CORONERS ACT, 1975 AS AMENDED

SOUTH *AUSTRALIA*

FINDING OF INQUEST

An Inquest taken on behalf of our Sovereign Lady the Queen at Adelaide in the State of South Australia, on the 9th and 10th days of March 2004 and the 30th day of April 2004, before Wayne Cromwell Chivell, a Coroner for the said State, concerning the death of Iris Patricia Young.

I, the said Coroner, find that, Iris Patricia Young aged 60 years, late of Unit 9, 225 Devonport Terrace, Prospect, South Australia died at the Royal Adelaide Hospital, North Terrace, Adelaide, South Australia on the 1st day of December 2000 as a result of haemorrhagic shock and respiratory failure due to right-sided haemothorax complicating right thoracentesis in a person with severe chronic obstructive pulmonary disease and metastatic breast carcinoma.

1. Introduction

1.1. Mrs Iris Patricia Young was aged 60 years at the time of her death.

1.2. Mrs Young had been diagnosed with breast cancer in 1993. She underwent a left-sided mastectomy that year. Unfortunately, the cancer metastasised to her bones and she underwent radiotherapy and chemotherapy as a result of which she was declared 'clear' in March 1999.

1.3. Mrs Young also suffered from severe chronic obstructive airways disease (COAD). She had been using a nebuliser at home.

1.4. Admission to the Royal Adelaide Hospital

Mrs Young was admitted to the Royal Adelaide Hospital (RAH) on 18 November 2000. She was complaining of an increase in her chronic back pain with radiation

down her back, an increase in anterior left chest pain and shortness of breath, lethargy and tiredness.

1.5. On examination by the admitting doctor, Mrs Young's past medical history was noted to include COAD, breast cancer, depression, osteoporosis, hypothyroidism, diverticular disease, and fibromyalgia. It was noted that she was still smoking cigarettes, although she told another doctor in the Emergency Department that she had stopped smoking four weeks ago. She was described during an earlier admission in 2000 as an 'unrepentant smoker'.

1.6. Following her admission, Mrs Young was given an infusion of Heparin, among other medications. Heparin is an anticoagulant medication which is given to prevent blood clotting, particularly deep vein thrombosis. This medication was continued for several days until 21 November 2000 when it was discontinued.

1.7. Further investigation of Mrs Young's condition, including CT scanning, demonstrated that the cancer had spread further into her lungs and liver. A pleural effusion (fluid in the pleural cavity) and marked mediastinal lymphadenopathy (enlargement of the lymph nodes in the space between the two lungs) were also noted.

1.8. Dr Tabitha Healey was then a Senior Registrar in Medical Oncology. Mrs Young had been transferred to the Oncology Ward on admission. As part of her treatment plan, Dr Healey directed that Mrs Young's right pleural effusion be 'tapped'. This is shorthand for thoracocentesis or thoracentesis (the surgical perforation of the chest wall and pleural space with a needle to aspirate fluid for diagnostic or therapeutic purposes or to remove a specimen for analysis). Dr Healey indicated in the clinical record that the purpose of this procedure was to improve Mrs Young's respiratory status, and to provide a specimen for cytology (cell analysis) (Exhibit C5a).

1.9. The thoracentesis was performed by Dr Bruno Franchi, the Resident Medical Officer. Dr Franchi was able to aspirate only 100mls of fluid or so.

1.10. On 27 November 2000 Mrs Young was reviewed by Dr Simon Hawkins, a Senior Registrar in the Thoracic Medicine Unit. Mrs Young's discharge from hospital was being discussed, and he suggested that home oxygen might be prescribed on palliative grounds if Mrs Young was able to survive the chemo/radiotherapy to the time of discharge. He suggested that before Mrs Young was discharged she should have a

repeat chest X-ray, and a repeat pleural fluid drainage in order to improve her lung function.

1.11. On 30 November 2000, Dr Franchi performed a further thoracentesis. The procedure was performed at 9:40am. This time the procedure was slightly more effective and 400mls of fluid were drained before drainage ceased spontaneously, and Dr Franchi was unable to restart it.

1.12. Following the procedure, Dr Franchi directed that Mrs Young be observed and her oxygen saturations taken hourly for three hours, and he directed that routine observations be performed after that. There is nothing in the clinical record to indicate that this direction was complied with.

1.13. At about 12:40pm Mrs Young suffered an acute deterioration in her condition. She complained of severe abdominal pain, shortness of breath, and a desire to use her bowels. Her blood pressure was unobtainable.

