the family; and

        (ii) the nature of the supervision during the autopsy.

14. A number of issues have been raised by Mr Douglas' family and I will address those issues where they arise within the above structure.

15. Within this document I have made a number of express findings and recommendations. Those findings and recommendations are consolidated at Annexure A.

Administration

16. When considering the evidence and making findings, I have applied the civil standard of proof being, "on the balance of probabilities", in the way described within *Briginshaw v Briginshaw* [1938] HCA 34 (30 June 1938).

17. Due to the complexity of the issues, I will refer to the evidence in some detail. A large part of that evidence is medical in nature, and includes medical terminology. I will refrain from exhaustively explaining each term and will instead assume the reader either understands the language or has the capacity to make his or her own enquiries. That approach is consistent with this report being a record of my reasons, findings and recommendations, rather than a work of education.

18. I will use the key described at Annexure B when making reference to the evidence.

19. A chronology of significant events is contained at Annexure C.

Non-publication Order

20. On 21 November 2016, I made an order prohibiting the identification of the pathologist Assoc Prof Sanjiv Jain. That order was on the basis that due to the complexity of the medical evidence, there was a real possibility that during the course of the hearing his actions and the resultant consequences could be misunderstood and erroneously reported. That in turn may have adversely and unfairly affected his reputation. With the release of this report that risk has now passed.

21. Section 40 of the *Coroners Act 1997* provides that hearings must be heard in public, unless a coroner is of the opinion that it is desirable in the public interest or the interests of justice to either hold the hearing in private or restrict the publication of evidence.

22. I received submissions on behalf of Assoc Prof Jain that the public interest would not be served by revoking the order because:

    (a) the public interest is not whether there was a misdiagnosis, but how the system allowed it to occur;

    (b) revocation may lead to the loss in confidence of a senior pathologist employed by the Territory; and

    (c) Assoc Prof Jain has demonstrated that he is a competent, respected and trusted pathologist over a long period of time, and revocation of the non-publication order may cause unnecessary alarm to those who hold reports authored by him.

23. The ordinary position is that coronial proceedings should be heard in public. This is for well-known public policy reasons. Simply because the misdiagnosis forms only one part of the narrative, that of itself does not establish a public interest reason sufficient to displace the ordinary position of transparency. Similarly, because the disclosure of the error might lead to some loss in confidence in Assoc Prof Jain and or possibly cause some degree of concern by those who have relied on his opinion in the past, that does not establish an sufficient public interest reason. There are competing public interest arguments that the public would benefit from full disclosure. Finally, I note that a complete reading of this document should provide the reader with a fair understanding

of Assoc Prof Jain's lengthy and peer recognised experience and competence, how and why the misdiagnosis was made, the actual consequences of that misdiagnosis, and the professional response taken by Assoc Prof Jain upon learning of his error.

24. I am not of the opinion that it remains desirable in the public interest or the interests of justice to restrict the publication of Assoc Prof Jain's identity and therefore revoke the non-publication order of 21 November 2016.

Manner and Cause of Death

Did Mr Douglas initially have cancer?

25. This issue requires the consideration of the following sub-issues:

    (a) Mr Douglas' medical history;

    (b) the CT Scan of March 2012;

    (c) was the mass present in February 2012 when an earlier x-ray was taken;

    (d) the biopsy of March 2012;

    (e) did Mr Douglas have an infection on initial presentation to TCH in March 2012;

    (f) the PET scan of April 2012;

    (g) the clinical indicators of cancer; and

    (h) what may have caused the extension of the mass into the chest wall and first rib?

Mr Douglas' medical history

26. In October 1997, Mr Douglas presented at TCH with a lump in his left axillary lymph node. He underwent an ultrasound fine needle biopsy and cultures were taken. No acid fast bacilli were detected, which suggested the absence of tuberculosis. In December 1997, Prof Peter Collignon diagnosed Mr Douglas as most likely having a mycobacterial disease of his lymph nodes [CB 6]. It remained unclear whether it was an atypical mycobacteria or mycobacterium tuberculosis. Mr Douglas was prescribed a full course of treatment [CB 15] that continued for approximately nine months. As best Prof Collignon could recall, Mr Douglas completed the course and complied with the medication regime. Upon review in March 1999, Prof Collignon reported Mr Douglas had been off therapy for six months with no problems. There were no longer abnormal lymph nodes in his left axilla.

27. Prof Collignon gave evidence that it was unlikely that such an infection could have moved to the lung [T 233.21].

28. Mr Douglas later presented to TCH in April 2008 with sweats and chills. In 2010, he presented with atrial fibrillation. It has not been suggested that either event may have impacted upon the events of 2012, although the atrial fibrillation was noted on the medical certificate of cause of death [CB 189]. On 27 July 2011, Mr Douglas underwent a chest X-ray in relation to an umbilical hernia [CB 33], which showed the lungs and pleural spaces were clear other than a left basal linear atelectasis.

29. On 28 September 2011, Mr Douglas presented to the Emergency Department of TCH [CB 38] with shortness of breath, left sided sharp, non-pleuritic pain around the apex of the lung and associated pain radiating to the back of his neck and tingling down his right arm. His chest X-ray [CB 39] showed no obvious lymphadenopathy, and the pleural reflections and lung fields were clear.

30. On 15 February 2012, Mr Douglas' GP referred him for an X-ray upon him presenting with a cough, fever and rigor. The X-ray again showed mild linear atelectasis at the left lower zone laterally but the remaining lungs appeared clear with no air space consolidation [CB 39].

31. A Ziehl Nielsen stain was applied to sputum on 19 March 2012 and no acid fast bacilli were detected in the culture.

The CT scan of March 2012

32. Mr Douglas continued to experience pain and his GP referred him for a CT Scan. On 26 March 2012, a CT scan was conducted that identified a mass present anteriorly and within the left upper lobe of approximately 40mm diameter. There was evidence of extension into the chest wall and destruction of the anterior aspect of the left first rib and probable direct extension into the deep aspect of the pectoralis major muscle. It also showed atelectasis within the posterior basal segment of the left lower lobe and nodular enlargement of the right adrenal gland.

33. There was also evidence of thrombus within the adjacent subclavian vein extending into superior vena cava. Additionally, there were prominent lymph nodes within the superior mediastinum.

34. Assoc Prof McCaughan gave evidence that the CT scan highlighted a large tumour or mass in the left upper lobe invading the chest wall with invasion of the extra pleural fat, destruction of the first rib and direct extension into the deep aspect of pectoralis major [T 427.5]. The report for the CT scan also commented on lymph nodes greater than 25mm that, according to Assoc Prof McCaughan, were abnormally large and a reference to the right adrenal gland that is a common site for metastasis.

Was the mass present in February when an earlier x-ray was taken?

35. If there had been no mass present on 15 February 2012, a mass would have need to have grown between that date and 26 March. Dr Brandon Nguyen, the treating radiation oncologist, when asked whether an infection expands quicker than a cancerous tumour, said some tumours grow very rapidly and will double in size from week to week. He said it was quite possible this was such a tumour [T 284.12].

36. Prof Martin Tattersall, an oncologist retained to provide expert evidence to the inquest, said that it was unlikely that a tumour would develop over such a short period [2T 10.17], but indicated a rapidly growing tumour, such as small cell lung cancer, could grow at the necessary rate. The mass was growing much more quickly than most adenocarcinomas would have done [2T 47.13]. He also explained the apex of the lung is not a common site for small cell lung cancer, but such cancer could grow quickly if it had been there [2T 47.10].

37. Prof Tattersall also suggested that the location of the mass was such that it may not have been visible in a normal X-ray. He described why that may be the case, with

reference to anatomy and the limitations associated with X-rays. He indicated that a special view X-ray or CT scan could be carried out for closer examination. There is no evidence that such imagery was conducted at that earlier time. Prof Tattersall also observed that it was possible that the mass was present, but smaller at that time.

The biopsy of March 2012

38. The core biopsy requested by TCH was ultimately extracted by Dr Thompson at his surgery on 30 March 2012, but returned to TCH for analysis by Assoc Prof Jain. On 3 April Assoc Prof Jain reported the sample had features of primary pulmonary adenocarcinoma (poorly differentiated) infiltrating fibrous tissue (probably desmoplastic tumour stroma) [CB 60].

39. Following Mr Douglas' autopsy this sample was reviewed by a number of pathologists, including Assoc Prof Jain. Notwithstanding the initial diagnosis, all agreed that malignant cells could not be observed within the sample [T 452].

Did Mr Douglas have an infection on initial presentation to TCH?

40. Mr Douglas' General Practitioner, Dr Clifford, referred Mr Douglas to the Emergency Department at TCH. In her covering letter [CB 42] she reported Mr Douglas had been unwell over the previous six weeks with a chesty cough, shortness of breath, fevers, night sweats and left shoulder and arm pain. Dr David Hurwitz, a treating respiratory physician at TCH, said this history was not definitive. It was consistent with tuberculosis, pneumonia or an infection, and was also suggestive of a malignancy [T 36.30].

