CORONERS ACT, 2003

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 13th, 15th, 16th, 17th, and 27th days of September 2010, ther 23rd day of December 2010, the 4th day of February 2011, the 7th day of March 2011 and the 22nd day of June 2011, by the Coroner's Court of the said State, constituted of Anthony Ernest Schapel, Deputy State Coroner, into the death of Ian Trengove.

The said Court finds that Ian Trengove aged 81 years, late of Unit 1, 386 Carrington Street, Adelaide, South Australia died at St Andrews Hospital, South Terrace, Adelaide, South Australia on the 30th day of March 2008 as a result of retropubic and retroperitoneal haemorrhage complicating pelvic fractures with contributing warfarin anticoagulation and ischaemic and hypertensive heart disease. The said Court finds that the circumstances of his death were as follows:

1. Introduction and reason for Inquest

1.1. Mr Ian Trengove was 81 years of age when he died as a patient at the St Andrews Hospital (St Andrews) in Adelaide on 30 March 2008. He had been admitted to St Andrews during the previous afternoon.

1.2. Mr Trengove appears to have been a reasonably active man in spite of an extensive medical history. He was performing the duties of a verger in St John's Anglican Church in the city when he suffered an accidental fall and experienced pain in his right hip. Staff there had been preparing for a wedding when Mr Trengove evidently missed a step. He was unable to weight bear and as things were to transpire he would later that day at St Andrews be diagnosed by way of an X-ray as having sustained a minimally displaced fracture or fractures of the pelvis.

1.3. Mr Trengove's past medical history included complete heart block with subsequent pacemaker insertion in 1981, hypertension, congestive cardiac failure, ischaemic heart disease with previous coronary artery stenting, atrial fibrillation, insertion of a pacemaker/defibrillator in 2006 and a transurethral resection of the prostate gland (TURP). Mr Trengove's condition of atrial fibrillation meant that he was placed on the anticoagulant drug, warfarin. Atrial fibrillation can give rise to the formation of blood clots which can then block important blood vessels and can cause among other things a stroke. Warfarin is an orally taken anticoagulant drug which reduces the tendency of the blood to clot. The patient takes warfarin on a daily basis. The anticoagulation level in a person's blood due to warfarinisation can be measured by a blood test. The result of the blood test is known as the patient's INR. A normal INR without any anticoagulant intervention is 1. A therapeutic INR for the condition of atrial fibrillation lies usually between 2 and 3 and this was regarded as Mr Trengove's therapeutic level. In order to maintain a person's INR within the therapeutic level, regular monitoring by way of a blood test is maintained and dosages of warfarin may need to be withheld or adjusted from time to time. Mr Trengove was on a prescription of 0.5mg warfarin per day.

1.4. Owing to the fact that warfarinisation reduces the tendency of blood to clot, a person who is taking warfarin may experience heavier than normal or excessive bleeding in the event of a traumatic injury that compromises the vascular system. Indeed, the bleeding, or haemorrhaging, may be uncontrollable to the point where the consequent blood loss presents as a significant danger to the person's life or wellbeing. Clearly, in such a situation the source of the bleeding has to be identified, the bleeding needs to be arrested and, in some cases, the loss of blood volume will have to be rectified by way of a transfusion. A person's level of anticoagulation as produced by the administration of warfarin may become excessively high and might exceed the therapeutic level by a significant margin. Depending upon the circumstances, if excessive anticoagulation is identified in a patient, one means of rectification would be to withhold that person's warfarin consumption until the INR level has descended to the therapeutic range. In more urgent circumstances the excessive anticoagulation might be reversed more rapidly by the administration of substances such as prothrombinex, fresh frozen plasma or vitamin K, which results in the replacement of clotting factors that have been depleted by warfarin.