1.14. Dr Franchi was called and he arrived somewhere between 1pm and 1:10pm. He found Mrs Young to be pale, sweaty and breathing rapidly. Her oxygen saturations were 94% which is an acceptable level.

1.15. In consultation with Dr Dorothy Keefe, a consultant in Medical Oncology, the presumptive diagnosis was of a pulmonary embolism.

1.16. Because it was difficult to obtain a blood pressure, Dr Franchi ordered that Mrs Young receive Haemaccel which is a blood volume-increasing agent. He also directed that an ECG, chest X-ray, and blood tests be undertaken.

1.17. More significantly, Dr Franchi also directed that Mrs Young receive Heparin. He prescribed a bolus dose of 5000 units, and then an infusion of 1000 units per hour. The bolus dose was administered at 1:25pm.

1.18. The mobile X-ray unit attended and chest X-rays were taken at 1:35pm.

1.19. It is not clear from the clinical record precisely when the X-ray films were delivered to the Oncology Ward, however, as a result of viewing those films Dr Franchi called Dr Hawkins, the Registrar in Thoracic Medicine. Dr Hawkins' note records that he was called at 2:10pm, so the X-rays must have arrived shortly before that. Dr

Hawkins arrived at 2:30pm, viewed the X-rays, and noted the presence of a large effusion in the right pleural cavity, so much so that the trachea was deviated to the left.

1.20. On the basis of this finding, it was apparent that the initial presumptive diagnosis of pulmonary embolism was incorrect. Mrs Young had a pleural effusion in the right chest cavity which was possibly a haemothorax (blood in the pleural space). This was a medical emergency and was a life-threatening situation for Mrs Young.

1.21. The administration of Heparin was ceased at 2:35pm, very soon after Dr Hawkins arrived, because if Mrs Young was haemorrhaging, the Heparin was likely to exacerbate her condition.

1.22. An emergency thoracentesis was performed by Dr Hawkins at 2:55pm and he noted the presence of frank blood. This confirmed the presence of a haemothorax. Dr Hawkins discussed the case with Associate Professor Scicchitano, who suggested that an opinion be obtained from the Cardio-Thoracic Surgical Unit. Dr B Thompson from that Unit reviewed Mrs Young and he agreed that she was suffering a tension haemothorax (where the pressure of the accumulated blood has pushed structures inside the chest out of position). Dr Hawkins' note reads:

> 'Given extensive medical comorbidities conservative management best.'
> (Exhibit C5)

1.23. Mrs Young was given Protamine at 3:10pm in order to reverse the anticoagulation effects of the Heparin.

1.24. A blood transfusion was commenced at 3:30pm. At 4pm a 24 gauge Argyle catheter was inserted, and an underwater seal drain was connected. 1200mls of blood was drained from the pleural cavity. After the fluid was removed, Dr Hawkins noted a significant improvement in Mrs Young's condition, particularly in relation to her oxygen saturations and blood pressure.

1.25. Mrs Young was transferred to the High Dependency Unit (HDU) at the Royal Adelaide Hospital at 5:25pm that afternoon. Unfortunately, her condition deteriorated further and, in discussion with HDU consultants, Dr Keefe, Mr John Stubberfield the Cardio-Thoracic Surgeon, and Mrs Young's family, it was agreed that conservative

measures would be persisted with, and that operative intervention was not indicated in view of Mrs Young's general condition.

1.26. The haemorrhage into the pleural space continued through the evening of 30 November 2000 and Mrs Young's condition continued to deteriorate. At 12:45am her respirations and pulse ceased, and her pupils were fixed and dilated. Dr David Wabnitz pronounced her life extinct at 1:15am on 1 December 2000 (see Exhibit C2).

2. Cause of death

2.1. A post-mortem examination of the body of the deceased was performed by Dr Yung Tran, a first-year trainee Pathologist at the Institute of Medical and Veterinary Science, under the supervision of Consultant Pathologist, Dr Andrew Ruszkiewicz.

2.2. Drs Tran and Ruszkiewicz determined that the cause of death was 'right sided haemothorax in a person with metastatic breast carcinoma, involving multiple organs including the lungs, mediastinum, lymph nodes, diaphragm and liver.' (Exhibit C7).

