

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 25th and 26th days of October 2004 and the 19th day of November 2004, before Wayne Cromwell Chivell, a Coroner for the said State, concerning the death of Ross Dudley Peddey.

I, the said Coroner, find that Ross Dudley Peddey aged 63 years, late of 2 Weroona Avenue, Park Holme, South Australia died at the Flinders Medical Centre, Bedford Park, South Australia on the 3rd day of July 2001 as a result of pneumonia complicating anoxic brain injury, due to post-operative haemorrhage as a result of damage to the inferior epigastric artery. I find that the circumstances of his death were as follows:

1. Introduction

- 1.1. Mr Ross Peddey was a 63 year-old man who was admitted to the Flinders Medical Centre in January 2001 with a diagnosis of a diverticular abscess of the sigmoid colon. This was surgically repaired by the performance of a sigmoid colectomy with an end to end anastomosis (joining), with a covering loop ileostomy to protect the anastomosis. This surgery proceeded without incident.
- 1.2. On 13 May 2001, Mr Peddey was readmitted to the Flinders Medical Centre for a procedure to close the ileostomy and restore intestinal continuity through the sigmoid colon. Because Mr Peddey had a prosthetic mitral valve in his heart, he had been receiving Warfarin therapy. This was suspended during the surgery to reduce the risk of haemorrhage. The operation was performed on 14 May 2001, and again there were no apparent complications.

- 1.3. In the early morning of 16 May 2001 Mr Peddey's condition deteriorated. He became sweaty and short of breath, his blood pressure had dropped and his pulse rate had increased. Investigations by the medical staff proceeded during the morning including a CT pulmonary angiogram to exclude the possibility of a pulmonary embolism. At about 12:25am, Mr Peddey suffered a cardiac arrest. A pulmonary embolism was excluded by the CT scan.
- 1.4. By this time it was realised that Mr Peddey had probably suffered a post-operative haemorrhage. Cardio-pulmonary resuscitation was commenced and Mr Peddey was transferred to the Intensive Care Unit where resuscitation continued.
- 1.5. Once the anti-clotting effects of Warfarin had been reversed by the administration of fresh frozen plasma, an emergency laparotomy was performed and it was found that Mr Peddey had suffered a major haemorrhage from an artery in the vicinity of the anastomotic site. The clots and old blood were evacuated, adhesions were divided, the sigmoid colon was resected and rejoined, the artery was oversewn and Mr Peddey was transferred to the Intensive Care Unit.
- 1.6. Unfortunately, Mr Peddey's condition failed to improve. He remained comatose on a ventilator for a period of four weeks until he was transferred to a surgical ward, where palliative care was administered with the consent of his family. His neurological condition gradually worsened and he eventually died on 3 July 2001.

2. Cause of death

- 2.1. Because Mr Peddey's death was not initially reported to the Coroner's Office, no post-mortem examination of his body was performed.
- 2.2. I have received a report from Dr Robert Britten-Jones, Emeritus Consultant Surgeon of the Royal Adelaide Hospital and a person of vast experience in the area of abdominal surgery. In Dr Britten-Jones' opinion, Mr Peddey died as a result of an intra-abdominal haemorrhage caused by damage to the inferior epigastric artery during the surgery to close the ileostomy on 14 May 2001. Dr Richard Sarre, the Director of the Colorectal Unit at Flinders Medical Centre, agreed with this (T153).

- 2.3. I accept Dr Britten-Jones' evidence, and find that Mr Peddey died as a result of pneumonia complicating anoxic brain injury, due to post-operative haemorrhage as a result of damage to the inferior epigastric artery..

3. **Background**

- 3.1. Mr Peddey was a 63 year-old man. When he was reviewed for surgery on 7 May 2001, he weighed 92.3 kilograms, he had a good exercise tolerance, was a non-smoker and light drinker. His medication included Warfarin (an anti-coagulant), Lipex (a cholesterol lowering drug), Aurorix (an anti-depressant), Lasix (a diuretic), and Slow-K (a potassium supplement).