41. Upon admission, Mr Douglas was observed to be afebrile [CB 45], which is more consistent with the absence of infection. He was commenced on Clexane [CB 50] for the thrombosis.

42. Prof Collignon said it was very unlikely that tuberculosis was involved because if it had there would have been scar tissue in the upper zones of the lung [T 253.18]. He went on to opine that unless Mr Douglas had been re-exposed to tuberculosis between the February X-ray and the March CT scan, and had a new infection, the chances he had tuberculosis anywhere in his body should be very low, and even lower given he had been previously treated for tuberculosis [T 253.38].

43. Dr Hurwitz [T 119] confirmed that if there was an infection in March or April 2012, one would expect to see an elevated white blood count and, depending upon the infection, an elevated neutrophil count. However, both were within their normal limits [T 120.1]. If there were a mycobacterial infection, he would have also expected to see a very different biopsy, including granulomata (the way the cells are collected) and also necrosis [T 58.29].

44. If the mass was an infection, the core biopsy would have shown infection cells. Assoc Prof Jain said that various cells that arise due to infection look different to epithelial cells. He said that in this case he did not see any features in the original biopsy to suggest mycobacterial, or any other, infection [T 183.29]. There was no evidence from the other pathologists who reviewed the core biopsy, that infection cells were observed.

The PET Scan of April 2012

45. Dr Hurwitz requested a PET scan, which was conducted on 11 April 2012 [CB 69]. Dr Hurwitz explained the positive emission tomography scan is a nuclear scan. An isotope is injected into the subject, that isotope is drawn to cells in the body that are rapidly dividing. On the resultant image, sites of both malignancy and infection light up [T 48.24].

46. Dr Hurwitz said the brightness of the illuminated patch on the chest wall on the PET scan tended to suggest it was more likely a malignancy rather than an infection [T 48.29]. Despite cross-examination on the topic, he maintained that view [T 118.6]. Prof Tattersall agreed PET avidity (brightness in this case) was consistent with both infection and malignancy, but typically infection was not described as intensely avid, as was done in the report for this scan [2T 14.30]. If there were recurrent tuberculosis he would have expected higher avidity in the left axillary region. [T 15.4]

47. Assoc Prof McCaughan indicated the PET scan confirmed the CT scan finding of a locally invasive cancer in the left upper lobe invading the muscles of the left wall and rib [T 425.19].

Clinical indicators for cancer

48. The clinicians who treated Mr Douglas believed he had cancer.

49. Dr Hammett and Dr Hurwitz's team [CB 56-59A], Dr Nguyen [CB 74-76] and Dr Sayed Ali, the treating medical oncologist, [CB 77-78] each took or received their own histories from Mr Douglas. Prof Tattersall assessed that the histories taken by each oncologist were "good ones" [T 15.46].

50. Dr Hurwitz indicated the particular concerns that suggested cancer rather than infection were Mr Douglas' smoking history, the weight loss, feeling unwell and what the mass looked like on the CT scan [T 50.33]. He indicated that if the core biopsy had come back negative, he would have requested they have another look [T 50.38].

51. Mr Douglas' calcium level was elevated on admission and again two days later [CB 53 & CB 54]. Dr Hurwitz indicated that this elevation was significant although on its own he could not say what was causing it [T 41.14]. It could occur from an underlying malignancy or something that had invaded the bones. CRP, an inflammation marker, was also elevated [T 41.7 & CB 54].

52. Prof Tattersall said elevated calcium was a well-known feature of cancer and could be directly caused by the cancer or by the tumour having spread to the bone [2T 12.39 & CB 54]. Sarcoidosis (inflammatory disease of the lymph glands in the area) was a possible alternative diagnosis but it was very unlikely in this setting [2T 13.1].

53. Prof Tattersall said he thought Mr Douglas had cancer initially, because the pattern of abnormality in the chest CT scan is not typical of anything other than a cancer growing in the upper part of the lung [2T 44.26]. Infection would not cause the bone changes or the lymphadenopathy seen on the CT scan. He opined, that in addition to cancer, there was also an infection contributing to the symptomatology. In relation to weight loss, Prof Tattersall said that was more suggestive of infection [2T 15.23].

54. Dr Nguyen said that even in the absence of the core biopsy, he would have had the view that Mr Douglas' clinical history and all the imagery were consistent with a diagnosis of lung cancer and he would have still favoured that diagnosis [T 272.10].

55. Assoc Prof David Ball saw Mr Douglas in October 2012. He opined that on balance Mr Douglas had a cancer, that was treated with chemo-radiotherapy therapy, and was later left with necrosis and infection [T 174.31].

What may have caused the extension of the mass into the chest wall and first rib?

56. Dr Hurwitz opined that the invasion into the first rib could be infection or malignancy, but more probably malignancy [T 117.8]. He would have been more concerned with a history of conventional mycobacterial tuberculosis rather than atypical mycobacteria because the atypical variants are generally not as aggressive as mycobacterial tuberculosis [T 38.33].

57. Assoc Prof McCaughan was of the view the invasion was caused by a tumour [T 425.20].

58. Dr John Tharion, a thoracic surgeon, expressed the view that a large mass had eroded the bone and Mr Douglas' calcium levels were high. In infections you see more new bone formation rather than just bone destruction [T 297.41].

59. Assoc Prof Ball said that bone destruction in March 2012 with the left upper lobe lesion is strongly indicative of cancer. Bone destruction would be unusual with tuberculosis [T 159.37].

60. Prof Tattersall said it is more common for a primary tumour to extend to the adjacent structures than an infection. On balance the speed suggested infection but the pattern of spread was more typical of a tumour [2T 12.17].

61. Dr Lavinia Hallam, the TCH pathologist who supervised the autopsy, said that if there was a tumour causing the invasion, one would expect to be able to follow the tumour through the pleura and she would not have expected to easily separate the pleura from the chest wall [T 440.10]. In response, Prof Tattersall said that the existence of two intact surfaces of the pleura did not mean the lung tumour had not invaded the chest wall. It could mean the access between the pleura had healed by the process of treatment [2T 39.13]. Further, there was some evidence the bone had subsequently become sclerotic (see [CB 98]), which is a sign of the treatment having been given in that site [2T 39.43]. In any event Dr Hallam noted in her report that the mass within the lung was adherent to the anterior chest wall adjacent to greenish purulent material on the parietal pleura [CB 197 & 198].

62. No block of the chest wall, for histological purposes, was taken during autopsy so it could not be analysed.

63. Dr Hallam also opined that she would have expected to have found general cellular disorganisation at the tumour site, even if the tissue had been destroyed and repaired. Her observations were that the underlying structure of the lung was still present in the blocks she examined [T 463]. I note that while seven blocks were taken at autopsy from the yellow area within the left upper lung, those blocks are each only very small samples of the area, and the sections taken from each block are even smaller again, approximately only 4 microns thick [T 131]. Accordingly, an examination of seven

samples of the area represents an examination of only a minute fraction of the mass. Further Prof Johan Duflou, a forensic pathologist retained to provide expert evidence to the inquest, explained that it is difficult to visually identify the location of cancer within the lung during an autopsy, particularly if there is consolidation, pneumonia or scarring present [2T 186.17].

64. Dr Ali testified that a range of cancers are found in the lung including adenocarcinoma, squamous cell carcinoma and small cell carcinoma, as well as a range of rarer cancers [T 331-2].

Conclusion

65. The preponderance of evidence weighs strongly in favour of a finding that Mr Douglas initially had cancer in his left upper lung, and the associated mass initially extended into the chest wall, first rib and pectoralis major muscle. That finding is available, notwithstanding the initial biopsy did not ultimately provide histological support for that conclusion, and that the biopsy process is considered the "gold standard" for lung cancer diagnosis. The absence of the positive biopsy simply means that the limited sample examined from the limited specimen taken from the mass, did not yield identifiable cancer cells.

66. I also note that had the pathologist's report of the initial biopsy returned a negative result for cancer, the treating clinicians uniformly indicated that they would have requested the biopsy be repeated.

67. Based on all the information, the clinicians were confident that Mr Douglas had cancer. While there were a number of symptoms consistent with infection, there were a significant number of indicators of cancer. They included:

    (a) Mr Douglas' long smoking history;

    (b) the absence of a fever upon admission to TCH in March 2012;

    (c) the absence of scar tissue within the lung early on;

    (d) the fact that Mr Douglas had been previously treated for tuberculosis;

    (e) the absence of an elevated white blood cell or neutrophil count;

    (f) the elevated calcium levels;

    (g) the absence within the biopsy of any features of infection;

    (h) the intense avidity of the PET scan;

    (i) the bone destruction without subsequent new bone formation;

    (j) the nature of the spread of the mass; and

    (k) the lymph node involvement.