2.3. The pathologists commented as follows:

> '1. The autopsy examination revealed the presence of widespread metastatic disease involving the lungs, mediastinum, lymph nodes, diaphragm, and liver.
>
> 2. Involvement of the respiratory system organs by metastatic cancer is often associated with development of pleural effusion(s). In such cases, the pleural effusion would further increase the level of respiratory impairment due to pre-existing metastatic pulmonary disease.
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> 3. Thoracocentesis is an invasive procedure used in the treatment of pleural effusions.
>
> 4. Haemorrhage into the pleural cavity is a recognised complication of thoracocentesis, particularly in patients who may have an underlying coagulopathy (clotting disorder) which can be associated with widespread metastatic cancer.
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> 5. Individuals with disseminated malignancy have a significantly increased risk of developing pulmonary thromboembolism, which is a condition associated with a high mortality. Pulmonary thromboembolism requires immediate intervention, often instituted on a presumptive diagnosis. The clinical presentation of pulmonary thromboembolism can mimic the respiratory difficulties observed in patients with various underlying disorders including pleural effusion and haemothorax.'
>
> (Exhibit C7, p1)

2.4. The pathologists noted that two bruises, consistent with thoracentesis procedures, were identified on the right lower back, inferior to the scapula. Although it is not completely clear from the clinical record, I infer that these two bruises were the sites

of the thoracenteses performed by Dr Franchi on 24 and 30 November 2000. The thoracentesis performed by Dr Hawkins after Mrs Young's collapse, was performed in the mid-axillary line (directly below the armpit) and was therefore in a different area. This was also the site of insertion of the 24 gauge Argyle catheter as part of the underwater seal drain.

3. Issues arising at inquest

3.1. The post-mortem examination

I heard evidence from Dr Andrew Holt, who is a Consultant in Critical Care Medicine at the Flinders Medical Centre. Dr Holt provided me with detailed and very helpful reports concerning the treatment administered to Mrs Young during this admission, and the subsequent investigation of the cause of death. The reports are Exhibits C8 and C8a respectively.

3.2. Dr Holt was critical of the pathologists on the ground that they did not identify the exact site of the haemorrhage into the pleural space at post-mortem. He said:

> 'My conclusion that there was a deficiency in the post mortem report in determining the exact site of haemorrhage, I believe remains valid. Given the external skin sites of the thoracocentesis were identified, I remain surprised that it is not possible to conclude the likelihood of one of them being the site of bleeding. Given the thoracocentesis caused the haemorrhage, then I would have expected some evidence of a source of bleeding from the chest wall in relation to the needle path or the needle path appearing to pass through tumour deposits on the parietal pleura. The reason to expect some evidence relates to the fact that the haemorrhage caused a tension haemothorax and I would expect that the source of bleeding of that magnitude to be apparent.'
>
> (Exhibit C8a, p2)

3.3. Dr Tran agreed with this criticism. She explained that she was concentrating more intensely upon the site of the chest drain which, in retrospect, and having regard to the chronology of events, was not relevant to the causation of the haemothorax (see her evidence at T126).

3.4. At the time of the post-mortem, Dr Tran was very inexperienced and I do not criticise her in relation to this issue. It is surprising, however, that this line of inquiry was not pursued in consultation between the two pathologists. The question of the causation of the haemothorax is central to an understanding of the cause of Mrs Young's death, which was the purpose of the post-mortem examination.

3.5. The second criticism made by Dr Holt was that he suggested that the cause of death postulated by the pathologists did not adequately reflect the role thoracentesis played. Based upon the clinical history and in particular the timing of Mrs Young's collapse, Dr Tran agreed that the haemothorax was caused by the thoracentesis performed by Dr Franchi on the morning of 30 November 2000 (T130).

3.6. Finally, Dr Holt disagreed with comment 4 in the post-mortem report quoted above. He explained that even after the haemothorax was detected, there was no evidence of a disseminated intravascular coagulopathy, which is sometimes associated with metastatic carcinoma. He said:

> 'At 2349 hours on 30 November 2000, D-Dimer fibrin degradation products were normal. I also note that at 1419 hours on 30 November 2000 the INR was normal at 1.1. Both these results are consistent with normal coagulation status.
>
> I conclude that any coagulopathy present at the time of the haemothorax was as a consequence of drugs administered with aspirin affecting platelet function and administration of the anticoagulant Heparin (as a consequence of the mistaken diagnosis of pulmonary embolism).'
>
> (Exhibit C8, p2)

3.7. Accordingly, Dr Holt disputes that Mrs Young was showing any signs of coagulopathy prior to the thoracentesis, whether as a result of her metastatic cancer or otherwise. His point was that any coagulopathy which may have contributed to her condition was caused by the Heparin and not by her underlying medical condition.