- 3.2. In December 1995, Mr Peddey was diagnosed with sub-acute bacterial endocarditis with mitral valve regurgitation and diffusely diseased right coronary artery. In January 1996 he was admitted to the Royal Adelaide Hospital, where surgery was performed and the diseased mitral valve was replaced with a synthetic valve, and a coronary artery bypass vein graft was performed. Warfarin was prescribed on discharge. Dr Britten-Jones commented:

'In any patient who has had a prosthetic heart valve inserted, such as Mr Peddey, it is essential that an anti-clotting agent is administered on a daily basis for life – otherwise blood clots may form on the foreign body (the prosthetic valve). If subsequent surgery becomes necessary, the long acting anti-clotting agent (warfarin) must be ceased three days before operation, and the short acting heparin substituted. This in turn is ceased four to six hours before the surgery and recommenced immediately afterwards and continued until warfarin again becomes effective (usually in 24-48 hours).'

(Exhibit C8, p2)

- 3.3. As I have already outlined, in January 2001 Mr Peddey was diagnosed with a diverticular abscess of the sigmoid colon which was repaired with an end to end anastomosis and covering loop ileostomy. The operation was performed by Dr Richard Sarre, Director of the Colorectal Unit at Flinders Medical Centre. Mr Peddey recovered well from this operation without complications.
- 3.4. On 13 May 2001 Mr Peddey was readmitted to the Flinders Medical Centre. Warfarin had been ceased two days earlier and short-acting Heparin was administered by intravenous infusion overnight and ceased at 0500 hours on 14 May 2001.

- 3.5. The surgery commenced at 1313 hours. It was performed by the Senior Registrar, Dr Leong. Again, it proceeded normally without apparent problems. The surgical record indicates that the operation was completed at 1335 hours, and Mr Peddey returned to the ward at 1635 hours.
- 3.6. The Heparin infusion was recommenced at 1700, and long-acting Warfarin was commenced at 1900 hours.
- 3.7. Mr Peddey's condition remained stable during 15 May 2001, although it was noted that he had a 'dizzy spell' while showering in the morning.
- 3.8. On 16 May 2001 at 0200 hours Mr Peddey's blood pressure was recorded at 120/70 with a pulse of 85. At approximately 0500 hours, Registered Nurse (RN) Asha Charlton found Mr Peddey having what she described as a 'fainting spell'. She said:

'At approximately 0500 - 0530 hours that morning we found him sitting on the edge of his bed, just with a blank stare. We popped him back into bed and he said he felt faint. He stated he was getting up to go to the toilet. A bottle was nearby. His observations were; blood pressure 95 on 60 which is a little bit low but relevant with a bit of a fainting spell. He didn't faint but he felt faint, he was sitting on the edge of the bed by himself and he was a bit tachycardic but he was quite anxious about feeling a little bit faint. But on checking on him again a little bit later he was feeling fine once we popped him back into bed. He did state to me that the same thing happened the day before.

I didn't make any notes of this incident in the case notes. I normally would write them in. I must of forgotten.

...

In reference to the drop in his blood pressure, because he had a dizzy spell I wasn't hugely concerned, it was a little bit low but nothing as I related it to the dizzy spell.'

(Exhibit C4, pp1-2)

- 3.9. Dr Britten-Jones disagreed. He said:

'I disagree that it can be assumed that the low blood pressure was due to a fainting attack. The recording of 95/60 is significantly low for Mr Peddey whose blood pressure was recorded the previous day on three occasions as between 130 and 140/80 and at 0200 hours on 16 May it was recorded at 120/70. Also, after a faint, once the patient is placed flat the blood pressure returns rapidly to normal. Also, the RN recorded the pulse at 115 per minute. It had been 85 at 0200 hours. Normally with a faint the pulse is slow.'