68. There is, however, inadequate evidence to determine what type of cancer was involved.

69. Finding No. 1: In March 2012, Mr Douglas was suffering from lung cancer.

If Mr Douglas initially had cancer, was the treatment appropriate?

70. On 5 April 2012, Dr Tharion reviewed Mr Douglas to determine his suitability for surgery. At that time no PET scan was available so Dr Tharion planned to await that scan [CB 61]. Mr Douglas did not see Dr Tharion again. Instead, Mr Douglas arranged to see Assoc Prof McCaughan in Sydney.

71. On 13 April Assoc Prof McCaughan reviewed Mr Douglas. He had available the questionnaire completed by Mr Douglas [IB A.100A], the recent CT and PET scans and the core biopsy report. At the hearing he conceded he may not have conducted a physical examination of Mr Douglas. He said any physical examination was redundant once he had a PET scan. He also had a core biopsy result and a CT scan that in combination clearly identified and confirmed Mr Douglas as not suitable for surgery due to the multilevel involvement in various lymph nodes [T 425.31]. He indicated there was no benefit in operating if you cannot remove all the cancer and there are disadvantages for a patient who undergoes surgery, which I understood to mean surgery involves significant discomfort and a range risks [T 425.6].

72. On 16 April Dr Hurwitz presented Mr Douglas' case at the TCH Lung Multidisciplinary Meeting [T 60.25]. The sign-in sheet for that meeting records Dr Nguyen and Dr Tharion, amongst others, also being present at the meeting [Exh O]. The focus of the meeting was to discuss management of the disease [T 51.6] and the best therapeutic approach [T 58.14]. It appears that as a result of the meeting, Dr Tharion signed a document, referring Mr Douglas to medical oncology [CB 73] & [T 292]. At the time of the hearing none of the attendees had a clear recollection of the meeting.

73. It is clear that when the clinicians were considering how to treat Mr Douglas they relied upon the diagnosis of adenocarcinoma by Assoc Prof Jain. There was no evidence that there was any basis for the clinicians to question the reliability of that diagnosis, and to therefore consider repeating the test or treating Mr Douglas for an another disease.

74. Mr Douglas was treated with a six week course of chemotherapy and radiation therapy.

75. A submission was made suggesting that Dr Nguyen and Dr Ali, being junior specialists, lacked adequate supervision by TCH and the multidisciplinary meeting failed to adequately consider Mr Douglas' case. While both Dr Nguyen and Dr Ali were new specialists, they were still formally accredited specialists, having studied, trained and practiced medicine for a significant period of time before accreditation. There was no evidence to suggest that they approached their roles in either an inadequate or inappropriate way. Further, the same was not put to them or any other witness. Similarly, there was no evidence to suggest that the members of the multidisciplinary meeting conducted themselves in an inadequate or inappropriate way, and again the same was not put to any witness. I do note that the meeting had only limited time to consider each individual case, and there was only very limited records in relation to who attended and what was discussed and concluded during the meeting. I will return to the issue of records when I deal with matters of public safety.

76. Dr Ali said that if the cancer had been squamous cell rather than adenocarcinoma, as assessed by the biopsy report, the only difference would have been be to change one of the drugs used. However, that difference would not have made a significant

difference. Had the cancer been a small cell cancer, treatment would have been almost similar. However, for other cancers it would be different [T 332.22].

Conclusion

77. Prof Tattersall assessed that the prescribed chemo-radiation therapy was appropriate for the diagnosis of lung cancer. There was no evidence suggesting otherwise.

78. Finding No. 2: The treatment for Mr Douglas' lung cancer was appropriate.

What was Mr Douglas' subsequent condition following cancer treatment?

79. This issue requires the consideration of the following sub issues:

    (a) the observations made at the time;

    (b) the further tests conducted;

    (c) the possibility of Actinomycosis;

    (d) the treatment administered; and

    (e) the observations at autopsy.

Observations

80. On 14 May 2012, the clinical notes refer to a "chest wall swelling" [CB 91].

81. On 23 July, approximately four weeks after treatment had finished, Mr Douglas was observed to have swelling and tenderness on his left upper chest [CB 92]. Dr Nguyen reported to Dr Clifford those symptoms could be due to radiotherapy, but the progression of the disease could not be ruled out [CB 97].

82. By 26 September, the swelling had increased in size and was very tender which was thought to be either tumour progression or possibly an uncommon differential diagnosis of radiation related necrosis [CB 111]. By 3 October the swelling had broken through the skin [CB 113] and by 11 October it had discharged through two sites resulting in a considerable reduction in pain [CB 119]. Those sites are depicted in a photograph taken by Mr Douglas [Exh L].

83. Assoc Prof Ball, upon review of Mr Douglas [CB 119], said his overall initial impression was of pectoral muscle necrosis associated with chemo-radiation treatment. He thought there may still be residual disease in the chest, but not in the pectoral muscle.

84. On 12 October, Dr Ali opined in his report to Dr Clifford that the mass was a progression of the tumour and the discharge on or about 7 October was a fungating tumour [CB 112].

85. On 16 October, Dr Rao, of the Peter Mac Centre, noted that the swelling could be a progression of tumour or a complication of radiation therapy or infection. Mr Douglas's case was reviewed at a multidisciplinary meeting at the Peter Mac Centre. The majority position was that the swelling was probably recurrent resistant disease but possibly an element of infection or necrosis [CB 128]. Assoc Prof Ball believed that it was radiation related but agreed in evidence that his was the minority view [T 165.10]. Prof Tattersall agreed with Assoc Prof Ball and opined in evidence that it was more likely to be infection than cancer progression [2T 22.10].

86. Prof Tattersall noted, without having personally examine Mr Douglas, that the discharge from the sinuses was serous (being thin and yellow) rather than pus (being thick and white) and then opined that the sinuses could be the result of either tumour or infection, and it was certainly uncontrolled [T 19.15]. At [2T 19.27] he said the nature of the discharge indicated interstitial fluid coming out. If it were fungating, one would see tumour at the base (or circumference) of the sinus.

87. Dr Hurwitz said that it would depend upon what it looked like to determine any clinical diagnosis. The differential diagnoses were infection, persistence or spread of the disease or a complication from the radiation therapy, plus or minus the chemotherapy. He did not see the mass or discharge to be able to comment any further [T 63].

88. Prof Collignon indicated the fungation would constitute very abnormal tissue but, could not say whether it was a fungated tumour or infection.

Further tests

89. Dr Nguyen gave evidence that ordinarily once radiotherapy treatment is completed patients are reviewed within two weeks to make sure they are recovering from the side effects of the treatment, including any swelling. An inflammatory response is common but it typically improves and resolves over six to eight weeks after treatment. Tumour progression is uncommon [T 275.27].

90. Dr Ali said that he ordinarily waits four weeks following chemotherapy before he re-assesses a patient's condition. However, when radiotherapy has also been administered, the effect of that treatment can last for a longer time and the peak effect may be four to six weeks following completion. As a consequence, clinicians cannot be sure when is the right time to request a CT scan and whether what is seen on a CT scan is the effect of treatment, cancer progression or resistance to treatment [T 333.2]. Accordingly, Dr Ali does not normally request CT scans at four weeks after treatment. However, in Mr Douglas' case a CT scan was requested at such time because Mr Douglas was unwell, losing weight and was experiencing considerable pain. Mr Douglas also had a bulge growing on his upper left chest [T 333.8].

91. On 24 July 2012, a CT scan was conducted [CB 98]. At the hearing, Dr Ali identified and marked the swelling on the CT slides [Exh K].

92. On 12 October, while Mr Douglas was being reviewed at the Peter Mac Centre, a further PET scan was arranged [CB 124]. The scan showed the pectoral muscle mass had almost completely resolved but significant FGD uptake (or avidity) at the primary site [CB 120].

93. Assoc Prof Ball indicated the areas circled in orange [Exh G] indicate increased glucose uptake and are most likely inflammatory changes from the radiation. He agreed they could be infection but from the distribution and involvement of the pleura suggested change from radiation [T 162.2].

94. On 22 October, a swab was taken from the discharge site of the chest lump [CB 130]. It showed "+++" gram positive cocci and heavy growth of staphylococcus aureus. Prof Collignon gave evidence that staphylococcus aureus is a "bug" that is commonly on the skin that can be aggressive but usually needs an opportunity to invade the body, such as a skin lesion [CB 234.23]. Prof Collignon opined the bacteria were not just on Mr Douglas' skin, but also in his chest cavity [T 243.2].

95. On 30 October, a further swab of the same site [CB 144] showed that polymorphs (white cells that are the first cells present to fight infection) were present in low numbers. Prof Collignon explained their existence was suggestive of inflammation and the most likely cause was infection [T 236.18]. The gram positive had reduced which suggested there were less of the gram positive bacteria present. This could have been due to treatment or poor sampling [T 236.30].

