



FINDING OF INQUEST

An Inquest taken on behalf of our Sovereign Lady the Queen at Adelaide in the State of South Australia, on the 22nd, 23rd and 24th days of February 2006, and the 19th day of May 2006, by the Coroner's Court of the said State, constituted of Elizabeth Ann Sheppard, a Coroner, into the death of Salvatore Falanga.

The said Court finds that Salvatore Falanga aged 77 years, late of 56 Henry Street, West Croydon died at the Queen Elizabeth Hospital, 28 Woodville Road, Woodville, South Australia on the 19th day of February 2003 as a result of complications of haemothorax and hypotension, following drainage of pleural effusion, post coronary by-pass surgery, in the setting of longstanding multi-organ disease related to diabetes, hypertension, hyperlipidaemia and previous smoking history. The said Court finds that the circumstances of his death were as follows:

1. Sequence of events

- 1.1. On 9 December 2002, Salvatore Falanga underwent elective coronary artery bypass grafting at Flinders Medical Centre (FMC). He had a history of ischaemic heart disease and suffered from type 2 diabetes, left carotid stenosis, chronic airways disease and hypertension. (T10) These conditions were being managed by his treating physicians, with a range of medications. The cardiac surgery and post-operative recovery proceeded relatively uneventfully, although he suffered some acute renal failure which resolved by the time he was discharged on 17 December 2002. (T14)
- 1.2. When Mr Falanga was discharged from FMC, he had a residual left pleural effusion. This collection of fluid in the pleural space is seen in a number of patients following by-pass surgery and usually resolves without further intervention. In the FMC

discharge summary, reference was made to the effusion and also to Mr Falanga's erratic blood sugar levels and his increased creatinine level. These blood results suggested that his diabetic condition and renal function needed ongoing attention. Adjustments were made to Mr Falanga's medications and follow-up with his general practitioner was suggested in 3 days and with his specialist in 4-6 weeks. (Exhibit C4a)

- 1.3. On 20 January 2003, Dr Katherine Gibb conducted the six week follow-up assessment in her private clinic at Woodville. A copy of the discharge summary was forwarded to her rooms in advance of this appointment. Dr Gibb is employed as a general physician at the Queen Elizabeth Hospital (TQEH) and is currently the Director of Physician Training. She held that position in January 2003 and was also the Director of the High Risk Pre-Operative Clinic and held a position as a consultant physician on the cardiology unit, although she was not a cardiologist. Dr Gibb had seen Mr Falanga in her private practice before his heart surgery and was familiar with his multiple health issues. According to Dr Gibb, she had been managing his health issues to prepare him for his by-pass surgery and to control his long term vascular risk factors. (T11) When she examined Mr Falanga, she recorded his blood pressure reading at 160/85 which prompted her to consider re-introducing Ramipril medication to reduce the blood pressure, if Mr Falanga's renal function was satisfactory. Dr Gibb ordered further blood tests to check on his creatinine level and changed his beta blocker medication from Metoprolol to Atenolol in response to Mr Falanga's complaints of headache. (Exhibit C4a)
- 1.4. During Dr Gibb's examination, she noted signs consistent with "left lower lobe consolidation" which she decided to investigate further with a chest x-ray and follow-up two weeks later. In evidence, Dr Gibb explained that she believed Mr Falanga's presentation was consistent with a post-operative effusion caused from trauma to the pleura during surgery. She added that by six weeks post surgery, she would have expected it to have resolved. (T20-22) When Dr Gibb received the chest x-ray report, she decided to arrange for a CAT scan to be done as well. This took place on 31 January and revealed a left sided pleural effusion reported as "large and loculated with free flowing component inferiorly." Dr Gibb noted in the CAT scan report that there was a small lymph node of "doubtful significance." (Exhibit C4a, T22)

- 1.5. By the 3 February 2003, Mr Falanga had complained to his general practitioner Dr Pasquale Cocchiaro, of chest pain and shortness of breath. Dr Cocchiaro referred Mr Falanga directly to Dr Gibb for earlier review. When Dr Gibb saw Mr Falanga the same day, she noted a deterioration in his condition and some shortness of breath. She decided that the effusion should be tapped to exclude an underlying malignant lesion or infection. (Exhibit C4a, T24)

2. First pleural tap – 3 February 2003

- 2.1. Dr Gibb arranged for Mr Falanga to attend outpatients at TQEH for a diagnostic pleural tap procedure to obtain a small sample of the fluid from the pleural cavity for cytological analysis. This procedure took place on 3 February and was performed uneventfully by Dr Simon Hay (RMO in the cardiology team) at an outpatient clinic, at TQEH. Dr Hay graduated in medicine from Manchester University in the United Kingdom in 1999. In February 2003, he was an RMO under-going post graduate physician training at TQEH. Dr Gibb expressed confidence in Dr Hay's ability and considered him to be an impressive RMO. (T28, T39) Dr Hay did not give evidence at the Inquest, but in January 2004, he was interviewed and asked if there were any problems encountered when he performed the first pleural tap. He answered as follows:

‘No, that was one of the most simple taps that I have done, it was just straightforward, anaesthetic, the needle went in nicely, he didn't have any pain whatsoever and got the required amount of fluid off very easily. He stayed for a couple of hours to get the post procedure checks, just to make sure he didn't have any problems, then he went home, absolutely fine.’