(Exhibit C8, pp2-3)

RN Charlton conceded that in retrospect it would have been appropriate if she had checked Mr Peddey's blood pressure again after he settled (T12).

3.10. Between 0800 and 0830 hours, Mr Peddey was seen by the Colorectal team comprising Dr Kate Stephens, a first-year Resident Medical Officer, Dr Kang, an Intern, and Dr Darren Tonkin, an Advanced General Surgical Trainee. Dr Stephens noted in the clinical record:

'Patient seen by Tonkin. Patient went to toilet, sweaty and short of breath. No pain, no calf tenderness. At 0550 hrs blood pressure 95/60, pulse rate 115, and oxygen saturation 98% on two litres of oxygen. Temperature 36C. Diagnosis – hypotensive.'

(Exhibit C3)

3.11. Dr Tonkin said that his concern at that stage was whether Mr Peddey was suffering a pulmonary embolism. He explained:

'A. At the initial time when I saw him my - the top of my list of problems was the pulmonary embolus because of the shortness of breath predominantly and also the low oxygen saturations ... those things all point to that. He was also a bit hypotensive which can sometimes go with a pulmonary embolus but I was also concerned that there may be some bleeding. So we tried to organise the blood tests and venous access and replacement fluids to counter that and to try and differentiate between those two we organised an urgent CAT scan to differentiate.

Q. Did you consider abdominal haemorrhaging at the time.

A. I initially thought of that but on examination his abdomen was non-tender, he was not very distended and he wasn't complaining of any pain and so while it was in the back of my mind it wasn't the main sort of diagnosis that I was concerned with.'

(Exhibit C6, p7)

3.12. Dr Tonkin requested an urgent CT pulmonary angiogram, and intravenous access to enable fluid resuscitation to stabilise Mr Peddey's blood pressure and to obtain blood samples. He said he left these tasks with Dr Kang, the Resident Medical Officer. He was sure that Dr Kang understood the urgency of the situation (T77). He then left for an Outpatients Clinic. Dr Kang is now overseas, so I did not have the benefit of his evidence on these matters.

3.13. Mr Peddey's blood pressure remained low throughout the morning (88/56 with a pulse of 113 at 0930 hours, 110/65 with a pulse of 116 at 1000 hours, 105/76 with a pulse of 126 at 1100 hours).

3.14. The surgical staff were unable to achieve intravenous access, no doubt due to Mr Peddey's low blood pressure, and so an Intensive Care Unit specialist was called. A central venous line was eventually inserted at 1100 hours. Haemaccel (a plasma

expander) was commenced and continued over a two hour period. A blood sample was forwarded to pathology for analysis.

- 3.15. Dr Kang arranged for Mr Peddey to be transferred for the performance of a CT pulmonary angiogram to exclude the pulmonary embolus.
- 3.16. Dr Tonkin was contacted at about midday and advised of the results of the blood investigations, which showed that Mr Peddey's haemoglobin level had fallen from 130 to 75.
- 3.17. Dr Tonkin said that he contacted Dr Sarre and advised him that, on the basis of the haemoglobin readings, he thought that Mr Peddey was bleeding into his abdomen and that he proposed to take Mr Peddey straight from the CT scan to the operating theatre. Dr Sarre agreed that this was appropriate, and began making his way from his rooms to the hospital. Dr Tonkin went down to the CT scan area. As Mr Peddey was being taken from the CT scan bed he suffered a cardiac arrest (Exhibit C6, p9)
- 3.18. As I have already mentioned, the CT pulmonary angiogram, performed at 1215 hours, showed no evidence of pulmonary embolus, but it did disclose extensive fluid within the peritoneal cavity. The Radiologist commented:

'The appearances are in keeping with extensive intra-abdominal haemorrhage.'