96. During the hearing Dr Ali, when comparing the CT scan of 28 November, which was taken after the serous discharge, with a CT scan, which was taken on 26 September before that discharge [Exh K] (originally MFI 8 & 9), noted that while the purulent material in the pectoral muscle had reduced the solid mass in the lung remained. He went on to opine that at that time the remaining solid mass within the lung was a separate complication due to a separate process [T 312.40].

97. Dr Ali described changes in the features of Mr Douglas's lungs between September and November 2012, by reference to lung arterial windows [T 315-319 & MFI 10, 11, 12 which became part of Exhibit K]. In particular he noted a net-like structure on the 26 September CT scan, which he indicated were changes likely caused by radiation therapy [T 316.8]. He also noted on the right hand side of the left lung some ground glass opacity suggestive of infection [T 316.11].

98. On the 8 November CT scan Dr Ali noted much more net-like structure and ground glass opacity in both lungs. The shadows indicated inflammation in the interstitial wall. The ground glass opacities indicate some other abnormality, either an atypical infection or some other process. [T 317.32]

99. On 9 November, the chest X-ray showed consolidative change throughout the right lung and within the mid to upper zones of the left lung, with interstitial thickening within the aerated left mid to lower zones. The X-ray report offered the differential diagnoses of overwhelming infection, radiation pneumonitis or drug reaction [CB 186].

100. By 28 November, both lung fields showed extensive ground-glass opacity and some reticular ("net-like") shadows [CB 147] and [T 318], indicative of extensive inflammation and interstitial swelling within the lungs [T 317.35].

Actinomycosis

101. In evidence, Prof Tattersall opined that actinomycosis could have played a part in the chest wall mass [2T 20.9]. He acknowledged he had discussed Mr Douglas' case with his wife, a respiratory physician [2T 23.13]. Actinomycosis is gram positive and lives in an anaerobic environment. There was necrotic tissue in the pectoral muscle and that was a possible location for an anaerobic infection. There was a gram positive infection in the chest swelling as at 22 October [CB 130]. The biopsy on 19 November [CB 171] which was to "target the periphery of the tumour" [CB 168] may not have sampled the necrotic area where any actinomycosis was likely to be present.

102. The possibility of bacterial actinomycosis was raised by clinicians on 12 November [CB 168]. In evidence, Prof Collignon did not consider it a high probability and expected it would have been treated by the penicillin. Even if it had been there, Mr Douglas was on treatment for it [T 244.7]. However Prof Collignon at [T 236.39] and Prof Tattersall at [2T 21.36] accepted that being an anaerobic necrotic environment an antibiotic would be less effective, if getting to that area at all.

103. Prof Duflou gave evidence [2T 178] that to look for actinomycosis he would look for nodular lesions and sulphur granules, neither of which were described in the autopsy report. The autopsy swabs came back with no bacterial growth [CB 200]. Further, there was no evidence of an actinomycotic abscess. No organisms, including actinomyces were seen. A specific methenamine silver stain for actinomyces was not applied but he did not think it was warranted at the time, only in retrospect [T 179.15].

104. That test was done on a sample taken from the mass in the left lobe during autopsy, which yielded a negative result [CB 199]. Ultimately it was Prof Duflou's view that actinomycosis at time of death was unlikely.

Treatment

105. On 24 October 2012, Mr Douglas was re-admitted to TCH for the purpose of receiving further chemotherapy. Upon admission he was assessed to have a respiratory infection and commenced on antibiotics [CB 140]. Following the earlier 22 October swab Prof Collignon opined that Mr Douglas had a "community type" (not multi-resistant hospital type) infection that was sensitive to penicillin. It was not one that would normally be picked up in a hospital [T 235.5]. Mr Douglas was treated with Flucloxacillin, which is the standard drug to treat such infections and one of the more effective drugs. Prof Collignon explained that treatment is normally effective unless there is something really wrong with the immune system or if it is in an abscess or necrotic tissue where the drugs cannot disperse [T 236.35]. He explained that antibiotics are only effective if they can be dispersed to where the bacteria are. Flucloxacillin usually controls an infection, but will not cure it, if there is significant dead tissue [T 236.43].

106. By early November, Mr Douglas had not been responding well to antibiotic treatment and was receiving Flucloxacillin and Ceftriaxone intravenously. On 5 November, a sputum sample was taken and three days later a report indicated a heavy growth of pseudomonas [CB 154]. This is another bacteria. It is very antibiotic resistant, but covered by Tazocin [T 239.11], a broad spectrum agent, described by Prof Collignon as an antibiotic with a component designed to protect it from being broken down [T 238.21]. Tazocin is also effective for treating staphylococcus aureus, although Flucloxacillin is more active against it. Prof Collignon explained that as the sample was sputum and not from a needle biopsy, it was uncertain if the pseudomonas was sourced from within the lung. He added that a needle biopsy is infrequently conducted because of the potential for complications such as a collapsed lung [T 239.38].

107. On 8 November, Mr Douglas had a temperature spike, despite receiving four doses of Tazocin [CB 154]. Prof Collignon reviewed Mr Douglas on 8 November. At that time Mr Douglas had a raised CRP, indicative of inflammation. Prof Collignon assessed that Mr Douglas was on the correct antibiotics [T 240.40], but if his condition did not improve they would try Benpen and Bactrim which are used together to treat staphylococcus aureus with the aim of better penetration into tissue [T 241.4]. Prof Collignon gave evidence that at this time, they did not know for certain what was causing the fever. The clinical notes for 9 November [CB 164] suggest there was a concern there was an infection in the middle of the mass, which the antibiotics could not reach [T 241.39].

108. Prof Collignon had seen Mr Douglas on three consecutive days from 8 – 10 November, and that was consistent with Mr Douglas being very ill [T 243.33].

109. On 26 November, Mr Douglas was reviewed by Prof Collignon [CB 175]. He was now on broad spectrum Meropenem which according to Prof Collignon is a "top of the line, run out of options type antibiotic" [T 245.9] and Bactrim. The X-ray also showed pneumonia in the left lower lobe. Mr Douglas had shortness of breath, tachycardia and low oxygen saturation levels in his blood that suggested his lungs were not functioning as well as they had functioned previously [T 245.14]. The clinical notes query whether there may be a pulmonary embolus or candida. Mr Douglas had a fever of 39°C that Prof Collignon described as "not a good sign". He observed that something was not under control [T 245].

110. Dr Hurwitz was asked about Mr Douglas' condition at 28 November. He said there was a risk of a mycobacterial infection but he said it was less likely as it should have been seen in the sputum [T 72.5].

111. In relation to a ward round on 8 December, Prof Collignon recalled that at that time they still did not know what was happening. There was a query whether the tumour was expanding or there was an inflammatory reaction to radiation pneumonitis [T 250.16]. Mr Douglas had self-medicated while he was at home with a corticosteroid with positive effect, which works opposite to an antibiotic and makes infection harder to fight [T 250.33]. Prof Collignon thought this suggested something else may be going on.

112. About this time Mr Douglas was also put on Clarithromyacin to cover atypical organisms, including tuberculosis to some extent [T 250.42].

Autopsy

113. Following Mr Douglas' death, a hospital, as opposed to coronial or forensic, autopsy was conducted. Mr Douglas' lungs weighed 1340g and 1060g respectively. Prof Duflou describes those weights as very abnormal. Anything above 400g for an average male is abnormal [2T 184.31].

114. Dr Hallam's autopsy report [CB 199] recorded her observations of greenish purulent material 40mm across on the parietal pleura. The lungs were emphysematous with an indistinct but large, firm and yellow area (mass) in the left upper lobe measuring 110mm x 100mm x 75 mm, with a central 35mm area of necrosis, and with the larger area adherent to the anterior chest wall. No other tumour masses were observed. The other lobes, on both the left and right lungs, showed multiple cavitating lesions consistent with pneumonic changes.

115. Dr Hallam's report also records her microscopic observations. The area in the left upper lobe showed no organisms, including actinomycetes or acid fast bacilli, but did show organising pneumonia. The other lobes showed acute pneumonia with hyaline membranes and alveolar exudates with fibrin and neutrophils.

116. Dr Hallam's evidence was that the emphysematous state of the lungs related to the abnormally large size of the air spaces within the lungs and was a feature of lung disease due to smoking. The condition can result in laboured breathing [T 384].

117. While no sample for histological examination was taken from the chest wall, a sample for microbiology was taken from that area. It returned a light growth of staphylococcus aureus [CB 200]. Dr Hallam opined that, notwithstanding the limitations of such samples taken during an autopsy, she was confident that the result suggested there

was an infection [T 391]. Prof Collignon also noted that result but explained they are difficult to interpret because bacteria grow up when you die because the white blood cells are not functioning. [T 246.27].