1.5. As alluded to earlier Mr Trengove had suffered a fall onto his right side. There is no evidence to suggest that the impact of his fall was grossly severe, but it was forceful enough to give rise to the pelvic fracture or fractures that were subsequently diagnosed. Specifically, an X-ray taken at St Andrews on the afternoon of 29 March 2008, reported as follows:

> 'There are minimally displaced fractures through the superior pubic ramus and the junction of pubic body and inferior ramus in the right side. No other pelvic fracture is seen. The hip joint (sic) show some minor degenerative change.' [1]

1.6. Following Mr Trengove's death, a post-mortem examination was conducted in respect of his body by Dr John Gilbert who is a forensic pathologist at Forensic Science South Australia. Dr Gilbert prepared a report[2] and he gave evidence in the Inquest. At autopsy Dr Gilbert was able to identify a fracture that is described in the X-ray as the fracture '*through the superior pubic ramus and the junction of the pubic body*'. Because of its anatomical position of the ramus in the right hand side, Dr Gilbert was not able to visualise any fracture of that bone. In any event, immediately behind the fracture in the pubic bone there was a large haematoma which was noted to be in association with, and therefore the product of, the pelvic fracture or fractures. This haematoma was also associated with tracking of blood into the pelvic soft tissues generally and into the lower part of the retroperitoneum on the right side. In his evidence Dr Gilbert explained that in the normal course of events such a fracture or fractures would only be expected to give rise to a small amount of bleeding, perhaps '*just a few mils of clot along a fracture of this type normally*'[3]. Dr Gilbert explains that with injury such as this there would be an expectation of some haemorrhage and bruising but not to the extent suffered by Mr Trengove[4]. Dr Gilbert was unable to identify precisely the exact site of the bleeding in this case, but could say that it certainly did not involve major blood vessels such as the femoral and iliac arteries and veins. Accordingly, he expressed the view that the bleeding could only have come from relatively small vessels in and around the site of the fracture. The salient feature of Dr Gilbert's evidence is that in his opinion, which I accept, the excessive bleeding in Mr Trengove's case was caused or exacerbated by his anticoagulation as the result of warfarin administration. It is known that when Mr Trengove's INR level was tested during the morning of the day of his death the level was 4.6 which exceeds the

[1] Exhibit C4, page 61
[2] Exhibit C2a
[3] Transcript, page 179
[4] Transcript, page 180

therapeutic level of between 2 and 3. Not only was Mr Trengove anticoagulated, but he was excessively anticoagulated. In Dr Gilbert's view this has given rise to the excessive bleeding associated with the fracture or fractures to his pelvis which, in the normal course, would not have given rise to such copious bleeding.

1.7. I add here that the bleeding into the retropubic space that Mr Trengove experienced is unlikely to have been detected by the naked eye unless there was overt swelling. It might, however, have been detected by a CT scan of his pelvis. No CT scan was administered prior to his death. The bleeding was not identified within St Andrews at any time before Mr Trengove died.

1.8. The INR of 4.6 as established on the morning of 30 March 2008 would not have been appreciably different at the time of his admission to St Andrews the afternoon before, notwithstanding that Mr Trengove overnight had received his usual dosage of warfarin. Therefore, his state of excessive anticoagulation would have existed at the time of the sustaining of his pelvic injury and at the time of his admission to St Andrews. This state of affairs could have been identified by way of an INR test upon his admission. Such a test in a man known to have been on warfarin ought to have been a matter of routine performance as soon as he was admitted to hospital, and yet it would not be performed until the following morning.

1.9. In his post mortem report Dr Gilbert has expressed the cause of Mr Trengove's death as retroperitoneal haemorrhage complicating pelvic fractures with contributing warfarin anticoagulation and ischaemic and hypertensive heart disease[5]. In his evidence before me Dr Gilbert revised that recitation to include reference to a retropubic haemorrhage that also existed[6]. Nothing turns on this amendment. I find that the cause of Mr Trengove's death was retropubic and retroperitoneal haemorrhage complicating pelvic fractures with contributing warfarin anticoagulation and ischaemic and hypertensive heart disease.

1.10. Notwithstanding Mr Trengove's history of heart disease, at autopsy no acute ischaemic changes were seen within the myocardium, which is the muscle of the heart, and there was no myocardial scarring indicative of previous myocardial infarction. There is thus no evidence that Mr Trengove had ever experienced a myocardial infarction (heart attack) either acutely or in the past. Although Mr

[5] Transcript, page 213
[6] Transcript, page 213

Trengove does not appear to have suffered a myocardial infarction or heart attack, Dr Gilbert explained that the cause of death was contributed to by his ischaemic and hypertensive heart disease. He suggested that in a situation of acute blood loss, as Mr Trengove had experienced, the oxygen carrying capacity of his blood was reduced with the result that his circulation was not able to keep up with the demands of his heart muscle for oxygen. His acute deterioration and death as a result was probably caused by a cardiac arrhythmia which would be the result of the compromise of the blood supply to his heart caused by the haemorrhage.