3.8. I accept Dr Holt's evidence on this topic and find that Mrs Young did not have any sign of coagulopathy prior to the thoracentesis procedure, and that any coagulopathy which exacerbated the haemorrhage was caused as a result of the administration of Heparin.

3.9. Taking all these matters into account, I will make a finding as to the cause of death which has been proposed by Dr Holt, but which is different from that proposed by the pathologists. I find that the cause of Mrs Young's death was haemorrhagic shock and respiratory failure due to right-sided haemothorax complicating right thoracentesis, in a person with severe chronic obstructive pulmonary disease and metastatic breast carcinoma.

3.10. <u>Thoracentesis - performance by Dr Franchi</u>

Dr Holt said that, in his opinion, both thoracentesis procedures performed by Dr

Franchi were appropriate treatment for Mrs Young's illness (T168). He said that the technique used by Dr Franchi in both thoracentesis procedures was in accordance with common practice and he had no criticism of the way in which Dr Franchi performed these procedures (T140-141). I accept Dr Holt's evidence on these matters, and find accordingly.

3.11. As to complications caused by thoracentesis, Dr Holt said:

> 'Injury to the lung and pneumothorax has been described in 4-30% of patients undergoing thoracentesis. A major risk factor for lung injury and pneumothorax is chronic obstructive pulmonary disease due to hyperinflation of the lung. The complication of haemothorax following thoracentesis is much less common. In the references I have cited, there appears to be 4-5 cases where intercostal catheters or blood transfusions were required due to haemorrhage following thoracentesis. This will put the rate of serious bleeding following thoracentesis down around 1%.'
>
> (Exhibit C8, p4)

3.12. Dr Tran found no sign of injury to the lung and, since the bleeding site was not investigated more thoroughly, the precise cause for the haemorrhage in Mrs Young's case cannot now be determined accurately. I was told that it is possible that Dr Franchi's needle may have passed through a cancerous lesion on the pleural surface, which was more likely to bleed than normal tissue. Alternatively, a major blood vessel, such as the intercostal artery, may have been damaged on insertion of the needle. It is possible that the artery was in an anomalous position. The fact that it was damaged does not necessarily imply any failure of technique on the part of Dr Franchi (see for example the evidence of Dr Tran at T130).

3.13. <u>Chest X-ray following thoracentesis</u>

Dr Holt suggested that a chest X-ray should be performed after every thoracentesis procedure. He said:

> 'It is recommended that a chest X-ray should be routinely performed following thoracentesis, the major reason being to detect the more common complication of pneumothorax (4). A chest X-ray may be variable help with respect to the diagnosis of haemorrhage from the procedure. A chest X-ray performed immediately after the procedure and in the setting of a slow rate of haemorrhage may show no abnormality. As previously mentioned, problematic haemorrhage is often diagnosed at the time of the procedure on the basis of heavy blood staining of the aspirate. The bleeding in Ms Young's case was substantive and should have been diagnosed with a chest X-ray.'
>
> (Exhibit C8, p5)

3.14. Dr Franchi told me that it is not usual procedure at the Royal Adelaide Hospital to perform a chest X-ray in every case following thoracentesis. He said that a chest X-ray would only be expected in cases where complications were expected, or where there were signs during the procedure that complications might arise (T27).

3.15. Dr Holt said that even if this approach was accepted, Mrs Young represented an increased risk of pneumothorax because of her COAD, and an increased risk of bleeding because of her known malignancy, so that even if chest X-rays were to be considered on a case-by-case basis, he believed that a chest X-ray was indicated in Mrs Young's case (T147).

3.16. Dr Holt said that in all his discussions with medical colleagues on this issue, the view was unanimous that, ideally, a chest X-ray should be performed in every case. However, he acknowledged that a failure to do so was not necessarily associated with a poor patient outcome (T148).

3.17. With the benefit of hindsight, it is likely that if a chest X-ray was performed within, say, an hour of the thoracentesis on 30 November 2000, the haemothorax may have been detected. Having been detected, appropriate interventions could have been undertaken and Heparin would not have been administered.