(Exhibit C3a)

- 2.2. Dr Hay noted that about 60 mls of blood stained fluid was obtained during the procedure and this was subsequently analysed. No malignant cells were identified. Dr Gibb explained in evidence that she recalled seeing the fluid removed, which she described as “lightly pink blood stained fluid”. (T29, T46)
- 2.3. Three days after the diagnostic pleural tap, Mr Falanga presented to Dr Cocchiaro, complaining of on-going pain on the left side of his chest and shortness of breath. Following a discussion between Dr Cocchiaro and Dr Gibb, Mr Falanga was admitted to TQEH under Dr Gibb's cardiac team in Ward 3B at 1:30pm on Thursday 6 February, for further assessment. An ECG was taken which excluded cardiac changes which might have explained Mr Falanga's symptoms. (T31)

- 2.4. When Mr Falanga was examined by Dr Hay that afternoon, he noted signs consistent with effusion following cardiac surgery. He ordered follow-up blood tests which showed a slight elevation in the urea and creatinine levels, indicating some degree of chronic renal impairment. (T34)

3. **Second pleural tap – Friday, 7 February 2003**

- 3.1. The following morning, Friday 7 February 2003, when Dr Hay reviewed Mr Falanga, he noted that his oxygen saturation levels had fallen to 88%, but was otherwise stable. In Dr Hay's view, the deterioration of his respiratory function confirmed the need to intervene by tapping the effusion. (Exhibit C5c) Sometime after 1:00 pm, Dr Hay attempted to drain the fluid from Mr Falanga's left pleural cavity. He explained what occurred as follows:

‘At the time, as I say I'd got into the space with the small needle which we put the local anaesthetic in with, and I had got fluid back quite easily, similar to the previous tap a few days before, but when I put in the larger needle which comes in the pleural aspiration pack, I was just having difficulty getting any significant amount of fluid back, and he probably had two litres of fluid in his chest, so therefore rather than continue to push in and out, you can cause bleeds, organ damage, you just stop and basically go for as I say an ultra sound guided drainage, which is usually a safer option but not always necessary because these things can be done without those measures.’

(Exhibit C3a)

- 3.2. Dr Hay explained that he had performed over twenty five of these procedures previously, but on this occasion, it was very painful for Mr Falanga. Dr Hay explained that he tapped out the area corresponding with the fluid level and introduced the needle into the intercostal space a couple of ribs lower. When introducing the needle he passed it over the rib, mindful that blood vessels known as the ‘neurovascular bundle’ normally present under the ribs may bleed if damaged. He stated that he had been taught to abandon the procedure for safety reasons after three unsuccessful attempts and this is what he did on this occasion. (Exhibit C3a)
- 3.3. Dr Gibb expressed the view in evidence that Dr Hay's actions were appropriate in the circumstances. (T41) She was not present during this procedure, but in evidence, Dr Gibb explained the reason why it might have been unsuccessful as follows:

‘I think the difficulties are patient distress which he had attempted to minimise with local anaesthetic. The other potential difficulty, a technical one with doing the tap, we know from the CAT scan that he had a loculated effusion which meant that there were pools of fluid around the pleura, and in attempting to drain a pleural effusion, you try and localise the area of fluid clinically and correlating it with the radiology, so the chest x-ray, but

sometimes this can be misleading and you can put a needle into an area that you think should have fluid in it and not be able to get any back'. (T40)

- 3.4. According to Dr Gibb, if an intercostal vessel is damaged, bleeding would be slow because the vessels are small. She claimed that at the time, she had never heard of a catastrophic bleed from a simple tap procedure. (T42)

4. **Third pleural tap with ultra-sound guidance – Friday, 7 February 2003**

- 4.1. A post procedure chest x-ray was ordered by Dr Hay in accordance with routine practice to ensure that his attempts had not produced a pneumothorax, which is a well known complication of the procedure. (T41) Having ruled this out, Dr Mosel from the Radiology Department was asked to perform an ultrasound guided drainage. This is a more precise tapping procedure with the benefit of more direct visual detail demonstrating the location of the fluid without having to rely on tapping the chest.
- 4.2. Dr Mosel was not spoken to when this matter was investigated for the Coroner and he did not give evidence at Inquest. Information about Dr Mosel's involvement is limited to the radiology report and his brief handwritten entry in Mr Falanga's case notes. According to the notes, at about 4:00 pm that afternoon, again under local anaesthesia, Dr Mosel introduced a needle "just inferior and medial to the previous puncture site" and successfully drained 660 mls of blood stained fluid. When he performed the procedure, Dr Mosel reportedly noticed a large haematoma which had developed in the area where Dr Hay had attempted his drainage earlier in the afternoon. The swelling and bruising was said to be the size of the palm of one's hand. In Dr Gibb's view, the finding of the haematoma as described, probably represents some bleeding into the subcutaneous tissues from trauma to an intercostal vessel as a result of Dr Hay's attempts to tap the effusion. I accept Dr Gibb's opinion concerning the haematoma and I find that this bruising and swelling occurred as a result of Dr Hay's unsuccessful procedure earlier that day. (Exhibit C5c)
- 4.3. According to Dr Gibb, there is insufficient detail in Dr Mosel's notes and report to indicate how heavily blood stained or otherwise, the 660 mls of fluid was which he drained during the procedure. The absence of this detail makes it difficult to determine whether there was fresh bleeding, possibly as a result of Dr Hay's procedure earlier in the day, or whether it was more of the same type of fluid taken during the diagnostic tap, three days earlier. (T46-48) The former would indicate that

Dr Hay's unsuccessful procedure may have caused fresh bleeding into the thoracic cavity, whereas the latter would suggest otherwise.