1.11. To my mind it is clear, and I so find, that the retropubic and retroperitoneal haemorrhage was caused by the fracture or fractures to Mr Trengove's pelvis. I further find that the predominant reason for the magnitude of the haemorrhage was Mr Trengove's state of anticoagulation caused by warfarin. I further find that Mr Trengove's acute deterioration and death was probably the result of an arrhythmia caused by the compromise of Mr Trengove's blood supply to the heart as caused by the haemorrhage.

1.12. There are two other matters that I should mention about Dr Gilbert's evidence. Firstly, Dr Gilbert expressed a view that if Mr Trengove's blood loss had been appreciated at an earlier point in time, his death might have been avoided. He said:

> 'I think that's quite possible. If it had been appreciated earlier, his warfarin rather than being ceased could have been reversed with vitamin K and he could have been transfused and put into an intensive care unit and I think there's a reasonable chance he could have survived if it had been appreciated at an earlier time that he was actually losing blood. But again, I'd defer to a clinician as to the definitive view on that.' [7]

On this issue, while Dr Gilbert suggested that he would defer to a clinician as to the ability to detect the existence of such a bleed clinically in a patient such as Mr Trengove, he suggested that periods of hypotension, which is low blood pressure, and overnight oxygen desaturations suggested that something '*wasn't quite right*'. While the origin could be cardiac, he suggests one ought to have thought about haemorrhage as another possibility bearing in mind that he was on warfarin[8]. An expert clinician was in fact asked by the Court to express a view about the same issue. I will come to the evidence of this witness in a moment but it is sufficient to say at this point that the expert agreed with Dr Gilbert's opinion. The physician responsible for Mr

[7] Transcript, page 192
[8] Transcript, page 207

Trengove's management at St Andrews, Dr Lakshmanan, expressed a more guarded view on that issue. Similarly, a second expert witness engaged by the physician's counsel, Dr Waters, also expressed a more conservative view. One of the questions at issue in this case is whether the bleeding ought to have been detected at an earlier point in time and, if it had been, whether there were means by which it could have been arrested thereby avoiding Mr Trengove's death.

2. Mr Trengove's clinical course at St Andrews Hospital

2.1. Mr Trengove was seen in the St Andrews Emergency Department by Dr Andrew Miller. Dr Miller is an experienced emergency medicine practitioner, although not an emergency physician. Dr Miller examined the X-rays that were taken of Mr Trengove's pelvis. Dr Miller did not regard the nature of the pelvic injuries as presenting any significant risk of bleeding. He did note, however, that Mr Trengove was on warfarin of 0.5mg per day.

2.2. Dr Miller prescribed morphine for Mr Trengove's pain and maxalon for nausea. He decided to admit Mr Trengove to St Andrews and contacted Dr Natesan Lakshmanan, a physician, to review Mr Trengove at the hospital. Dr Miller wrote up Mr Trengove's medication chart that included reference to the daily administration of warfarin. Dr Miller prescribed warfarin for him in accordance with Mr Trengove's existing dosage. In the event, that meant that Mr Trengove would be given his daily dosage of 0.5mg at 8pm that evening.

2.3. One of the last notations made by Dr Miller during the course of that afternoon was as follows:

> 'Check bloods in AM' [9]

This is a reference to Mr Trengove being subjected to routine blood tests that would usually be taken from a patient within the Emergency Department itself. The blood tests would examine such features as potassium, urea, creatinine and bicarbonate, all of which would be relevant to the patient's renal function, as well as glucose levels and other features of the patient's chemistry. The patient's haemoglobin level would be tested as part of this process. Haemoglobin is the component of a person's blood that carries oxygen to the tissues. A lower than normal haemoglobin level might,

[9] Exhibit C4

depending on the circumstances, indicate anaemia through blood loss from the circulation. Importantly in Mr Trengove's circumstances, a blood test taken upon his arrival at the Emergency Department would include an INR test to determine his level of anticoagulation, and in particular whether or not his INR was within the therapeutic range for the condition for which it was prescribed. Dr Miller did not order any blood tests to be undertaken while Mr Trengove was in his care. Rather, as already alluded to, one of his final orders was that the bloods should be checked in the morning.