3.18. In view of an established practice at the Royal Adelaide Hospital which is against performing a routine chest X-ray after every thoracentesis, it would be unfair to criticise Dr Franchi because he did not perform one in Mrs Young's case. Even acknowledging Dr Holt's point about Mrs Young's increased susceptibility to complications, there was no sign following the thoracentesis on 30 November that she had suffered any adverse consequences from the procedure until the time of her collapse at 12:40pm. Having regard to that, I do not think it is appropriate to criticise Dr Franchi for not performing a chest X-ray as a matter of routine following the thoracentesis.

3.19. I will discuss whether the established practice at the Royal Adelaide Hospital in this area should be reviewed later in these findings.

3.20. It is noted that Dr Franchi ordered a chest X-ray as soon as Mrs Young's condition deteriorated, and this was performed within a maximum of 35 minutes. The films were made available and read by Dr Franchi within a further 30 minutes. Dr Holt said

that in his opinion, these times were 'reasonable', having regard to the logistical issues involved (T165-166). I accept this evidence, and find that there was no undue delay in the performance of the chest X-ray once it was ordered.

3.21. Diagnosis of Mrs Young's condition following her collapse

It is acknowledged by all of the clinicians who gave evidence that the initial provisional diagnosis of pulmonary embolism was wrong.

3.22. Dr Keefe pointed to the symptoms displayed by Mrs Young following her collapse, namely abdominal pain, a desire to open her bowels, shortness of breath, pallor, sweating, a weak and very rapid pulse, soft and non-tender abdomen, and unrecordable blood pressure, and argued that these symptoms were all consistent with a picture of a pulmonary embolism. She said:

> 'As we are all aware that was not the correct diagnoses, however, Mrs Young was at an extremely high risk of a pulmonary embolus. She had a widespread malignancy, she had chronic lung disease, she had been largely confined to bed for the last couple of weeks. She was also over the age of 50 which is yet another risk factor for these events. Pulmonary embolus is extremely common in cancer patients, it is in fact the second commonest cause of death in cancer patients. It is something that one sees all the time in a cancer ward. As against that the diagnosis of haemothorax post procedure is about 1:1000. It was a clinical decision based on likelihoods, and it was the wrong diagnosis.' (T86)

3.23. The dilemma faced by Drs Keefe and Franchi was that if Mrs Young had suffered a pulmonary embolus, she needed to receive urgent treatment. Dr Keefe produced an article in a publication of the American College of Chest Physicians (*Chest.*2002:121:877-905.) by Kenneth E Wood, DO, FCCP of the University of Wisconsin. Dr Holt described this as 'an excellent article covering the diagnosis and management of pulmonary embolus' (T153). In that article, the author comments:

> 'Physical findings and standard data crudely estimate the severity of the embolic event in patients without prior cardiopulmonary disease (CPD) but are unreliable indicators in patients with prior CPD. In either case, the presence of shock defines a threefold to sevenfold increase in mortality, with a majority of deaths occurring within 1 hour of presentation. A rapid integration of historical information and physical findings with readily available laboratory data and a structured physiologic approach to diagnosis and resuscitation are necessary for optimal therapeutics in this "golden hour".'

Later, the author states:

> '**Heparin**
> When PE (pulmonary embolism) is first suspected, patients should receive heparin at therapeutic doses until PE is excluded, provided that no contraindications to

> anticoagulation are present … The efficacy of heparin is attributed to an impairment of clot propagation and the prevention of recurrent PE. The risk of recurrent venous thromboembolism is highest in the early stages, and, because recurrent PE is reported to be the most common cause of death in hemodynamically stable patients, it is crucial to rapidly achieve a therapeutic level of anticoagulation. An inability to establish an early therapeutic level for the activated partial thromboplastin time (aPTT) is associated with a higher rate of recurrence and impairs the efficacy of anticoagulation therapy with warfarin.' (my underlining)
>
> (Exhibit C6c, p1 and pp21-22)

3.24. Dr Holt said that, in his opinion, pulmonary embolism was not the most likely diagnosis, even on the clinical findings, particularly having regard to the fact that thoracentesis had been performed so recently prior to the collapse (T152). He pointed out that Mrs Young's symptoms, particularly her pallor and the fact that she was achieving 94% oxygen saturation, were inconsistent with a pulmonary embolism (T163). He said that Drs Keefe and Franchi were not managing a pulmonary embolism, they were managing sudden shock a short time after an invasive procedure. He said that in that situation, the first priority is to resuscitate the low blood pressure using 'vasopressors'. He said:

> 'And I think that (the article) summarises the major role of Heparin on p.21 where its predominant role is stopping a pulmonary embolus getting worse or a further pulmonary embolus. Let's say massive pulmonary embolus with shock was a reasonable diagnosis, I would have to ask why just giving Heparin and moving to a side room was all that was done. The article that I have just referred to emphasises the role, the predominant role of vasopressor to resuscitate the circulation. They are mentioned further up on p.21 as being more important in the early emergency management of massive pulmonary embolus. The limitation of the treatment of pulmonary embolus I'm unsure why (that) occurred, and whether or not that partial or limitation of treatment of pulmonary embolus was discussed with the patient or the family. The administration of vasopressors would have required a special catheter to be inserted and would have required moving to the intensive care unit, which of course both was done once the cause of her collapse was noted to be haemorrhage rather than pulmonary embolus.' (T153)

3.25. In considering a differential diagnosis to that of pulmonary embolism, the most likely one was a pneumothorax (air in the pleural cavity - punctured lung). This was much more likely than haemothorax. Dr Keefe said that pneumothorax could be rejected on clinical grounds, and Dr Holt accepted this (T164). Dr Keefe argued that haemothorax, with a likelihood of perhaps 1 in 1000 (Dr Holt's estimate was higher at 1%) made pulmonary embolism the overwhelmingly likely diagnosis in the circumstances (T86).

3.26. It is always difficult to resolve disagreements about clinical issues where the views of experienced clinicians are involved. Obviously, the clinician who is present with the patient at the time, as Dr Keefe was, has the best opportunity to gauge the patient's condition. That is not to say that there were not objective criteria which could be assessed later, but the advantage of the attending physician must be acknowledged. Dr Holt is an expert in critical care medicine. If Mrs Young had been transferred to the HDU as soon as she collapsed, I suspect that much greater emphasis would have been placed on resuscitation, and less on Heparin therapy. In the context of the Oncology Ward, the misdiagnosis was understandable. For reasons which I will presently outline, the decision not to transfer her was less so.

3.27. Did the misdiagnosis and administration of Heparin affect the outcome?

Dr Holt said that this is a very difficult question to answer having regard to complicating factors in Mrs Young's case, in particular her chronic lung disease, her disseminated malignancy, and the severity of her condition at around 3pm that day. He said:

> 'It is my belief that that bleeding started soon after the - at the time of the thoracentesis and slowly accumulated over the morning. The fact that there had not been noticed any major - there had not been documented any problems other than the apparent complaint of discomfort is quite compatible to the fact that there is physiological reserves and somebody only decompensates once they've lost a critical amount of blood volume. So it was occurring over the afternoon. So how sick she was, the sequelae of her shock, the amount of compression of her lung was all a function of the passage of time and the rate of her bleeding. Would things been substantially different at what was probably 1.30 let's say, the answer to that is quite possibly. Clearly the time to have detected this problem was sometime between 9.40 and 12.40 when she still had a compensated circulation and was not shocked and did not have compressed lung to the same degree.' (T151)

3.28. Dr Keefe emphasised, and I accept, that the provision of Heparin did not cause the bleeding to start, it merely exacerbated it. It was the thoracentesis which initiated the bleeding.

3.29. After the administration of Protamine, Dr Keefe said that the blood test results indicated that Mrs Young's clotting factors were returning to normal. She said:

> 'A: After the Protamine was started, that should have stopped any new damage from the Heparin occurring, but it wouldn't have reversed the damage the Heparin had already caused.
>
> Q: It's impossible to quantify what that is?
>
> A: That's right.' (T96)

3.30. On the basis of that evidence, I find that the administration of Heparin did exacerbate Mrs Young's haemorrhage, but it is impossible to predict whether the outcome would have been different. The most that can be said is that there is a chance that it might have been.

3.31. Why was Mrs Young not transferred to HDU earlier?

I have already mentioned the evidence of Dr Holt when he pointed out, after referring to the article by Dr Wood, that the first priority in resuscitating Mrs Young after her collapse at 12:40pm was the administration of vasopressors to restore her blood pressure (T153). This would have required moving the patient to the High Dependency Unit for the insertion of a special catheter. It may be that, if this had been done as a matter of urgency, the chest X-ray might also have been obtained earlier, the haemothorax might have been diagnosed and the Heparin not administered.