(Exhibit C3)
- 3.19. The emergency team was called to the CT area at 1227 hours. On arrival, Mr Peddey had no cardiac output. Cardio-pulmonary resuscitation was commenced and his heart went into ventricular fibrillation. Normal cardiac rhythm was restored after 10 to 15 minutes by electric defibrillation. Mr Peddey was transferred to the Intensive Care Unit where resuscitation continued. Fresh frozen plasma was administered to block the anti-coagulation effect of Warfarin and reduce the INR level to normal limits before surgery could be undertaken.
- 3.20. At 1320 hours, emergency surgery was performed by Dr Kapoor, the Senior Registrar, with Dr Tonkin assisting. Dr Sarre was present in the theatre. At surgery, a large amount of blood and blood clots were found in the abdominal cavity. There was a haematoma around the join in the sigmoid colon. There was bleeding from an artery in the rectus muscle. There was no evidence of leakage of stomach contents. The anastomosis was resected and the ends rejoined. The artery was oversewn, the

abdominal cavity was washed out. By this time the bleeding had been controlled so Mr Peddey's abdomen was closed and he returned to the Intensive Care Unit (Exhibit C6, pp14-15).

- 3.21. As I have already outlined, Mr Peddey failed to recover from this incident, and after he was transferred from the Intensive Care Unit to the Surgical Ward and received palliative care, he eventually died on 3 July 2001.

4. Issues arising at inquest

- 4.1. Dr Britten-Jones explained the causation of Mr Peddey's haemorrhage as follows:

'In the performance of a closure of an ileostomy it is important to avoid damaging the inferior epigastric artery, which runs behind the rectus muscle and may run quite close to the edge of the rectus, which is the usual site for an ileostomy. It is very likely that this vessel was damaged at the operation to close the ileostomy on 14 May 2001, but was unrecognised at the time ... Normal clotting mechanisms would have sealed the damaged vessel, as Mr Peddey's anticoagulation therapy (warfarin) had been adequately reversed, as indicated by the INR level within normal limits immediately prior to surgery, and the heparin infusion ceased six and a half hours beforehand.

It is very likely the Mr Peddey bled in the early hours of 16 May 2001, when the anticoagulation therapy would have been fully effective, as Mr Peddey had received two 5mg doses of warfarin on successive nights after the initial operation and the heparin infusion was in place. In fact, at 1115 hrs on 16 May 2001 the INR level was recorded as 3.4, a level at which the ability of the blood to clot is greatly diminished, and it can be assumed that at 0550 hrs the level would still have been within the therapeutic range.'

(Exhibit C8, pp5-6)

- 4.2. Having regard to Mr Peddey's symptoms, Dr Britten-Jones acknowledged that there were a number of differential diagnoses available. He said:

'It would seem, then, that Mr Peddey remained with low blood pressure and rapid pulse for six hours from 0500 hrs to 1100 hrs. Mr Peddey had no complaint of abdominal or chest pain during the morning, and Dr Tonkin states that the abdomen was slightly distended and tympanitic, with bowel sounds present. The possible diagnoses to consider at Dr Tonkin's assessment would be myocardial infarct, septicaemia, pulmonary embolus and internal haemorrhage. However, the urgent necessity was resuscitation of a hypotensive patient. Unfortunately, due to circumstances of which I am unaware, no such resuscitation would appear to have been commenced until 1100 hrs. Certainly mitigating circumstances were the fact that it was impossible to insert a peripheral drip line, which I am sure Dr Tonkin would have done if possible; also, the fact that the abnormal EGG report at 0900 hrs indicated the possibility of myocardial infarct, may have misled him.

...