118. Dr Hallam observed that organising pneumonia can result from bacterial infection of the lungs [T 395].

Conclusion

119. There is a degree of uncertainty about the precise pathology of Mr Douglas' condition following his chemo-radiotherapy treatment. A number of opinions were expressed during his treatment, but these were made without the benefit of the information obtained during the autopsy. The autopsy found no evidence of cancer progress following the initial treatment. There was evidence of necrosis and recent inflammation within the lungs. There was evidence of infection within the sputum and the chest wall. Despite targeted and repeated antibiotic treatment, Mr Douglas had suffered a prolonged fever.

120. At autopsy, Mr Douglas' lungs were observed to be emphysematic, and that was consistent with his long and significant smoking history [T 384.20]. Significant and progressive changes were observed throughout the lobes. This is suggestive that such changes were not due solely to the targeted radiotherapy, which was applied at an area in the left upper lung and chest wall.

121. It is clear from the above, that for the month or so before Mr Douglas' death he experienced significant respiratory infection. The test results suggest possibly more than one infection. The nature of the infection was actively considered and a range of tests and treatments were contemplated and administered. There was no evidence that the approach taken by his treating clinicians was wanting.

122. The medical certificate of cause of death [CB 189] recorded cause of death as radiation pneumonitis with the antecedent cause of non-small cell lung cancer.

123. The final post mortem report [CB 202] and Dr Hallam's evidence [T 463] was that Mr Douglas died from organising pneumonia, overlying empyema and chest wall involvement and extensive acute pneumonia with hyaline membranes.

124. Dr Nguyen's view at the time of Mr Douglas' death, was that the cause of death was likely to have been acute pneumonia with a possible involvement of interstitial pneumonitis by which he meant an inflammatory condition in the lung of which radiation is one possible cause [T 278.41].

125. Prof Tattersall opined that Mr Douglas died of uncontrolled infection [2T 23.29]. Because the lung pathology was on both sides of the lung, he said that it is untenable that it was radiation related. He opined that organising pneumonia was not the cause but may have been part of the picture when he died [2T 24.1].

126. Prof Duflou's view [IB B.478] was that Mr Douglas died of interstitial lung disease. When asked about this he said it is not cancerous. He included within the interstitial lung diseases: emphysema, interstitial pneumonias, many of which have unknown causes, radiation pneumonitis and organising pneumonia [2T 180.18]. He said there was certainly a degree of radiation pneumonitis [2T 180.39], but exactly what mechanism and exactly what cause he could not say [2T 181.4]. In relation to Prof

Tattersall's opinion that the cause of death was uncontrolled infection, he said the proximate cause of death could be pneumonia, but Mr Douglas would not have died of pneumonia if he did not have lung disease [2T 181.10].

Findings – Manner and cause of death

127. In light of the above evidence and the range of views that the evidence supports, it is difficult to make a precise finding about the nature and cause of death. However, a number of consistent factors permeate the evidence and opinions, and they allow the following findings to be made.

128. Finding No. 3: Following Mr Douglas' re-admission to The Canberra Hospital on 24 October 2012, Mr Douglas received systematic and vigorous treatment for a respiratory infection.

129. Finding No. 4: On 11 December 2012, Mr Douglas died at The Canberra Hospital.

130. Finding No. 5: Mr Douglas' death was directly caused by respiratory failure, with antecedent causes including:

    (a) chronic emphysematic lung disease;

    (b) inflammatory lung damage, including possibly from radiation therapy; and

    (c) acute respiratory infection of unknown type and origin.

Matters of Public Safety

131. As described above, a number of matters of public safety have arisen from the evidence. Those matters will be addressed in the order and structure described at paragraph 13.

How did the false positive for the initial biopsy occur?

132. Assoc Prof Jain is a qualified pathologist and has been working as a staff specialist at TCH for a number of decades. He is currently employed as the Director of ACT Pathology. He routinely assesses biopsies for the presence and type of cancer [T 130].

133. As described above, on 3 April 2012 Assoc Prof Jain assessed and reported on the core biopsy taken in March 2012 by Dr Thompson from Mr Douglas' left upper lobe. In his report [CB 60] he opined, "the fibrous tissue core contains malignant cells as single cells or small groups of cells without formation of glands" and diagnosed primary pulmonary carcinoma.

134. Following Mr Douglas' death other pathologists in the department reviewed the slides for that same biopsy and assessed that it did not evidence malignancy. Those pathologists included Dr Genevieve Bennett, Dr Jane Dahlstrom, Dr Michael Brown, Dr Hallam and Dr Huw Llewellyn [T 465.33]. Assoc Prof Jain also subsequently viewed the slides and conceded the same diagnosis. He explained during the hearing that when he reviewed the slides, he saw the same things in the same cells but they were put in a different context, presumably due to the subsequent observations and testing of Mr Douglas as well as the view of Dr Hallam, and he interpreted them as reactive to inflammation, rather than malignant [T 136]. At my request, Prof Duflou subsequently

attempted to review the slide of the original biopsy. However, the material within the slide had degraded, which prevented any useful histological assessment.

135. To Assoc Prof Jain's credit, he conceded his mistake when the possibility was brought to his attention and after he had the opportunity to review the slide and reconsider his diagnosis. He also promptly filed a Riskman Report bringing the matter to the attention of his superiors at TCH [IB A.146].

136. While neither Dr Llewellyn and Dr Bennett had a positive recollection of reviewing the slide, each accepted they had recorded their assessments in a silent field on the electronic pathological database [Exh R & S]. Dr Llewellyn made an assessment of fibrosis rather than malignancy, and included the comment, "The distinction is subtle and I have the benefit of retrospect." Dr Bennett, when assessing the biopsy as benign, included the comment, "I do not think the entrapped epithelial cells present are cytologically very atypical, and I think the pattern of the keratin stains is demonstrating underlying residual alveolar spaces, distorted and focally obliterated by the inflammatory process. I can find no convincing evidence of malignancy in these sections."

137. The evidence left open the possibility that Dr Maya Cherian, another TCH Pathologist, may have reviewed the slide for the purpose of the Lung Multidisciplinary Meeting of April 2012, and did not form a different view to that of Assoc Prof Jain's initial assessment. She ordinarily attended the meetings [T 184.29] and was recorded as doing so [Exh O]. Her usual practice was to review the slides when attending these meetings and if her assessment differed from the original diagnosis to make a record in the database [2T 104.28]. Dr Cherian did not have a memory of attending the meeting [Exh Q] and there is no record in the database of a different opinion by Dr Cherian.

138. It was clear from the evidence that while an assessment by an experienced pathologist of a core biopsy was considered by treating clinicians as the gold standard for the diagnosis of lung cancer, the actual assessment by the pathologist involved the exercise of judgment and reliance on clinical information, rather than the application of any blind, binary or purely objective test. An assessment was therefore open to error and possibly differing opinions.

139. Finding No. 6: On 3 April 2012, Assoc Prof Sanjiv Jain, when reporting on the core biopsy sample taken from Mr Douglas on 30 March 2012, incorrectly diagnosed the sample as malignant and as evidencing primary pulmonary adenocarcinoma. The sample involved features making diagnosis difficult.

140. Finding No. 7: Treating clinicians relied upon Assoc Prof Jain's diagnosis, as the diagnosis informed treating clinicians about what treatment would be appropriate. It also deterred clinicians from repeating the biopsy that may have in turn provided a reliable diagnosis.

Why did the ordinary review of the biopsy not occur?

141. In 2012 and since approximately 1990, ACT Pathology maintained a practice where a second pathologist reviewed core biopsies. That review would occur proximate to a Tuesday quality assurance meeting, and may occur after the pathology report was released. If the diagnosis was changed the pathologist would issue an amended report and telephone the treating clinician [T 138].

142. That review arrangement was self-imposed by the department, and Assoc Prof Jain and Prof Collignon were not aware of a similar practice being followed in 2012 anywhere else within Australia [T 221 & T 265].

143. Assoc Prof Jain's pathology report was released on Tuesday 3 April 2012. The following weekend was Easter and there was no Tuesday meeting on 10 April. As a consequence, a second pathologist did not review Mr Douglas' core biopsy [T 138.11].

144. On 8 January 2013, almost immediately following the discovery of the diagnostic error and the failure to review the original diagnosis, Assoc Prof Jain implemented a change to the automatic review process within ACT Pathology. Rather than having the review process initiated by secretarial staff and conducted at a quality assurance meeting, all tumour diagnoses related to core biopsies would be correlated by a senior registrar and reviewed by a second pathologist rostered to do so [Exh V & T 138.24].

145. Further, following a Clinical Review Committee's consideration of the misdiagnosis, a further adjustment was made to the review process. The resultant process now involves a mandatory review of all lung core biopsies by a second pathologist. A protocol is in place that describes how and when that second review is to take place, and how amended reports are to be prepared and issued. Additionally there are quarterly audits to ensure compliance with this arrangement [IB B.573 & B.581]. Assoc Prof Jain and Dr Cherian both explained that in addition to those formal arrangements, the department does not issue a report on a new malignancy without another pathologist reviewing the biopsy and any disagreement is recorded in the silent field [T 138.30 & 2T 110.27].