2.4. Dr Miller gave a statement to the police on 24 April 2010 that was ultimately signed on 13 May 2010[10]. In two places in that statement he dealt with the issue as to why he did not order blood tests that would normally be regarded as routine in a patient's admission to an emergency department. In the first instance, he stated that the blood test was ordered for the next morning, as opposed to being done on admission, because it was not necessary in order to assess Mr Trengove's presenting problem[11]. In this context he described the blood tests that would be performed the following morning as 'opportunistic blood tests'. At another section of his statement he suggested that he did not consider the performance of blood tests as an urgent requirement because it was not considered an urgent part of the immediate assessment of the patient[12]. Those two passages suggest very strongly that Dr Miller's failure to order blood tests while Mr Trengove was under his care was a decision consciously made. Dr Miller gave evidence in the Inquest. In his evidence he said that he would ordinarily request blood tests during the assessment process and would do so at the time of the patient's presentation. When asked as to why he did not follow that practice he said that he could not recall the reasons for it but suggested that, from a reconstruction of the events, it was an oversight. When he became aware of that oversight later in the day, he decided to defer the blood tests until the following morning. Dr Miller said that at that stage he did not feel that his management of Mr Trengove would be likely to have been significantly altered by the results of blood tests taken at that time. Dr Miller's statements made closer to the event as to why he didn't order bloods and his surmise as given in evidence are to my mind not consistent.

[10] Exhibit C10
[11] Exhibit C10, page 4
[12] Exhibit C10a, page 6

2.5. In the event, blood tests taken at 8:45am the following morning revealed a picture of renal impairment at that time. Mr Trengove's haemoglobin count was 103 which is below normal, but not catastrophically so. The blood report suggests that this haemoglobin level reflects 'mild anaemia'[13]. Mr Trengove's INR was revealed to be 4.6 which is above the therapeutic range for him. There was discussion during the Inquest as to whether or not the picture of renal impairment as depicted by these blood results may have been revealed the previous afternoon if bloods had been taken from Mr Trengove at that time. The answer is, not necessarily. What seemed to be the subject of universal agreement during the Inquest, however, was that it was likely that Mr Trengove's INR level would have been virtually the same if blood tests had been taken from him the day before. There is also no dispute that if this INR level had been revealed during the course of the afternoon of 29 March 2008, as opposed to the following morning, Mr Trengove's warfarin administration that evening would have been withheld. The question as to whether or not his anticoagulation would have required active reversal, either to a therapeutic level or to a level of normality, is another issue that will be briefly discussed below.

2.6. In any event, Dr Miller did not regard Mr Trengove as exhibiting any sign of haemorrhaging from his pelvic injury. Dr Miller in essence relied upon Mr Trengove's clinical presentation in this regard and also upon Dr Miller's own experience and professional opinion that the pelvic injuries, as revealed by the X-rays, would in the normal course of events not have given rise to any significant bleeding. That assessment would have to be regarded as flawed due to the absence of any knowledge on Dr Miller's part that Mr Trengove's anticoagulation was excessive as would have been revealed by the routine blood test that Dr Miller should have ordered.

2.7. Mr Trengove was handed over to Dr Lakshmanan who examined Mr Trengove that day. Mr Trengove from that point onwards would be regarded as under the care of Dr Lakshmanan. Dr Lakshmanan is a consultant physician in private practice. He has visiting rights at St Andrews among other private hospitals. Dr Lakshmanan made notes of his examination[14]. Dr Lakshmanan specifically noted that Mr Trengove was on 0.5mg of warfarin per day. He noted his previous medical history which, it will be observed, does not make any reference to any chronic condition of renal failure. Dr

[13] Exhibit C4, page 53
[14] Exhibit C4, page 22

Lakshmanan naturally noted the pelvic injuries as revealed by the X-rays. Dr Lakshmanan gave an interview to police on 19 April 2010[15]. In his statement Dr Lakshmanan asserts that he can offer no explanation as to why blood samples were not taken from Mr Trengove. He said the blood samples are usually taken in an emergency department and stated that he was surprised that no blood sample was taken upon Mr Trengove's admission. Dr Lakshmanan gave evidence in the Inquest. In his evidence he suggested that he would have expected any abnormality as revealed by a blood test to have been drawn to his attention, and none was. The difficulty with this is that Dr Miller's note that blood tests would not be taken until the following morning would have been plain for him to see.