The four possible diagnoses mentioned above were doubtless considered by Dr Tonkin. An electrocardiogram was ordered and was reported as showing a T-wave abnormality with possible lateral ischaemia. Septicaemia was unlikely as the temperature was sub-normal, although it had been elevated the evening before. Also, there were no signs of an infective focus for sepsis. A pulmonary embolus was highly unlikely with a patient on warfarin therapy, and also a heparin infusion running until 0700 hrs on the morning of 16 May 2001. It is easy to be wise in retrospect, but it would appear to have been unwise of Dr Tonkin to order a pulmonary angiogram to exclude a pulmonary embolus. It was not performed until 1215 hrs, and it was during this examination that the patient experienced the cardiac arrest. As stated, the likelihood of pulmonary embolus was very low and the classic signs of haemorrhage were present, but I can appreciate the diagnostic dilemma in a junior surgical registrar faced with this problem. I think that a more experienced senior clinician, faced with the situation between 0800 and 0900 hrs, would have diagnosed internal abdominal haemorrhage, knowing that injury of the inferior epigastric artery is a known hazard of the operation of creating or closing an ileostomy, or, if unsure, at least have ordered an urgent abdominal ultrasound, a rapid and relatively simple investigation for confirming free fluid in the peritoneal cavity.'

(Exhibit C8, pp6-7)

- 4.3. In oral evidence, Dr Britten-Jones acknowledged that the tests ordered by Dr Tonkin, namely an electro-cardiograph and blood investigations including haemoglobin and coagulation studies, were appropriate in view of the above list of differential diagnoses (T178). If the results of these tests had been obtained in a timely fashion, the clinicians would have been alerted at an early stage that Mr Peddey's problem was not a pulmonary embolus, but internal haemorrhage.
- 4.4. Dr Tonkin said that he left Mr Peddey to attend an Outpatients Clinic at between 0900 and 0930 hours (T76). By this time neither he nor the two interns could obtain intravenous access. Clearly Mr Peddey's low blood pressure had led to collapse of his peripheral veins. It was left to Dr Kang to arrange the blood investigations, but before he could do so he needed to obtain a blood sample. Being unable to obtain intravenous access, he needed to contact the Intensive Care Unit and ask for expert help to gain intravenous access. This was not achieved until 1100 hours. The blood was then transferred to the Pathology Department, and the results were not communicated to Dr Tonkin until some time after 1215 hours. Shortly after that Mr Peddey suffered a cardiac arrest.
- 4.5. Dr Britten-Jones said that this delay was critical in the causation of Mr Peddey's death. Had Mr Peddey's haemoglobin levels been available to the clinicians at an earlier stage, urgent operative intervention could have taken place before he lost so

much blood, and before his cardiac arrest, and the outcome might have been very different (T170).

- 4.6. RN Charlton acknowledged that her failure to record Mr Peddey's 'dizzy spell' in the clinical record at 0530 hours, her failure to obtain a follow-up blood pressure reading after he settled back in bed and her failure to record in the clinical record Mr Peddey's complaints of pain which necessitated the dose of Morphine at 0430 hours were all errors which, in retrospect, should not have occurred.
- 4.7. That being said, I accept that although Mr Peddey's blood pressure had dropped significantly from a systolic reading of 130 to 95, his condition was not so critical at that stage that RN Charlton should have recognised an emergency.
- 4.8. I note that a protocol for staff at Flinders Medical Centre has been developed since Mr Peddey's death which establishes criteria upon which a Medical Emergency Team (MET) should be called. One of those criteria is a drop in blood pressure below 90 systolic. Mr Peddey's blood pressure had not dropped to that extent while RN Charlton was responsible for his care.
- 4.9. By the time Dr Tonkin was in charge of Mr Peddey's care, however, his blood pressure had dropped to 88/56 at 0900 hours, and 80/50 at 0930 hours. This condition did call for the intervention of the MET and the protocol required that the Intensive Critical Care Unit should have been called. The protocol reads in part:

'In recent years the cardiac arrest protocols worldwide have been under scrutiny to assess their effectiveness. Survival post in-hospital cardiac arrest has remained static for the last 30 years between 14-16%. Up to 80% of these were preceded by clinical instability, which had it been recognized and appropriately treated; the cardiac arrest may have been avoided.

Traditionally the cardiac arrest team was only called once the patient had arrested and if the patient hadn't arrested then the person who called the team was asked to justify their decision, reinforcing the late decision to call the emergency team.