- 4.4. The medical notes disclose that Mr Falanga received two Panadeine Forte tablets for pain at 7:00 pm. His vital signs were monitored hourly after Dr Mosel's procedure until 8:45pm, at which time his blood pressure was within normal range at about 140/70. His oxygen saturation levels were 98% at 8:45pm and the puncture site was noted to be dry. The pulse rate was steady at around 66 per minute, although the rate was governed by his beta blocker medication. (Exhibit C5c)
- 4.5. Dr Hay left the hospital at the end of the day and was not due to work again until Sunday. Dr Gibb was not rostered to cover the weekend and had no involvement in Mr Falanga's management until her return to the hospital on Monday. The responsibility for Mr Falanga's management fell to the registrar, RMO and intern for the cardiology team, supervised by the consultant cardiologist on call. (T50)
- 4.6. Over the following few hours, Mr Falanga's condition deteriorated.

5. Deterioration in the early hours of Saturday, 8 February 2003

- 5.1. At 1:30 am on 8 February 2003, the evening medical intern, Dr George Chu attended Mr Falanga after he complained of chest pain in the region of the pleural tap site. Dr Chu's involvement in Mr Falanga's management is identified by reference to his entries in Mr Falanga's case notes. When Dr Chu attended Mr Falanga, he noted that his blood pressure had fallen to 100/70 and his oxygen saturations had come down to 88%. Mr Falanga was clammy and perspiring, but he was not short of breath. His pulse rate was recorded at around 60, but again, this rate was artificially controlled by his beta blocker medication and therefore was potentially misleading. (T17, T53)
- 5.2. During this assessment, Dr Chu consulted the medical registrar to discuss how to manage the situation. Dr Chu concluded that recurrent effusion or haemorrhaging into the chest were the likely causes of Mr Falanga's condition. Having reviewed the case notes, Dr Gibb explained in evidence that what Dr Chu observed was consistent with signs of significant bleeding. (T52)
- 5.3. Dr Chu instigated a strategy of intravenous fluids including Gelofusine which was a volume expanding product designed to raise blood pressure. He also ordered cross-matching of blood, in anticipation of the need for blood transfusion, a repeat chest

x-ray, ECG and blood tests to investigate the cause of Mr Falanga's deterioration. Half hourly nursing observations were commenced and Maxalon was administered for nausea. (Exhibit C5c, T54, T179) Evidence called at the Inquest suggests that this was a critical time when Mr Falanga's renal function would have been vulnerable to an episode of low blood pressure.

- 5.4. Dr Chu reviewed Mr Falanga again at 3:27 am, 5:16 am and 7:32 am and liaised with the medical registrar on each occasion. Dr Chu did not give evidence at Inquest and the identity of the relevant registrar who advised him during this time was not determined. Blood tests revealed a haemoglobin level of 110, which according to Dr Gibb, was on the low side, but was not regarded as a major problem. (T55) However, results from blood obtained at 3:27 am showed an increasing creatinine level which over the remaining period in hospital demonstrated a steady decline in renal function.
- 5.5. According to the chart of nursing observations, Mr Falanga's blood pressure dropped to 80/50 at 4:00 am and by 5:00 am had improved to 120/80. At 6:00 am the haemoglobin results from blood taken at 5:16 am indicated that the level had fallen to 88. Dr Gibb explained in evidence that because Mr Falanga was "intravascularly dehydrated", the real haemoglobin would have been even a little lower than 88. (T57)
- 5.6. After liaising with the medical registrar at 5:16 am, Dr Chu noted that there would be a concern if Mr Falanga's blood pressure dropped below a systolic reading of 80. On reflection, Dr Gibb considered that the medical registrar may have failed to account for Mr Falanga's severe co-morbidities in nominating this low reading as a level of potential concern. In Dr Gibb's view, in a person with Mr Falanga's history and medical problems, the aim should have been to maintain his blood pressure at least above 100 systolic. (T55) Dr Chu recorded Mr Falanga's blood pressure at 6:00 am as 140/70, although the nurses recorded blood pressure readings of 100/60 at 6:00 am and 7:00 am. By 8:00 am, the blood pressure dropped to 90/50. (Exhibit C5c)
- 5.7. The case notes record that at 6:00 am, Mr Falanga complained of abdominal pain and was given Panadeine Forte. Dr Chu examined his abdomen and found that it was soft and there was no rebound tenderness, which according to Dr Gibb, would have reassured Dr Chu that the abdominal pain did not represent an intra-abdominal bleed. (T56) Dr Gibb considered that Dr Chu's reviews of Mr Falanga were very thorough.