2.8. Dr Lakshmanan's plan for Mr Trengove was essentially pain relief and gradual mobilisation. There was no suggestion that Mr Trengove would undergo surgery. Dr Lakshmanan did not view Mr Trengove as exhibiting any evidence of haemorrhaging.

2.9. Mr Trengove was admitted to a medical ward at St Andrews where, overnight, he was looked after by the nursing staff. The general medical ward to which Mr Trengove was admitted did not have a medical practitioner on duty. The intensive care unit was staffed by one medical officer.

2.10. Mr Trengove's condition deteriorated during the course of the night. At first, Mr Trengove complained of nausea and vomiting. He stated that he had vomited up painkilling and anti-nausea tablets that he had taken. These had included endone, panadol and maxalon. He also complained of supra pubic pain at a level of 7 out of 10 which is significant. That night an agency nurse by the name of David Clayton was on duty in the medical ward. Nurse Clayton telephoned Dr Lakshmanan about Mr Trengove at approximately 11:20pm. Dr Lakshmanan ordered stemetil and indocid, the latter of which is a non-steroidal anti-inflammatory drug (NSAID)[16], to be given per rectum. These were given to Mr Trengove at 12:10am.

2.11. At about 5:40am Nurse Clayton made a note of Mr Trengove's condition as it had unfolded during the night. The note is comprehensive, was made at a time before Mr Trengove died and constitutes a reliable description of the night's events. Nurse Clayton noted the complaints of pain. He described his phone call to Dr Lakshmanan. He recorded the administration of stemetil and indocid. He also noted that further

[15] Exhibit C13b
[16] Non steroidal anti-inflammatory drugs

complaints of pain were made as a result of which endone had to be administered at 3:45am. Some of Mr Trengove's vital signs became matters of concern. Nurse Clayton noted fluctuating bradycardia, which is an abnormally slow heart rate, and hypotension, which is low blood pressure. He noted also a worrying oxygen saturation of 88% at one point in time. Mr Trengove was also noted to complain of an inability to void urine. He was noted to be very drowsy two minutes after receiving the endone, that he was rousable to voice but was very confused and disorientated when woken. A bladder scan was performed which, on the face of it, indicated that Mr Trengove's bladder contained 499 millilitres of urine at 4:30am. As it transpires this particular revelation may have been confused with an accumulation of blood within the abdomen. Mr Trengove was unable to void urine, although he did not complain of bladder discomfort. Nurse Clayton made a note to the effect that Mr Trengove may have been suffering from some sleep apnoea. Nurse Clayton also noted that when given oxygen, Mr Trengove's observations improved but that he was still unable to void. It was decided that Mr Trengove would be reassessed during the early morning shift change. There seems little doubt that Mr Trengove should have been medically reviewed during the course of the night as his condition worsened.

2.12. At 7:30am Mr Trengove's blood pressure was recorded as 80/60 which is remarkably low. At approximately 8am a Nurse Edwards, who had not been part of the overnight nursing shift, telephoned Dr Lakshmanan about Mr Trengove's condition. Specifically, it is noted that Dr Lakshmanan was advised by Nurse Edwards of the blood pressure recording of 80/60 that had been identified at 7:30am. She also told Dr Lakshmanan about Mr Trengove's inability to void and that his bladder was mildly distended. To relieve that difficulty Dr Lakshmanan ordered an indwelling catheter to be inserted. Dr Lakshmanan indicated that he would review Mr Trengove that morning. It was noted at 8:30am that the indwelling catheter was inserted with 'no difficulty'[17]. The issue as to whether Mr Trengove produced urine at that point was a matter that gave rise to a level of irritating uncertainty during the course of the Inquest. I say irritating because it should have been the subject of clear, contemporaneous and accurate documentation. The presence or absence of urinary output was said to have been an indication as to whether Mr Trengove's circulation may have been compromised as the result of bleeding, not an unimportant consideration.

[17] Exhibit C4, page 23

2.13. In the event, I was satisfied that there had been a urinary output on catheterisation. The question as to whether or not Mr Trengove's catheterisation gave rise to urinary output was not settled until a very late stage of this Inquest. There are two relevant witness statements relating to this issue. The first is that of a registered nurse by the name of Susan McKay, the urology nurse on duty at St Andrews, who had been asked by nursing staff on Mr Trengove's ward to insert the catheter. It was Nurse McKay who made the note to the effect that there had been no difficulty about the insertion of the catheter.