146. Prof Collignon explained that the number of samples that have gone through the new quality assurance process is close to 2,000, and there has not yet been a discrepancy between the opinions of the first and second pathologists. He questioned the cost benefit of continuing to have two pathologists review each core biopsy [T 256.26].

147. Finding No. 8: In April 2012, ACT Pathology had in place a process of reviewing lung core biopsies. That process was linked to a weekly quality assurance meeting. However, due to the Easter long weekend the quality assurance meeting of 10 April 2012 did not take place and the core biopsy taken from Mr Douglas' lung on 30 March 2012 was not reviewed by a second pathologist.

148. Finding No. 9: In 2013, ACT Pathology established a quality assurance process, decoupled from any meeting, with a protocol clearly setting out how biopsies, including all lung core biopsies, would be reviewed by a second pathologist. That process includes quarterly audits to ensure compliance and adequately addresses the systemic failings associated with Mr Douglas' biopsy.

149. Recommendation No. 1: The Canberra Hospital continue to periodically review its quality assurance processes for core biopsies, to ensure that such processes appropriately balance the need to minimise the risk of errors with the costs of associated control measures. Such processes should involve an element to check and ensure compliance.

How was the report from the Peter Mac Centre misunderstood?

150. In April 2012 and following the diagnosis of adenocarcinoma, a tissue block from Mr Douglas' lung core biopsy was sent to the Peter Mac Centre for Epidermal Growth

Factor Receptor Mutation Analysis (EGFR). EGFR is a test associated with a second line treatment, behind those of chemotherapy and radiotherapy. The test looks for a particular mutation of a protein on the surface of the malignant cell, the presence of which indicates the appropriateness of a particular pharmacological treatment [T 325].

151. The Peter Mac Centre conducted the testing and provided a report [CB 84] to TCH indicating that no mutation was detected. The report also contained the comment:

> *Biopsies contain a variable proportion of tumour cells to normal cells and there is likely to be a variable proportion of clonally selected tumour cells within the tumour itself. Each sample is subject to pathology review and macro-dissection to increase assay sensitivity. Nevertheless, if tumour cells represent less than 20% of the cells analysed the sensitivity of the assay may be reduced.*

152. The report also contained the following statements located prominently towards the top of the document, "Histological Typing: Adenocarcinoma" and "Proportion tumour cells per marked area: 60%".

153. Prof Tattersall, Assoc Prof Jain, Dr Hurwitz and Dr Ali each expressed a belief that the EGFR process may involve an independent assessment of whether the tissue was malignant [2T 46.17], [T 139-140], [T 99.37] and [T 328.39]. However that was not the case.

154. Dr Stephen Fox, Director of Pathology at the Peter Mac Centre, and Dr Simone Birch, a former registrar at the same centre, both indicated that the centre presumed the original diagnosis of malignancy was correct and it did not conduct its own histological assessment for malignancy. Dr Birch, who examined the slides produced from the tissue block, indicated her task was merely to determine whether there was sufficient and viable tissue on the slides and to assess the percentage of cellularity. Cellularity relates to the counting or distinguishing of malignant cells from non-malignant cells [2T 138-139]. Dr Fox explained that it is assumed that any unusual cells are the previously diagnosed malignant cells [Exh T].

155. It was submitted that the failing by some staff at TCH to appreciate that the EGFR process was not a second histological diagnosis represented a gross failing. It was suggested that had the misunderstanding not occurred the treatment team may have considered other diagnoses. However, even if every clinician fully understood the limitations of the EGFR process, the results from that process would not have provided them with any reason to doubt the diagnosis of adenocarcinoma from the original biopsy.

156. Since the above misunderstanding came to light, the Peter Mac Centre has changed the text on its reports [Exh Y]. The relevant passage now reads:

> *The sample was reviewed by a pathologist and was considered to contain x% tumour cells within the area selected for analysis. (Note this is not a formal pathology review and is based solely on an H&E of the tissue provided and not on ancillary clinical or pathology information that may be available elsewhere).*

157. While there can be no criticism of the language used within this new passage, as it is consistent with the processes conducted at the Peter Mac Centre, there remains the risk that the passage may still be misunderstood. The passage may be read to mean that some pathological assessment occurred and tumour cells were observed. It is

also silent about the degree to which the process depended upon the original diagnosis.

158. Finding No. 10: A number of clinicians erroneously assumed the Epidermal Growth Factor Receptor Mutation Analysis conducted by the Peter MacCallum Cancer Institute critically reassessed the original diagnosis of adenocarcinoma.

159. Recommendation No. 2: The Peter MacCallum Cancer Institute consider reviewing the words used within its reports for the Epidermal Growth Factor Receptor Mutation Analysis to ensure that treating clinicians are disabused of any erroneous assumption that the test either reassesses the original diagnosis or positively identifies tumour cells.

The limited records of the April 2012 Lung Multidisciplinary Meeting

160. The Lung Multidisciplinary Meeting was, and appears to remain, an important step for clinicians when deciding how to treat a new malignancy within the lung. There was a scarcity of evidence about whom precisely attended the meeting, what was discussed and what was agreed or decided as an outcome at that meeting. For example, it remains uncertain if a particular pathologist attended the meeting and shared any observations about the content of the slides from the biopsy. Further, the sign-in sheet [Exh O] was not initially located and only produced late in the hearing.

161. It is not surprising that treating clinicians, without notes, would have difficulty recalling a discussion about an individual patient during a particular meeting. Best practice would suggest clear records should be made and kept for such important meetings. Those records would ideally include who participated in the discussion, what information was before the meeting and what was the outcome of the discussion. The final aspect would be of some significance, not simply to aid any reconstruction of the meeting, but to ensure that any outcome of the meeting was not subsequently misunderstood or distorted.

162. Finding No. 11: There were limited records available from the Lung Multidisciplinary Meeting of 16 April 2012.

163. Recommendation No. 3: The Canberra Hospital consider introducing a protocol that require appropriate records be made of Lung Multidisciplinary Meetings, and that such records be appropriately stored.

The scope of the autopsy and communication between clinicians and the family

164. Dr Hallam explained that TCH only performs between five and 10 adult autopsies a year [T 412.23].

165. Assoc Prof Jain explained there was no formal process for triggering a hospital autopsy, and the same may be initiated by the treating clinician or family of the patient. Either way consent is required from the next of kin. There were consent forms available and a number of pamphlets for a family's benefit. Should a pathologist consider it may be appropriate to extend an autopsy beyond what was consented to, there is no protocol about how to obtain that additional consent [T 176-177].

166. Mrs Douglas consented to a partial autopsy of the lungs and chest wall [CB 192].

167. In 2012, Registered Nurse Judith Rafferty was employed at TCH as a Nurse Care Coordinator with the role of providing support to patients living with cancer, and their families. Sister Rafferty indicated that following Mr Douglas' death she received a request from Mrs Kerry Douglas, Mr Douglas' wife, for an autopsy to be conducted [IB B.636]. She denied offering any advice about whether such a procedure should be conducted, or whether it should be full or partial in nature. Sister Rafferty referred to an email [IB B.644] from Ms Niki Reilly, Mr Douglas' daughter, which suggested Sister Rafferty advised the family that only a partial autopsy was necessary. Sister Rafferty disputed giving such advice. That discrepancy was not addressed further during the hearing.

168. Dr Ali did not recall speaking to Mrs Douglas about the autopsy.

169. Dr Nguyen recalled speaking to Mrs Douglas in relation to whether an autopsy should be conducted [T 278]. He thought the procedure had utility due to the unusual features of the case. Because it was clear that Mr Douglas died from respiratory failure, he was satisfied that only a partial autopsy was required, limited to the lungs, in order to determine cause of death [T 279].

170. Prof Duflou agreed that in retrospect it would have also been valuable to look at the right adrenal gland, but access to that organ was tricky and would fall outside the scope of the limited autopsy [2T 199.40]. He assessed that, in all the circumstances, the limited autopsy was appropriate [2T 200.24].

171. Finding No. 12: Following Mr Douglas' death, The Canberra Hospital conducted an autopsy, limited to the lungs and chest wall, as consented to by Mr Douglas' wife, Mrs Kerry Douglas. The limited nature of that autopsy was appropriate given what was known at the time.

172. The autopsy was conducted by Dr Ayesha Ajmal, a registrar, and was supervised by Dr Hallam. It was limited to the lungs and chest wall. Prof Duflou said the ribs were part of the chest wall [2T 199.20], although he said it was uncommon to take samples from the ribs.

173. During the autopsy, Dr Hallam did not consider it necessary to extend the scope of the autopsy [T 458.29], and accordingly made no request for additional consent. Dr Hallam's concerns about the original diagnosis arose some time later when she examined the slides from the block samples.