- 5.8. When the cardiology RMO took over management from Dr Chu on the morning of the 8 February, two units of transfused blood were ordered, commencing at 8:30 am. (Exhibit C5c)

6. Insertion of ‘pigtail’ chest drain – 12 noon, Saturday, 8 February

- 6.1. Professor Ruffin, from the thoracic unit was consulted during the morning and advised that a “pig tail catheter” be inserted to drain the chest. A thoracic surgeon is said to have been alerted to the situation in case urgent surgery was called for. According to the notes, at midday on 8 February, an intercostal chest tube was inserted by Dr White, or another doctor on his behalf, using ultrasound guidance. When the catheter was inserted, 600 mls of blood drained rapidly from the chest cavity. The catheter was left in place to continue draining and to enable nurses to monitor the volume of blood loss. The radiology entry in Mr Falanga’s notes requested that Mr Falanga be monitored carefully and that a repeat chest x-ray be conducted at 4:00 pm. Clearly, there was a concern about ongoing bleeding into the thoracic cavity, known as haemothorax.
- 6.2. A nursing note recorded blood pressure readings taken at 1:15 pm at between 85/55 and 106/55. The nurse had also noted that Mr Falanga was to be reviewed before being given the second unit of blood and that 1950 mls of heavily blood stained fluid had drained from the pigtail chest drain. According to Dr Gibb, the low blood pressure at around this time was suggestive of on-going hypovolaemia. (T64) During the early afternoon, senior cardiac registrar, Dr Radendran reviewed Mr Falanga and noted that he was “clinically stable” with a blood pressure of 115/55. The second unit of blood was approved for transfusion. It appears that Dr Radendran was mindful of renal function at this point. She instructed nurses to maintain a strict fluid balance chart. (T66) Nurses were also instructed to report if Mr Falanga deteriorated further by reference to his blood pressure, decreasing urine output or oxygen saturation levels.
- 6.3. At Inquest, Dr Gibb was asked to express a view about the management of Mr Falanga in her absence. In Dr Gibb’s view, the response by medical staff was appropriate as far as it went, but was not as aggressive as it should have been. She elaborated as follows:

‘At this stage I think there was no appreciation of the severity of the renal insult that I think had essentially been sustained by now, and it's often a very fine line to walk

between giving people too much fluid and not enough fluid. It's very easy for me sitting here saying "They should have given him more fluid" and in retrospect that's an easy comment to make, but this was a gentleman who had just had a coronary artery bypass, he had evidence of reduced heart function, which had been clearly documented. He had had admissions to the Queen Elizabeth Hospital with fluid overload on his lungs related to his heart function. And although I'm saying, yes, we should have been more aggressive with his fluid function, it is a very fine line, and certainly if he had not been passing a lot of urine, if you start pushing fluid intravascularly, and you are not making urine, there is nowhere for it to go, so it can lead to evidence of fluid overload, so fluid accumulating, usually within the lungs. But at this stage the medical emergency cover did make a comment that the jugular venous pressure was not elevated, and it's a difficult clinical sign to interpret, but it is looking for a pulsation of a vein in the neck and I find it easier to read if it's full than if it's low, and it tends to reflect the amount of fluid that's available within the system, and if you are approaching fluid overload, you get elevation of the jugular venous pressure, which was not - the medical emergency cover said specifically that it was not elevated. So although it's a fine line, and although I'm coming in saying "Yes, she should have given more fluid", I think was evidence clinically that there was no fluid overload, and taking the blood pressure into account I think I can say in hindsight there was evidence of under-replacement of fluid.' (T67-68)

- 6.4. Over the next twenty four hours, almost four litres of blood stained fluid drained from Mr Falanga's chest drain. He was losing more fluid than he was receiving by way of intravenous supplements and blood transfusions. Dr Chu managed Mr Falanga again overnight on 8/9 February and noted his observations comprehensively in the medical notes. He altered the intravenous fluids to reduce his elevated potassium level, which was an appropriate thing to do, according to Dr Gibb. (T70) At 2:27 am Dr Chu was called back to see Mr Falanga after it was reported that he had leapt out of bed and disconnected his chest drain. Mr Falanga's blood pressure was 90/65 and he seemed to be slightly confused. Dr Chu expressed his concern about the ongoing nature of Mr Falanga's hypotensive episode and requested a review by the cardiology team to formulate a management plan. (Exhibit C5c)

7. **Input by Renal and Cardiology Units – Sunday, 9 February 2003**

- 7.1. On the morning of 9 February, a renal unit assessment was conducted by Dr Khor who noted that Mr Falanga had passed only 50 mls of urine since 5:00 pm the previous day and that he had been incontinent at 3:00 am. Mr Falanga was in negative fluid balance and was given more intravenous fluid. Dr Khor suggested that the cardiology team consider the insertion of a urinary catheter for close monitoring of Mr Falanga's fluid balance. (Exhibit C5c)

- 7.2. Dr Hay returned to work this morning for the Cardiology Unit. He appraised himself of the events occurring in his absence and recognised that it was becoming increasingly difficult to monitor Mr Falanga's fluid balance, because of his confused state and his incontinence.
- 7.3. Throughout this period, Mr Falanga's observations were being recorded. His blood pressure remained low at between 60-90 systolic. Meanwhile, Mr Falanga's creatinine level was increasing. Cardiologist, Dr Zeitz reviewed Mr Falanga and suggested additional blood transfusion, intravenous therapy, maintenance of a fluid balance chart and repeat blood tests. Dr Hay recorded the notes of this review and concluded with the entry "should stabilise". Mr Falanga had a urinary catheter inserted at around 1:30 pm but later threatened to remove it as well as his intravenous drip. (T76) During the latter part of Sunday 9 February, the cardiology RMO made several entries in Mr Falanga's notes to indicate ongoing assessment and discussions with the renal consultant Dr Campbell, who reviewed Mr Falanga concerning a problem with the urinary catheter and his condition generally. Frusemide was administered and a repeat chest x-ray was ordered for later that evening. During this day, Mr Falanga received 4 units of transfused blood. (Exhibit C5c)