In 1990 Professor Ken Hillman (Liverpool Hospital NSW) recognized this problem and started the MET (Medical Emergency Team). He developed new standardized calling criteria for the emergency team that could be used by anyone throughout the hospital. The aim was to provide early assessment and treatment of patients who are seriously ill and at risk of cardiopulmonary arrest thus improving their outcome. The results in hospitals that have changed over to the MET system has been dramatic, with a marked reduction in the morbidity (reduced ICU/HDU admissions and overall length of stay in hospital) and mortality (up to 50-65% in some institutions) associated with this group of patients.'

(Exhibit C4a)

- 4.10. It is now impossible to know why there was so much delay in obtaining intravenous access, and conveying the results of the blood investigations to Dr Tonkin. Dr Kang has returned to Malaysia so I have no information as to what difficulties he may have experienced that morning. He made no notes in the clinical record of his activities during the morning which, in my view, is highly unsatisfactory. In particular, I have no evidence that he knew of Mr Peddey's very low blood pressure at 0900 and 0930 hours, and why it does not appear that he took any urgent action about that. Dr Sarre agreed that it would have been 'more appropriate' if the test results had been received and acted upon earlier (T113).
- 4.11. Further, there is no information in the clinical record as to the identity of the person from the Intensive Care Unit who eventually inserted a central venous line. It has therefore not been possible to ascertain whether there were any delays in the attendance of that person.
- 4.12. Dr Britten-Jones said that if Dr Kang had encountered difficulties in obtaining someone from the Intensive Care Unit, there must have been other people in the Flinders Medical Centre that morning who could have assisted him to obtain venous access. Someone from the Anaesthetics Department or Radiology may have been able to assist (T180).
- 4.13. Dr Tonkin told me that he appreciated that it was urgent to obtain the information he was seeking (T61). Dr Stephens told me that she also appreciated the urgency of the situation (T46). If Dr Kang had appreciated the urgency of the situation, it is inconceivable that he could not have obtained assistance earlier than he did.
- 4.14. Dr Tonkin, however, left Mr Peddey in the care of Dr Kang while he went off to the Outpatients Clinic. He did not hear again from Dr Kang throughout the morning. He explained that he expected that if Dr Kang was having difficulty, or if the tests conveyed worrying information, he would have heard from him. He said:

'There's delegation of tasks that has to happen, and I felt confident that Dr Kang understood that there was an urgency to arranging the tests and to resuscitate Mr Peddey. I had no reason to believe that he would neglect that, or that there should be a problem. In my mind I had the idea that on the top of my list was a pulmonary embolus. I can't specifically remember what I thought, but I would assume that I guess the old adage that 'No news is good news' would apply, and that normally as I said the interns in the lab don't tend to call you with normal results, otherwise you would be on the phone all day.'
(T77)

Dr Sarre supported this point of view (T115).

4.15. In fact, Dr Tonkin did not hear from Dr Kang for upwards of three hours. In my opinion, appreciating that Mr Peddey's condition was urgent, indeed critical, Dr Tonkin should have checked Mr Peddey's condition at a much earlier time. It was not appropriate to assume that 'no news was good news' when he left the care of a critically ill patient to a first-year Resident Medical Officer. As Dr Britten-Jones said:

'A. The onus would lie with Dr Tonkin in my opinion, because he had a patient who was very sick, very sick and if a doctor has such a patient, the doctor in charge as he appeared to be at that time, it's his responsibility in my opinion to ensure that everything is being done correctly and rapidly.

Q. With all due respect that's not really practical in a hospital setting, that's an ideal situation isn't it. I mean certainly if he'd remained on the ward that might be appropriate.