174. The consent form [CB 92] has a front page that addresses the issue of consent, and a second page that provides an opportunity for the treating clinician to provide a clinical history and test results, and direct the pathologist's attention to any specific matters for examination. It also provides a point of contact for the benefit of the pathologist. In Mr Douglas' case that second page was not completed. Dr Hallam noted that it was common for the second page not to be completed. However, the hospital notes were provided and there were verbal conversations between Dr Hallam and the clinicians before the autopsy commenced [IB A.321].

175. In September 2012, and shortly prior to Mr Douglas' death, TCH released the protocol *Standard Operating Procedures: Post Mortem Examinations and Retention of Body Tissue – Adult Patients* [IB B.608]. That protocol provides comprehensive guidance in relation to requesting a hospital autopsy. While that protocol required a detailed history to be included on the request/consent form, which was not done in Mr Douglas' case,

that detailed information was nevertheless conveyed and there was no adverse consequence due to the failure to place that information on the designated form.

Supervision during the autopsy

176. In 2013, Dr Ajmal was a registrar training to be a pathologist. At the time of Mr Douglas' autopsy she had only conducted one previous adult autopsy. That autopsy was limited to the brain, and Dr Hallam had supervised her. [2T 58.41].

177. Dr Hallam testified she was not routinely doing autopsies at that time but had extensive experience in autopsy pathology. She stated she discussed with Dr Brown, another senior pathologist in the department, that she was rostered to conduct reviews of surgical biopsies, so her supervision of Dr Ajmal would be intermittent. Dr Hallam did not recall how many surgical reviews she had to do, but the usual workload was 20 to 25 reports [T 412.34]. Dr Hallam stated she knew Dr Ajmal had done one adult autopsy. Dr Hallam and Dr Brown agreed that as it was a limited autopsy, Dr Ajmal would be a suitable registrar to conduct the autopsy [T 379.15].

178. Dr Hallam stated she asked Dr Ajmal to do the gross examination, then went away and came back when Dr Ajmal had completed that process [T 412.41]. Dr Ajmal said Dr Hallam did the external appearance and Dr Ajmal took notes [2T 59.36]. Dr Ajmal could not say whether Dr Hallam was present while all the external examination was undertaken as Dr Hallam was "in and out". She did not recall which parts Dr Hallam was present for. When Dr Ajmal did some work, Dr Hallam came back and checked it [2T 59.45].

179. The chest was cut open by the mortuary technician. Dr Hallam was not present at this point but came back and looked at the organs in situ. Dr Ajmal did not notice anything exceptional about the organs. She was concentrating on the process and was depended on Dr Hallam's "descriptions and lead" [2T 62.27].

180. On instruction Dr Ajmal took a swab from a white cream coloured lesion on the chest wall. The lungs were removed by the mortuary assistant with only Dr Ajmal present [2T 63.42]. Dr Ajmal then sliced the lung into sections. She did not now recall specific directions but tried to cut them as she had been directed at the time by Dr Hallam. She recalled Dr Hallam being present for this [2T 65.15]. Dr Hallam's evidence was that she had not been present at that time [T 383.28].

181. Dr Hallam's evidence was that she said to Dr Ajmal, "please can you take some photographs and then sample extensively and I don't think that we need to keep the lungs" [T 385.34]. Further, her evidence was "I told her specifically to sample the area in the left upper lobe extensively and to take samples from the chest wall" [T 385.36]. She then left the autopsy [T 386.18]. When asked whether she was present when the blocks were taken, she replied, "I do not remember being present and I think I probably wasn't" [T 444.11].

182. Dr Ajmal confirmed no photographs were taken. She could not recall if she was asked to do so [2T 70.13]. Dr Hallam did not check to ensure photos had been taken.

183. Dr Ajmal agreed that blocks 1 to 8, described within the autopsy report [CB 198], were taken from the lung mass in the left upper lobe and the other blocks were taken from the areas named. She said Dr Hallam told her where to take the blocks from but could not specifically recall instructions such as taking blocks from the edge of the mass [2T

69.5]. She could not recall if she did take the samples from the edge of the mass [2T 69.23]. Dr Ajmal could not recall being asked to take samples from the chest wall [2T 69.10] and did not do so.

184. In light of Dr Hallam's specific memory of the above requests, and Dr Ajmal's uncertainty about those requests, the fact that the chest wall was of particular interest at the autopsy, and that Dr Ajmal was new to the process, I accept the evidence of Dr Hallam that the requests were made.

185. Dr Ajmal could not recall whether Dr Hallam came back to check what she had done after Dr Ajmal had removed the tissue samples and placed them in the appropriate specimen cassettes [2T 73.1].

186. Dr Hallam agreed that she knew at the time of the autopsy there had been an invasion of the chest wall [T 444.30] and that it was important to make sure that sections of the chest wall were taken to consider the nature of the invasion [T 444.35]. She agreed it was not done, saying, "that wasn't done and I take full responsibility" [T 444.38]. She acknowledged that in retrospect directing the registrar to do it led to tissue not being sent to microbiology that limited the information that they could provide to the family [T 444.40]. She also agreed that she could not say that the registrar sampled the edge area of the mass [T 445.5].

187. Prof Duflou indicated it would have been particularly useful, in this case, to have a sample from the chest wall [2T 199.28]. He also explained that while photographs are now routinely taken during autopsies, the process is very disruptive and the images may show nothing of the interior of the body. They do not capture what is actually seen [2T 205.12].

188. Following this autopsy the Clinical Review Committee considered the matter and made recommendations. In response, TCH reviewed the *Medical Officers Post Mortem Manual*. Chapter 2 of that manual, *Hospital Post Mortems*, was last updated in 2015 and now contains, at (2.5) [IB B.618], the express requirement

> *The supervising Pathologist is to determine the level of proficiency of the attending Registrar before the Registrar can work unsupervised and left unattended during the post mortem procedure.*

189. It was submitted that I may consider recommending specific criteria be established for determining a registrar's proficiency for the purpose of performing an autopsy unsupervised. Clearly I am not in a position to specify any such criteria. Further, I query whether it is realistic to expect TCH to reduce the process of assessing such proficiency to a list of universally helpful criteria. I anticipate the manual has been intentionally left without such a list, to allow clinical judgment by seasoned pathologists to operate unfettered based upon the unique circumstances of each case and each registrar.

190. In any event the extant requirement is for the pathologist to positively determine a registrar's proficiency before the latter is left unsupervised during an autopsy. The requirement inherently requires that determination to be undertaken in a deliberate and considered way, and would also inform what level of supervision the pathologist should maintain.

191. Assoc Prof Jain agreed that since the revised protocol, there has been an increased focus on supervision [T 193.13].

192. Finding No. 13: During the autopsy Dr Lavinia Hallam directed the assisting registrar, to take photographs of the body and a section sample from the chest wall. The photographs and section sample were not taken. Dr Hallam did not check whether the photographs and section sample were taken, and did not discover that they were not taken until after the completion of the autopsy.

Conclusion

193. I acknowledge that these reasons, findings and recommendations bring to an end a process that may have been perceived as particularly long for Mr Douglas family. It is my hope that this inquest has illuminated the circumstances surrounding Mr Douglas' death in a way that is useful to his family and the treating clinicians.

194. I would like to formally record my appreciation to counsel, their instructors, parties, investigating police, experts and witnesses for the way they participated in this inquest. The volume of material was significant and the issues numerous. The inquest may have taken much longer had individuals resolved to be less helpful.

195. Finally, I would like to repeat my condolences to the family of Mr Douglas and thank them for their patience while this process has unfolded.

I certify that the preceding one hundred and ninety five (195) numbered paragraphs and the following three annexures are a true copy of the reasons, findings and recommendations of his Honour Coroner Theakston.