8. Monday, 10 February 2003

- 8.1. Dr Khor noted at 12:30 am on 10 February that there had been no urine output despite Mr Falanga receiving 40 mg of Frusemide three hours earlier. At 7:50 am, Dr Chu was called to see Mr Falanga after nurses reported that he had removed his intravenous drip. According to the note, Mr Falanga "flatly refused" to have a fresh drip inserted. In the afternoon of 10 February 2003, Dr Vicki Campbell from the Renal Unit assessed Mr Falanga and noted that Mr Falanga's "JVP" had increased and that there were "basal creps". She suggested that Mr Falanga needed an indwelling catheter and a central venous line. It was suggested that a transfer to the high dependency unit should be considered. (Exhibit C5c)
- 8.2. Dr Gibb returned to the ward this day and noted the events of the weekend. She considered that Mr Falanga was now haemodynamically stable, but that he had very poor urine output which was a sign of renal impairment. (T81) According to Dr Gibb, she agreed that a central venous line should be inserted which would provide for

more accurate haemodynamic monitoring. (T88) A “vascath” was inserted on Monday at about 5:00 pm and was monitored by nurses on the ward. (Exhibit C5c)

- 8.3. In evidence at Inquest, Dr Gibb said that she was disappointed that she was not notified of Mr Falanga’s deterioration over the weekend, but acknowledged that she was not “on call” over that period and didn’t usually come into the hospital on weekends. (T50-51) In hindsight, I consider that it would have been prudent for Dr Chu or the registrar to have contacted Dr Gibb, to enable her to contribute to the appropriate management of the situation. After all, it was Dr Gibb who was most familiar with the patient and was aware of Mr Falanga’s renal compromise following by-pass surgery. This knowledge would have alerted her to Mr Falanga’s particular risk of further compromise from any episode of hypovolaemia.
- 8.4. According to Dr Gibb, on 10 February, she assessed the difficulties in managing Mr Falanga on the ward, and requested transfer to the high dependency unit (HDU), but was told that no bed was available at that time. This didn’t surprise her because HDU beds were generally in demand and were used mainly for post-surgical cases. The advantage of being in this HDU according to Dr Gibb was the provision of extra nursing support and to relieve pressure on the ward nurses. (T93) However, according to Dr Gibb, the medical management in HDU would have been the same, that is, her cardiology team would have remained responsible for Mr Falanga’s management, together with input from the renal specialists. In February 2003, there was no regular supervision by intensive care specialists of patients in HDU. (T87)

9. Commencement of haemodialysis

- 9.1. Dr Gibb considered that renal dialysis may be necessary to deal with the deterioration in Mr Falanga’s renal function. The Renal Unit physicians reviewed Mr Falanga throughout this period and a decision was taken to commence haemodialysis on 12 February, via the vascath. After about fifteen minutes of this procedure, the vascath developed an obstruction which required a fresh one to be inserted. On 13 February, haemodialysis was recommenced and took place over a two hour period in Ward 3C. Meanwhile, blood draining from the pigtail catheter had reduced to the point where the cardiology physicians considered removing it, but ultimately decided not to.

- 9.2. On 14 February, Dr Campbell for the Renal Unit noted that dialysis was probably not required this day, but that the situation would be reviewed daily. Ultimately a nursing note indicates that Mr Falanga received two and a half hours of dialysis again this day on Ward 3C. Dr Gibb saw Mr Falanga the same day at which time it was noted that Mr Falanga had produced 200 mls of urine in twenty four hours. Mr Falanga experienced periods of hypotension again and showed signs of confusion, which must have been a concerning sign. (Exhibit C5c)
- 9.3. Despite ongoing input by medical practitioners from the renal unit, and cardiology unit over the following two days, Mr Falanga's renal function failed to improve. Dr Gibb summarised the situation as follows:

‘... I think this was an elderly gentleman who was recognised relatively early on to have had significant intrathoracic bleeding, he was monitored on the cardiology ward. He had regular nursing observations at a much more frequent level than is standard and these were being done half hourly over various intervals. He had frequent medical reviews by several different specialities and I think the treatment that was given was given appropriately, although, in retrospect, perhaps not aggressively enough.’ (T81)

10. Review by Intensive Care Consultant – 16 February 2003

- 10.1. On 14 February, the renal unit suggested a possible transfer to ICU or the HDU. On 16 February 2003, ICU consultant Dr Michael O’Fathartaigh reviewed Mr Falanga and formed the impression that he was still hypovolaemic. At 3:00 pm his blood pressure reading was said to be 80/40. Dr O’Fathartaigh considered the possibility that Mr Falanga may have developed pneumonia and or an empyema (a collection of pus in the thoracic cavity). Assessment of blood gases and a repeat chest x-ray was ordered to investigate these possibilities. Dr O’Fathartaigh explained that he did not think that urgent renal dialysis was necessary when he assessed Mr Falanga. (T141-3)
- In evidence, Dr O’Fathartaigh explained why he reached this view:

‘Mr Falanga was receiving dialysis treatment and I think he had dialysis on two days; the 14th, which is Friday, was the last one. The renal team were involved and obviously thinking of when he would next need dialysis and, once again, they were making a clinical judgment on when dialysis would be best indicated. In terms of immediate dialysis - I mean immediate dialysis, dialysis - at the time of us seeing him on the Sunday, the 16th, he didn't need dialysis on the criteria that his results show; that his potassium wasn't high, he didn't have a lot of acid in the body's system, he wasn't fluid overloaded, or in cardiac shock because of a fluid overload, he wasn't in acute pulmonary oedema, where fluid accumulated in the lungs, his gas exchange was really quite good. And, also, when you have dialysis, it's good to be well filled, in terms of having enough fluid on board, because, in the process of dialysis, it can upset the cardiovascular

balance. So, they were the reasons why it wasn't deemed immediately urgent to do it at that time.' (T154)

- 10.2. Dr O'Fathartaigh did not believe that a transfer to the intensive care unit was required but instead, he advised that Mr Falanga be transferred to HDU for hydration and to monitor his blood pressure in the expectation that he would improve. (Exhibit C5c, T133) In evidence, Dr O'Fathartaigh confirmed that patients in the HDU were at that time, managed by the 'home' team consultants as opposed to the ICU consultants.

11. Transfer to High Dependency Unit – 17 February 2003

After reviews by the renal unit, a decision was made for Mr Falanga to be transferred to the HDU in the early hours of 17 February 2003. Later that day, Mr Falanga was noted to be "confused and disorientated". Mr Falanga's medical notes indicate that he was not producing urine and required ongoing haemodialysis. He received three hours of dialysis via the vascath during the afternoon. Meanwhile, swabs taken routinely from the chest catheter revealed signs of infection, for which antibiotics were administered. An entry in Mr Falanga's notes suggest that a respiratory physician reviewed Mr Falanga during the afternoon and suggested that an empyema had complicated his pleural effusion and that it would require surgical drainage by a cardiothoracic surgeon. (Exhibit C5c)

12. Transfer to the Intensive Care Unit – 18 February 2003

- 12.1. On 18 February, the renal unit consultant reviewed Mr Falanga and considered that Mr Falanga's condition was deteriorating. His prognosis was now regarded as poor, and treatment was maintained at a "supportive" level with intravenous antibiotics, dialysis and "inotropes". After a review of Mr Falanga's chest x-rays the cardiothoracic surgeon decided against surgical intervention because Mr Falanga was regarded as too great a risk for surgery. Mr Falanga suffered a further episode of hypotension and was transferred to the intensive care unit with a provisional diagnosis of "septic shock" and multi-organ dysfunction.
- 12.2. At approximately 1:45 am on 19 February, Mr Falanga had repeated episodes of cardiac arrest, and was unable to be resuscitated. He was declared deceased at 2:10 am. At the time of death, doctors from the intensive care unit considered that apart from acute renal failure, one suggested cause of Mr Falanga's demise was "septic shock" due to empyema.

13. Post mortem examination

13.1. A post mortem examination was conducted by forensic pathologist Dr Allan Cala on 20 February 2003. A copy of Dr Cala's report was received into evidence. In summary, Dr Cala's examination revealed a haemothorax, but no evidence of empyema. Other findings of significance included severe native coronary atherosclerosis, scarred kidneys and old infarctions in the thalamus and cerebellum. Dr Cala noted that the coronary artery bypass grafts were patent.

13.2. When describing the 'respiratory system' findings, Dr Cala reported as follows:

'The left lung weighed 600 grams and the right lung weighed 710 grams.

There was a large amount of clotted blood and un-clotted blood in the left pleural cavity.

There was a densely adherent amount of blood clot of an undetermined amount overlying the pleura of the left lung particularly laterally and posteriorly. The parietal pleura appeared thickened particularly overlying the 3rd to 5th ribs posteriorly'.

A pleural catheter was present in the left pleural cavity, situated between the 9th and 10th ribs, however this was kinked 20mm from its distal end.

The parenchyma of the left lung appeared normal with no evidence of emphysema or other abnormality present.

There was no evidence of pneumonia or pulmonary thromboembolism in either lung.

The right lung appeared normal.

There was approximately 100 mls of blood in the right pleural cavity.'

(Exhibit C2a)

13.3. Dr Cala conducted microscopic examination of sections of the pleura and lungs which is said to have revealed the following:

'Sections of the left parietal pleura show an acute inflammation infiltrate mixed with fibrin lining the pleura. No definite organisms are seen. Deep to this is early granulation tissue consisting of fibroblasts and numerous capillary size blood vessels.

Sections of the left lung show haemorrhage overlying the pleural surface, deep to which is early granulation tissue. The adjacent lung shows collapse and areas of fibrous thickening and chronic inflammation. There is no evidence of pneumonia. Sections of the right lung show no significant abnormality.'

(Exhibit C2a)

13.4. I accept these observations made by Dr Cala at post mortem and during his microscopic examination. According to Dr Gibb, the presence of granulation tissue is suggestive of a healing response of several days duration. (T96) I take this to refer to the trauma caused by one or more of the pleural tap procedures.

13.5. Dr Cala does not make any comment in his report concerning the status of the haematoma said to have been caused by the second pleural tap procedure. Nor is

there specific reference to the source of the bleeding into the pleural cavity which ultimately led to Mr Falanga's fatal hypotensive episode.