2.14. Nurse McKay's statement was taken on 18 December 2010. Nurse McKay no longer works at St Andrews. In her statement[18] Nurse McKay asserts that she has no recollection of this matter, but is able to reconstruct what happened from her notes and from her usual practices. Nurse McKay states that the timing of her note at 8:30am suggests that she probably performed the catheterisation between 8:15am and 8:30am. In her statement Nurse McKay asserts that if the insertion of the catheter had not resulted in the output of urine she would have documented that fact. There is no record of the output amount in this particular case. Nurse McKay's explanation for this is that the output was possibly minimal and likely to have been concentrated due to the patient's hydration status. If immediately following catheterisation the output had been significant she would have recorded that fact. In summary, Nurse McKay expresses confidence that there was some urinary output. If not, she would have discussed that with nursing staff and possibly with the patient's medical officer as well. She would not have recorded 'no difficulty'. In any event she would have arranged for nursing staff in the ward to contact the patient's treating doctor.

2.15. That there was a urinary output is supported by the affidavit of registered nurse Christa Edwards. Since these events Nurse Edwards has taken up residence in the Netherlands. Her whereabouts were established at a late stage of the Inquest. A statement was taken from her by an Australian Federal Police agent. It is evident from the contents of Nurse Edwards' statement that she has a vivid recollection of Mr Trengove and his difficulties on the ward. She was a relatively inexperienced registered nurse at the time and she asserts that she was emotionally disturbed by these events. Nurse Edwards was the registered nurse who telephoned Dr Lakshmanan at 8am. She reported the very low blood pressure of 80/60 and Mr

[18] Exhibit C20a

Trengove's inability to void. She was the member of the nursing staff whom Dr Lakshmanan instructed to catheterise Mr Trengove. Nurse Edwards obtained the services of a urology nurse to perform the catheterisation. That was Nurse McKay. Nurse Edwards was not present when the catheterisation took place. However, when she returned to the ward she viewed the indwelling catheter and bag and observed the volume and colour of the urine. From her memory the bag was slightly under half full and contained slightly concentrated amber urine. Nurse Edwards recalls that at one point she checked the catheter for kinks and found that there was amber urine in the tube leading to the bag. She recalls that she encouraged Mr Trengove to take sips of water to improve his hydration. In her statement Nurse Edwards says that she is able to recall this incident because of the relief she felt when the catheterisation resulted in Mr Trengove's inability to void being rectified. It was one less thing for her to stress about in the course of a stressful shift.

2.16. I find that there was a urinary output when Mr Trengove was catheterised, but that the output was not a copious one and that the urine was concentrated.

2.17. The blood sample was taken from Mr Trengove at 8:45am and I have already referred to the results which would have been made available at approximately 9:15am.

2.18. Dr Lakshmanan examined Mr Trengove sometime between 9am and 10:15am. Dr Lakshmanan made notes of his examination. He commenced them at the foot of the page on which Nurse Clayton had made his notes at 5:40am[19]. Dr Lakshmanan noted that Mr Trengove denied any pain. He made a note in respect of Mr Trengove; 'appear drowsy (after endone)' and then 'breathing shallow - sleep apnoea'. Dr Lakshmanan told me that he was of the view that Mr Trengove was dehydrated and that he may have suffered a myocardial infarction (or heart attack) during the preceding period. He made arrangements for Mr Trengove to be seen by a cardiologist. The cardiology review did not take place because Mr Trengove died later that day. Dr Lakshmanan gave evidence that the abnormal potassium, urea and creatinine results were not wholly explained by an acute renal failure. Rather he suggested that, together with the bicarbonate result, it indicated that Mr Trengove had suffered chronic renal failure but with an acute and more recent component. But there is no evidence from Mr Trengove's history to suggest that he had had chronic renal failure up to the time of his admission to St Andrews. Dr Lakshmanan did not regard

[19] Exhibit C4, page 23

the haemoglobin result as unusually low or indicative of a haemorrhage. In short, although Dr Lakshmanan ordered further administration of warfarin to be withheld, he did not form any view that Mr Trengove was experiencing a haemorrhage. Dr Lakshmanan's explanation for the failure to consider the possibility of bleeding is that it was not his experience that isolated, minimally displaced stable fractures of the pubic rami lead to excessive or life threatening bleeding, that his own examination of Mr Trengove on the afternoon of 29 March had not revealed any sign or symptom of circulatory impairment or bleeding, he had no reason to suspect before being advised of the blood results that Mr Trengove's anticoagulation was above therapeutic levels, that he attributed Mr Trengove's symptoms to an entirely plausible explanation consistent with his known medical history namely an acute cardiac event and that he lacked a full appreciation of the extent of Mr Trengove's symptoms in the early hours of 30 March.