3.32. Dr Keefe's explanation for the limitation on Mrs Young's treatment was that she discussed the issue of further treatment with Mrs Young and her daughter and it was agreed that Mrs Young would remain on the Oncology Ward and would not be transferred to HDU. Dr Keefe's note in the clinical record reads:

> 'DW (discussed with) pt (patient) and daughter - for full ward measures but not for transfer to ITU (Intensive Therapy Unit or HDU).' (T107)

3.33. When asked to explain what this meant, Dr Keefe said:

> 'That means that any treatment that we could give on the ward would be given, but that if we got to a situation where we couldn't support Mrs Young successfully without sending her to intensive care then the decision would be made that she wouldn't go to intensive care. So that there would come a time after which if she hadn't improved then we would realise that this was terminal care.'
>
> (Exhibit C5)

3.34. Mrs Young's daughter, Kathryn Young, vehemently denied that she had any such conversation with Dr Keefe, either in her mother's presence or otherwise. Ms Young said that she was told by Dr Keefe that her mother was suffering from a major blood clot in her lung, that she was very ill and would die. She said that Dr Keefe advised her to 'call who you can' because her sister was interstate. She said Dr Keefe told her that they were giving her mother something to alleviate the pain, and that they would

move her to a side room and 'basically she would die within the next 10 or 15 minutes' (T180).

3.35. When asked whether there was any discussion about moving Mrs Young to the HDU, Ms Young replied:

> 'I did question whether there was anything else that could be done as far as moving her to somewhere where she could get better treatment and she has just said that that wasn't available. She said that Mum was going to die anyway and she just needed to be comfortable.' (T180)

3.36. Somewhat inconsistently with that evidence, Ms Young acknowledged that the doctors were giving her mother medication to reduce the blood clot as well as medication to ease the pain (T188).

3.37. The clinical record clearly demonstrates that not only was Mrs Young given Heparin, she was also given low dose oxygen, Ventolin, Haemaccel, and Morphine for the pain. Dr Franchi sent blood samples off for testing, obtained an urgent ECG, and requested a mobile X-ray.

3.38. It is clear that Dr Keefe explained to Ms Young that her mother's situation was very grave and that it was likely that she would die. However, I do not accept Ms Young's assertion that the doctors were merely providing her mother with palliative care at that stage.

3.39. Ms Young conceded that it was her mother's attitude that she did not wish to be mechanically ventilated on a respirator in the event of a cardiac arrest, although she insisted that this was during a previous admission to hospital. She said:

> 'Certainly, going back maybe when she was – the second time she was diagnosed with cancer she had said "I don't want to be a vegetable on a ventilator", you know, but at no point was that discussion relevant to this trip to hospital.' (T191)

3.40. There is no entry in the clinical record during this final admission which details Mrs Young's wishes in relation to this issue. There is nothing documented in relation to her previous admissions either.

3.41. This is a most unsatisfactory state of affairs. It is not acceptable that there should be a misunderstanding between clinicians and the family of the deceased about such a serious issue. The question whether Mrs Young was to receive active treatment or

palliative care, and the full significance of these options, should have been discussed and a clear, conscious decision reached, and recorded in the clinical record.

3.42. It is clear that by the time a haemothorax was diagnosed, decompressed, and Mrs Young was transferred to the HDU her situation was extremely grave. It is not possible to conclude that the outcome would have been any different if she had been transferred to the HDU at an earlier stage, although, on the evidence of Dr Holt, that cannot be excluded as a possibility. The fact that Mrs Young was not transferred to the HDU as a result of an apparent misunderstanding between Dr Keefe and Ms Kathryn Young is a matter of serious concern.

3.43. Since the oral evidence was completed, I have received correspondence from Mr John Homburg, counsel for Drs Franchi, Keefe and Tran, with which he enclosed a copy of a document entitled 'Guidelines for No Cardio-Pulmonary Resuscitation Orders' developed by the Royal Adelaide Hospital. The Guidelines form part of the Medical Policies and Procedures Manual.

3.44. The order endorsed in the clinical record by Dr Keefe was described as a 'Ward Measures Only' order, which is not quite the same as a 'No CPR' order, however the effect is the same.