14. Relationship between the pleural taps and haemothorax

The evidence is capable of supporting a finding that, one of the pleural tap procedures performed by Dr Hay or Dr Mosel at TQEH either initiated or exacerbated bleeding into the pleural cavity which led to hypovolaemia and ultimately death from renal failure. The initial diagnostic tap conducted by Dr Hay does not appear to have caused any trauma or bleeding and I discount this procedure as a contributing factor in Mr Falanga's demise. But because a large haematoma developed within hours of Dr Hay's second unsuccessful procedure, it is logical to conclude that blood vessels were traumatised in such a manner that there may have been bleeding into the pleural cavity as well as the subcutaneous tissue. But given the lack of clarity in the evidence about this topic, I find that the evidence is not capable of supporting any definite conclusion about the relative contribution that might have been made by the two procedures.

15. Cause of death

- 15.1. As to the direct cause of death, Dr Cala expressed the view that Mr Falanga died as a result of "complications of treatment of left pleural effusion." (Exhibit C2a)
- 15.2. Dr Gibb took issue with this description and considered that the cause of death should make reference to haemothorax, hypotension and Mr Falanga's co-morbidities. (T98, Exhibit C4c) I agree with Dr Gibb that the cause of death should reflect those factors. I find that Mr Falanga died as a result of complications of haemothorax and hypotension, following drainage of pleural effusion, post coronary by-pass surgery, in the setting of longstanding multi-organ disease, notably renal disease, related to diabetes, hypertension, hyperlipidaemia and previous smoking history. (T187)

16. Review of the medical management of Mr Falanga

- 16.1. Dr William Cobain was asked to review the management of Mr Falanga at TQEH and to provide a report for the Coroner. Dr Cobain is an experienced physician who specialises in perioperative care, which involves making assessments of patients before major surgery to determine their fitness to survive surgery. After graduating in medicine in 1980, Dr Cobain studied internal medicine and then specialised in

intensive care from 1988 until reverting back to his present field as a consultant physician at the Royal Adelaide Hospital.

- 16.2. Dr Cobain's report was received into evidence and a copy was made available for Dr Gibb to consider before she was required to give evidence at inquest. Dr Cobain also gave evidence during which he elaborated on aspects of his report. (Exhibit C8)
- 16.3. According to Dr Cobain, the pleural effusion originally observed in Mr Falanga following his surgery was not unusual and its treatment normally depended upon the presenting symptoms. (T159) As far as the unsuccessful attempted tap performed by Dr Hay is concerned, Dr Cobain indicated that he would have preferred to abandon the procedure after the second attempt, but he conceded that others would try one more time. (T168) In the circumstances, I am not prepared to criticise Dr Hay for attempting his procedure three times.
- 16.4. When commenting upon the technique used to perform the pleural taps, Dr Cobain said that he was trained to administer atropine before doing the tap, to reduce the risk of a vagal response to the procedure, which sometimes causes a patient to faint. Dr Gibb and Dr O'Fathartaigh disagreed with Dr Cobain about the need to do this. A good deal of evidence was devoted to this topic and whether some of Mr Falanga's presenting signs and symptoms in the early hours of 8 February might be attributed to a vagal reaction to the tapping procedure or a separate vagal response due to localised irritation of the pleura. Ultimately, the evidence indicated to me that it was not a relevant factor in Mr Falanga's situation and I make no further reference to it in these Findings. (T162)
- 16.5. When Dr Chu was called to assess Mr Falanga in the early hours of 8 February, Dr Cobain considered that the presentation was not only consistent with significant bleeding, but was also consistent with a heart attack, pulmonary embolus or pneumothorax, all of which had to be worked through and investigated. Dr Cobain considered that Dr Chu's assessments were thorough, but added that with the benefit of hindsight, he would have interpreted the situation with more alarm and would have considered more aggressive strategies to resuscitate Mr Falanga. He explained that by "aggressive", he meant "how aggressively one fills the patient with fluids, monitors their response in order to optimise outcome." He emphasised that this type of action is quite involved and requires highly skilled staff and that the earlier it is done, the

better the outcome generally, especially in patients like Mr Falanga who have “multiple other co-existent illnesses where their ability to withstand these stresses is less”. (T172, T176)

- 16.6. Dr Cobain expressed the view that the haematoma which developed after Dr Hay’s unsuccessful attempt to tap Mr Falanga’s effusion, may have been completely subcutaneous and was most likely reflecting an intercostal artery injury, as opposed to a vein. He was unable to say with any certainty whether at this time, there was haemorrhage into the pleural space. (T170) In his view, the blood which drained into the pleural cavity ultimately was a significant amount and that made him suspect that it was trauma to an intercostal artery, rather than a vein which was responsible. He conceded that there may have been further haemorrhage as a consequence of the ultrasound guided pleural tap. (T171)
- 16.7. Dr Cobain indicated in his report that by about 10:30 pm on 8 February, when Dr Chu reviewed Mr Falanga, a more aggressive approach to his management was required and it needed to be carried out hastily in an intensive care setting. He summarised his opinion as follows:
- ‘In my opinion, in view of his pre-morbid conditions, the hypotension and increasing oliguric renal failure in the setting of a likely bleed after one of the pleural taps, all of the above would justify a consultation request for management in a High Dependency Unit setting where appropriate detailed cardiovascular evaluation and resuscitation could be initiated and all relevant therapeutic options considered rapidly.’
(Exhibit C8)
- 16.8. Dr Cobain stated that to maximise Mr Falanga’s chances of recovery, his hypovolaemia needed to be managed as carefully as possible. In his view:
- ‘Vigorous haemodynamic resuscitation at that point with appropriate central lines and or pulmonary artery catheters to optimise cardiac filling pressures and output could reasonably have been requested.’
(Exhibit C8)
- 16.9. Dr Cobain explained that it was the hypovolaemia resulting from the bleeding which led to his hypotension which in turn “caused his pre-existing mild renal impairment to deteriorate significantly to the point where dialysis was required”. (Exhibit C8) In evidence, he stated that the doctors did a very good job but because Mr Falanga was not in a suitable environment, their actions were not carried out quickly enough. Dr Cobain emphasised that his main concern was with the developments in the early

hours of 8 February which was the period where important decisions needed to be made. He elaborated on the challenges for junior doctors such as Dr Chu as follows:

‘Ideally, the more junior doctors on call overnight faced with such a patient need to realise that the patient may be sufficiently unwell that they are not confident in managing the patient. They would often then immediately refer to their more senior registrar who is on call and who is often able to make such decisions that are appropriate. If such a senior registrar sees the patient and feels that they also are unable to guide management confidently, then ideally they should be able to access the on-call clinician responsible for the patient’s care.’ (T178)

16.10. Dr Gibb agreed with the essential features of Dr Cobain’s opinion, but considered that by 10 February, a transfer to HDU was probably too late to have made any difference to the outcome. (T99) Dr Gibb explained that she believed the major damage had occurred over the weekend and that Mr Falanga’s renal function never recovered at all after that. (T95) She said that she would have wanted an intensive care unit (ICU) consult during that weekend because ‘they’ may have recognised the potential renal damage resulting from hypovolaemia and treated him more aggressively with the assistance of an intravenous central line. Dr Gibb explained how this might have helped as follows:

‘If he had had a central line inserted, for instance, it would have provided more immediate monitoring of his filling pressures, his state of volume hydration, and that may have been able to guide the treating team more appropriately in terms of providing adequate volume expansion without going the other way and causing volume overload, which may have precipitated fluid accumulation in the lungs’ (T88)

16.11. According to Dr Gibb, she expected that the cardiologist ‘on call’ over the weekend would recognise the need for an ICU consult. Dr Zeitz was not interviewed during the course of the Coroner’s investigation and he did not give evidence at Inquest. I am not prepared to criticise Dr Zeitz for decisions taken concerning Mr Falanga’s management without providing him with an opportunity of explaining the nature of his involvement and what factors influenced his clinical judgment at the relevant time. As Dr Gibb explained in evidence, balancing the relative risks involved is “a very fine line to walk” and is made much easier with the benefit of hindsight. (T67)

16.12. Dr Cobain acknowledged that if TQEH high dependency unit at the time, was not managed by intensive care specialists, then it would not have provided the type of monitoring which he believed was called for in this case. (T181) He also acknowledged that even if Mr Falanga had been cared for in an intensive care facility as suggested, because of his other pre-morbid medical problems, the outcome may

have been no different. I accept Dr Cobain's view that if Mr Falanga had been managed in an intensive care unit, it may not have altered the outcome. I also find that if Mr Falanga had been managed in the HDU setting as it then existed at TQEH, the outcome may not have been any different.

- 16.13. As far as access to ICU is concerned, Dr Cobain emphasised that it was necessary for a clinician or the registrar to be very direct about whether they are having difficulty managing a patient and if the patient cannot be accommodated appropriately in the hospital, then in rare cases, they need to consider transfer to another hospital. (T183)
- 16.14. Mr Falanga's medical notes reveal that the medical and nursing staff at TQEH invested a significant amount of effort in trying to manage the complexity of Mr Falanga's condition. After reflecting on the available evidence, I find that notwithstanding the collective effort in managing the situation, someone should have agitated for an ICU assessment during the early phase of Mr Falanga's deterioration. I endorse the views expressed by Drs Gibb and Cobain that the management during the weekend of 8 and 9 February should have been more aggressive than it was.
- 16.15. The consultant on call for the cardiology team was ultimately the person responsible for Mr Falanga's management and for ensuring that he was being managed in an appropriate manner and in a setting which was suited to the circumstances. There is no indication in the notes that the registrar who Dr Chu was receiving advice from, attended upon Mr Falanga in person during the early hours of 8 February 2003. I consider that it would have been better to have someone more senior than Dr Chu assess Mr Falanga. A more senior doctor may have recognised that the situation justified an attempt to organise a transfer to ICU. It appears unreasonable to expect the staff on the ward to cope with the ongoing demands which Mr Falanga's condition generated. Of course it would have depended upon the availability of beds in ICU at the time. At the end of the day, it comes down to the exercise of clinical judgment, made much more confidently in hindsight. I find that whilst a more aggressive approach may not have altered the outcome, it would have maximised Mr Falanga's chances of recovery.

17. Changes to High Dependency Unit

- 17.1. Dr O'Fathartaigh explained that since 2005, there have been changes at TQEH to the operation of the HDU. Patients housed in HDU are now reviewed daily by a member

of the Intensive Care Unit, under the supervision of the ICU consultant seven days a week. The cover does not extend to overnight, at which time problems are directed from the home team doctors to the intensive care unit registrar. These changes were introduced according to Dr O’Fathartaigh, because there was a growing awareness of the need to provide greater monitoring for increasing numbers of ill people in hospital. (T127) The improvements made to the HDU at TQEH are sensible and hopefully will provide ward staff with more options for future management of challenging patients such as Mr Falanga.

Key Words: Haemothorax; Hospital treatment;

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

Seal the 19th day of May, 2006.

Coroner