A. No, I wouldn't agree with that. I think in any hospital setting it should be, maybe it's not, but it should be and in my experience it usually has been that the person responsible for a very sick patient has to take the responsibility seriously and follow it up –

Q. How do they do that if they're in another part of the hospital where they've also got patients they're dealing with.

A. Yes, I realise all those difficulties and I can appreciate them, but nevertheless it is perhaps a slightly mitigating circumstance, but only slightly mitigating in my opinion. ' (T179-T-180)

4.16. It would be easy and simplistic to visit responsibility for this debacle on Dr Kang, who is not here to defend himself. However, it must be remembered that Dr Kang was a first-year Resident Medical Officer with little surgical training. He was being supervised by Dr Tonkin whose responsibility it was to care for the patient. Dr Kang's responsibility was merely to act on Dr Tonkin's directions. I agree with Dr Britten-Jones' evidence that responsibility for Mr Peddey's treatment rested with Dr Tonkin and not with Dr Kang.

4.17. Dr Sarre said he agreed with Dr Britten-Jones' overall assessment (T122). He pointed out that each of the consultants, including himself, was a visitor to the hospital, and had other commitments. He said that there is no standing order or protocol by which registrars report cases to consultants. He said:

'No, there's nothing in the form of a written protocol and indeed the level of supervision of the registrars depends on their experience, their ability and the nature of the cases involved with. So there is no standing order for notification under particular circumstances or events.' (T120)

He said the degree of supervision also varied from consultant to consultant, and that of the four in the Colorectal Unit at Flinders Medical Centre, two of them are more intense managers. The less intense ones, including him, are more inclined to rely on the judgment of the registrar (T145).

- 4.18. Dr Sarre said he was still unable to explain the delay between 8:30am and 11:00am when a blood sample was obtained. He said:

'I still don't understand why or how that result wasn't available a lot sooner. The difficulty was getting intravenous access, even a needle in to get a blood test. Now it's easy in retrospect to say well the reason for that is the blood vessels are all collapsed because he had such a small blood volume going, but that wasn't entirely clear at the time. The next step if you are having difficulty yourself is to summon the intensivists, the intensive care people to do it. That's what they did, but as you are aware there was a delay.' (T129)

5. **Conclusions**

- 5.1. It is a matter of great concern that Mr Peddey had been showing symptoms of serious illness since 0530 hours on 16 May 2001 and obvious signs of collapse and hypovolemic shock since about 0830 hours that day. The fact that he did not receive effective treatment until after 1100 hours, and in particular fresh frozen plasma to remove the anti-coagulant effects of Warfarin and Heparin until after his cardiac arrest at 1227 hours, is deplorable.
- 5.2. In the absence of Dr Kang and evidence from the Intensive Care Unit staff member who gained intravenous access at 1100 hours, it has not been possible to ascertain why this significant lapse in appropriate standards of treatment has occurred. I note that Dr Sarre has not, to this day, been able to establish why these delays occurred.
- 5.3. It is simplistic to blame Dr Kang, or 'the system', as counsel for Flinders Medical Centre sought to do.
- 5.4. It is incumbent upon Dr Sarre, and Flinders Medical Centre generally, to establish why such a substantial lapse has occurred, and to take steps to ensure that it will not happen again. At this stage, I cannot be sure that this has occurred.

6. **Recommendations**

6.1. I am empowered pursuant to Section 25(2) of the Coroner's Act 1975 to make recommendations which might, in my opinion, 'prevent, or reduce the likelihood of, a recurrence of an event similar to the event that was the subject of the inquest'.

6.2. I recommend as follows:

- The Minister of Health and the Chief Executive Officer of Flinders Medical Centre should address the following issues arising from Mr Peddey's case:
 - Whether Dr Kang experienced undue delays when he sought the assistance of a clinician from the Intensive Care Unit so that intravenous access could be obtained, and for what reason;
 - Whether Dr Kang experienced undue delays in obtaining a CT pulmonary angiogram (until 1215 hours) and for what reason;
 - Any other difficulties which junior and middle ranking medical staff at Flinders Medical Centre might be experiencing in obtaining appropriate treatment for patients in need of urgent care.

Key Words: Hospital Treatment; Intra-Operative Haemorrhage

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

Seal the 18th day of November, 2004.

Coroner