Associate: Sam Lynch

Date: 30 January 2018

Annexure A

Findings and Recommendations

	Findings	Paragraph
1	In March 2012, Mr Douglas was suffering from lung cancer.	69
2	The treatment for Mr Douglas' lung cancer was appropriate.	78
3	Following Mr Douglas' re-admission to The Canberra Hospital on 24 October 2012, Mr Douglas received systematic and vigorous treatment for a respiratory infection.	128
4	On 11 December 2012, Mr Douglas died at The Canberra Hospital.	129
5	Mr Douglas' death was directly caused by respiratory failure, with antecedent causes including: (a) chronic emphysematic lung disease; (b) inflammatory lung damage, including possibly from radiation therapy; and (c) acute respiratory infection of unknown type and origin.	130
6	On 3 April 2012, Assoc Prof Sanjiv Jain, when reporting on the core biopsy sample taken from Mr Douglas on 30 March 2012, incorrectly diagnosed the sample as malignant and as evidencing primary pulmonary adenocarcinoma. The sample involved features making diagnosis difficult.	139
7	Treating clinicians relied upon Assoc Prof Jain's diagnosis, as the diagnosis informed treating clinicians about what treatment would be appropriate. It also deterred clinicians from repeating the biopsy that may have in turn provided a reliable diagnosis.	140
8	In April 2012, ACT Pathology had in place a process of reviewing lung core biopsies. That process was linked to a weekly quality assurance meeting. However, due to the Easter long weekend the quality assurance meeting of 10 April 2012 did not take place and the core biopsy taken from Mr Douglas' lung on 30 March 2012 was not reviewed by a second pathologist.	147
9	In 2013, ACT Pathology established a quality assurance process, decoupled from any meeting, with a protocol clearly setting out how biopsies, including all lung core biopsies, would be reviewed by a second pathologist. That process includes quarterly audits to ensure compliance and adequately addresses the systemic failings associated with Mr Douglas' biopsy.	148
10	A number of clinicians erroneously assumed the Epidermal Growth Factor Receptor Mutation Analysis conducted by the Peter MacCallum Cancer Institute critically reassessed the original diagnosis of adenocarcinoma.	158

	Findings	Paragraph
11	There were limited records available from the Lung Multidisciplinary Meeting of 16 April 2012.	162
12	Following Mr Douglas' death, The Canberra Hospital conducted an autopsy, limited to the lungs and chest wall, as consented to by Mr Douglas' wife, Mrs Kerry Douglas. The limited nature of that autopsy was appropriate given what was known at the time.	171
13	During the autopsy Dr Lavinia Hallam directed the assisting registrar, to take photographs of the body and a section sample from the chest wall. The photographs and section sample were not taken. Dr Hallam did not check whether the photographs and section sample were taken, and did not discover that they were not taken until after the completion of the autopsy.	192

	Recommendations	Paragraph
1	The Canberra Hospital continue to periodically review its quality assurance processes for core biopsies, to ensure that such processes appropriately balance the need to minimise the risk of errors with the costs of associated control measures. Such processes should involve an element to check and ensure compliance.	149
2	The Peter MacCallum Cancer Institute consider reviewing the words used within its reports for the Epidermal Growth Factor Receptor Mutation Analysis to ensure that treating clinicians are disabused of any erroneous assumption that the test either reassesses the original diagnosis or positively identifies tumour cells.	159
3	The Canberra Hospital consider introducing a protocol that require appropriate records be made of Lung Multidisciplinary Meetings, and that such records be appropriately stored.	163

Annexure B

Key to Evidence References

[T x.y] – Transcript for the hearings conducted across 21 to 25 November 2016, at page x, line y.

[2T x.y] – Transcript for the hearings conducted across 26 to 28 April 2017, at page x, line y.

[CB x] – Chronological bundle of documents, at page x.

[IB x.y] – Inquest Brief, folder x, at page y.

[Exh x] – Exhibit x.

Annexure C

Chronology of Significant Events

Date	Event	Evidence
27 November 1945	Robert Douglas was born.	
29 October 1997	Mr Douglas underwent fine needle aspiration cytology to left axillary node which showed some necrotic tissue and no obvious malignant cells present.	CB1
29 October 1997	Microbiology test of fluid from fine needle lymph node showed no microbial growth after 72 hours.	CB3
10 December 1997	Mr Douglas seen by Dr Collignon at TCH with possible diagnosis of tuberculosis or atypical mycobacterial infection.	CB6
23 January 1998	Dr Collignon reported to Dr Tait that, for practical purposes, he had to assume mycobacterium tuberculosis, even though there were a number of unusual features. In any case, even if it was an atypical mycobacteria, he had responded very well to the medications.	CB15
29 March 1999	Prof Collignon reviewed Mr Douglas who had been off the full course of therapy for six months with no problems. It remained unclear whether he had atypical or atypical mycobacterium.	CB19
22 April 2008	Mr Douglas presented to Prof Collignon having had six months of sweats and fevers. Prof Collignon reported the cause of the sweats and chills was not clear, but it was unlikely that Mr Douglas had a recurrence of the atypical mycobacteria from 1997.	CB22
18 October 2010	Mr Douglas admitted to TCH with atrial fibrillation.	CB30
28 September 2011	Mr Douglas presented to TCH Emergency Department with sharp pain and associated pain radiating to the back of the neck and tingling down right arm.	CB38
15 February 2012	Mr Douglas has x-ray of chest as recommended by Dr Clifford, his GP. Reported mild linear atelectasis/parenchymal scarring at left lower zone laterally and the remaining lungs appear clear. No S-based consolidation was seen.	CB39A
19 March 2012	Mycobacteriology test on sputum for acid fast stain which was negative in both microscopy and culture.	CB40

Date	Event	Evidence
26 March 2012	Dr Clifford referred Mr Douglas for a CT scan of the chest which identified a mass anteriorly within left upper lobe measuring approximately 40mm in diameter. Evidence of extension into the chest wall with destruction of the anterior aspect of the left first rib and probable direct extension into the deep aspect of pectoralis major.	CB41
26 March 2012	Mr Douglas presents to TCH on referral from Dr Clifford. History of six weeks unwell with chesty cough, fevers and night sweats and left shoulder pain.	CB42, CB43
28 March 2012	Examination by Drs Hammett and Needham noting temperature of 36.4 and a plan for a CT FNA, PET scan.	CB52
28 March 2012	Corrected calcium reading of 2.67, up from 2.59.	CB53; CB54
30 March 2012	Core biopsy of left upper lobe mass.	CB59 to 60
5 April 2012	Mr Douglas reviewed by Dr Tharion.	
5 April 2012	Mr Douglas discharged from TCH.	CB62
11 April 2012	Mr Douglas underwent a PET scan. The left upper lobe lesion was intensely FDG avid.	CB69
13 April 2012	Mr Douglas reviewed by Assoc Prof McCaughan.	CB70
16 April 2012	Mr Douglas' case was reviewed at the Lung Multi-Disciplinary Meeting.	CB71
18 April 2012	Dr Nguyen takes history when Mr Douglas attends radiation oncology.	CB74-76
23 April 2012	Dr Ali takes history when Mr Douglas' attends chemotherapy.	CB77-78
27 April 2012	Report from Peter McCallum Centre on negative EGFR mutations.	CB84
10 May 2012	Mr Douglas commenced six weeks of radiation and chemotherapy.	CB90
14 May 2012	First report of chest wall swelling.	CB91
20 June 2012	Six week course of radiation treatment ends.	
24 July 2012	CT of chest undertaken.	CB98
30 August 2012	Noted that Mr Douglas' chest mass has increased and was hard.	CB106

Date	Event	Evidence
26 September 2012	Chest CT scan.	CB111
11 October 2012	Assoc Prof Ball at Peter McCallum Centre reviewed Mr Douglas.	CB119, 120
12 October 2012	PET scan at the request of the Peter McCallum Centre.	CB124
18 October 2012	Mr Douglas' case was discussed at the Peter McCallum LMDT meeting.	CB128
22 October 2012	A swab taken from Mr Douglas showed staphylococcus aureus in left chest lump.	CB130
24 October 2012	Chest x-ray taken of Mr Douglas.	CB137
25 October 2012	NCDI core biopsy of chest wall mass.	CB138
26 October 2012	Mr Douglas re-admitted to TCH under Dr Chua and receives chemotherapy.	CB139, 140
30 October 2012	Swab of chest and light growth of staphylococcus aureus.	CB144
31 October 2012	A ZN stain from sputum sample and no stain, nor culture, grown.	CB145
1 November 2012	Chest CT and compared with CT of 11 April 2012.	CB147
8 November 2012	Mr Douglas reviewed by Prof Collignon.	CB154
12 November 2012	Mr Douglas' case considered at the Lung MDM.	CB166
12 November 2012	Prof Collignon considered possible bacterial actinomycosis.	CB168
19 November 2012	A core biopsy of chest undertaken. No cancer cells found.	CB170, 171
23 November 2012	Microbiology of chest wall abscess shows no growth.	CB173
28 November 2012	CT scan of chest.	CB177

Date	Event	Evidence
1 December 2012	Sputum test and respiratory tract flora detected.	CB182
9 December 2012	X-ray showed consolidative change throughout the right lung and within the left mid to upper zones with a differential diagnosis of overwhelming infection, radiation pneumonitis and drug reaction.	CB 186
11 December 2012	Mr Douglas died.	
13 December 2012	Post-Mortem of Mr Douglas commences. Gross examination conducted and samples sent off for analysis. Final report completed on 10 Jan 2013.	CB193
9 January 2013	Dr Hallam consults with Dr Jain regarding core biopsy results. Dr Hallam also consults with Drs Bennett, Llewellyn and others. Opinions entered by Drs Bennett and Llewellyn into “silent field” of core biopsy report of 30 March 2012.	
23 April 2013	ACT Health refers review of Mr Douglas’ death to the Chief Coroner.	