3.45. The Guidelines give detailed advice to medical and nursing staff, as follows:

> '1. A 'No Cardio-pulmonary Resuscitation Order' should be recorded as a formal order in the patient's progress notes in a clear and unambiguous manner.
>
> 2. The senior attending medical officer (ie Consultant or Registrar) is responsible for the decision for a 'No Cardio-pulmonary Resuscitation Order'. In making this decision the senior attending medical officer should involve appropriate members of the health care team (e.g. nurses, allied health professionals, other medical staff), in the decision making, although the final decision on the medical side remains the responsibility of the senior attending medical officer.
>
> A 'No Cardio-pulmonary Resuscitation Order' should incorporate a brief description of what discussion took place with the patient and/or family members/surrogate, and if appropriate:
>
> (a) a statement of the patient's wishes (when the patient is competent) or
>
> (b) the role of the family/surrogate (when the patient is incompetent)
>
> 3. Where a decision has been made **not** to involve a patient or surrogate in decisions regarding resuscitation status, an explanation should be provided in the progress notes as to the rationale underlying the decision.

4. A 'No Cardio-pulmonary Resuscitation Order' should include a statement of the medical condition to justify a 'No Cardio-pulmonary Resuscitation Order'.
5. A 'No Cardio-pulmonary Resuscitation Order' should include a statement about the scope of the order, specifying the management plan (curative and/or palliative) subsequent to the 'No Cardio-pulmonary Resuscitation Order '.
6. A 'No Cardio-pulmonary Resuscitation Order ' should be subject to review on a regular basis and can be rescinded at any time. Any review should be implemented and documented in the patient's progress notes in the manner specified above.' (p3-4)

3.46. The document then goes on to give examples of the appropriate wording to be entered in the clinical record. The examples are clear about with whom the issue has been discussed, the reasons why the order is being made, and the fact that specific members of the family have been consulted and agree with the order. I reproduce Example 2 here:

> '*I have not specifically discussed a 'no cardio-pulmonary resuscitation order' with Mr Jones but he understands that no further active treatment will reverse the fact that he is dying due to cardiac failure as a result of his severe ischaemic heart disease. Mr Jones' treatment on the ward should be continuation of his usual medical therapy and comfort measures, but not cardio pulmonary resuscitation in the event of a cardiac arrest.*
>
> *I have discussed the situation with Mr Jones' son who is in agreement that, in the event of cardiac or respiratory arrest, cardio-pulmonary resuscitation would be inappropriate and should not be commenced.*' (p4)

I note that there is no requirement that the patient or relative should acknowledge the agreement in writing. In my view this would be a wise precaution.

3.47. Dr Keefe's entry is nowhere near as detailed as that. In particular, she gave no reasons for making the order, and did not specifically state that Mrs Young understood and agreed with the order. There is no written acknowledgement by Mrs Young or her daughter that these issues have been discussed

3.48. The clinician must take responsibility for explaining the issues clearly with both the patient and relatives in attendance. If the patient and/or relative is not to acknowledge in writing what has been imparted to them, then the clinician must accept responsibility if a misunderstanding arises.

3.49. Ms Young's evidence was that she was anxious not to make final decisions until her sisters and her mother's partner could be present (T183). As it transpired, a decision was made which could have affected the outcome of Mrs Young's illness which is

now disputed by Ms Young. Dr Keefe must take responsibility for the misunderstanding that has occurred.

4. Recommendations

4.1. I am advised by Mr Homburg that two committees at the Royal Adelaide Hospital are reviewing these issues at the moment. He advised that:

> 'Decisions about life-sustaining medical intervention and/or withdrawal of treatment more frequently occur in circumstances when a patient suddenly deteriorates or comes to the hospital consequent on an emergency.'

4.2. As I have pointed out, when decisions of such great importance are made in the midst of a crisis, when everyone involved is highly stressed, the risk of misunderstandings is high.

4.3. Pursuant to Section 25(2) of the Coroner's Act, 1975, I recommend that when policies for the application of withdrawal of treatment are developed, the committees involved should consider how such misunderstandings might be avoided. Perhaps the intervention of a social worker might be encouraged in these situations.

4.4. Certainly, where possible, the reasons given for a particular treatment approach in these situations should be recorded in the clinical record, and, where possible, acknowledged in writing by the patient and the senior available next-of-kin or other legally authorised person.

4.5. As to the issue of X-rays following thoracentesis, in light of Dr Holt's evidence I recommend that the Royal Adelaide Hospital reviews the practice whereby X-rays are not routinely performed. I do not say that an X-ray should be performed in every case, but in high-risk patients such as Mrs Young, an X-ray should be considered after thoracentesis.

Key Words: Hospital Treatment; Thoracentesis; Haemothorax; Palliative Care

In witness whereof the said Coroner has hereunto set and subscribed his hand and

Seal the 30th day of April, 2004.

Coroner