2.19. Dr Lakshmanan left the hospital after he examined Mr Trengove.

2.20. In his evidence before me Dr Lakshmanan professed not to have a clear recollection of these events. He was unable to assist as to whether or not Mr Trengove had been passing urine since he was catheterised. Dr Lakshmanan made a concession that he had failed to check whether Mr Trengove had produced a urinary output on catheterisation. I am not certain whether this concession was appropriately made having regard to the fact that when he made that concession it was understood that there had been no urinary output. As it transpired, the evidence ultimately revealed that there had been some output.

2.21. Dr Lakshmanan suggested that he did not read Nurse Clayton's note of 5:40am. Specifically, he asserts that he was not aware of the fluctuating bradycardia that had been detected and noted by Clayton. He said in evidence that knowledge of fluctuating bradycardia was a matter that would have altered his management of Mr Trengove. And so it should have. If Dr Lakshmanan was unaware of significant features of Mr Trengove's condition throughout the night, it was not for want of any accurate note taking by nursing staff. That Dr Lakshmanan would not have read Nurse Clayton's note that contained reference to bradycardia is intrinsically difficult to believe. In his own note Dr Lakshmanan has reproduced in almost identical terms two notations made by Nurse Clayton in the latter's 5:40am entry. Nurse Clayton had recorded that Mr Trengove was 'very drowsy 2/60 after endone given'. Dr

Lakshmanan himself noted 'appear drowsy (after endone)'. Nurse Clayton noted '?some sleep apnoea evident'. Dr Lakshmanan noted 'breathing shallow - sleep apnoea'. There is in my view nothing inherently unlikely about the proposition that Dr Lakshmanan would have read Nurse Clayton's note. On the contrary, Dr Lakshmanan has indicated that he commenced his note on the very same page. There would have been no reason for Dr Lakshmanan not to know that Mr Trengove's presentation throughout the night had included a number of worrying matters such as his descending oxygen saturation that had to be normalised with oxygen, his fluctuating bradycardia and hypotension, his inability to void and his ongoing drowsiness. These were all matters that Dr Lakshmanan himself said, when viewed collectively, may have made a difference to his management. I do not believe that Dr Lakshmanan did not read Clayton's note. I believe that he read the note but was oblivious to what it signified as far as Mr Trengove's condition was concerned.

2.22. Dr Lakshmanan became aware of the blood results. He viewed them as demonstrating an acute on chronic renal failure. His differential diagnoses included dehydration and a possible myocardial infarction. Any diagnosis that Dr Lakshmanan made was flawed in the sense that he had not properly understood the significance of Nurse Clayton's account of the night's events. It does not appear to have occurred to Dr Lakshmanan that Mr Trengove's presentation could be due to haemorrhage. If that did occur to him there is no evidence that he did anything to eliminate it as a possible diagnosis. As will be seen, expert evidence from two sources would suggest that at that point in time a pelvic bleed ought to have been at the top of the list of differential diagnoses.

2.23. Dr Lakshmanan's plan was essentially to rehydrate Mr Trengove by way of a saline fluid administration. He also made some adjustments to Mr Trengove's pain management. He suggested that Mr Trengove be encouraged to eat and drink. I have already mentioned his plan to have Mr Trengove cardiologically reviewed. Notwithstanding his suspicion that Mr Trengove may have experienced a myocardial infarction, Dr Lakshmanan did not immediately order any of the usual blood tests such as repeat Troponin tests[20]. Rather, he told me that he contemplated such tests

[20] A Troponin T test is designed to detect and measure, within certain limitations, the existence of Troponin in the blood stream. The existence of Troponin in the blood stream can be the reflection of myocardial damage caused by an acute infarction or heart attack.

being performed the following day. He made a note that cardiac enzymes should be tested the following morning. This was a plan that was flawed in itself.

2.24. Mr Trengove died at 12:20pm that day. It is noted that at 12:10pm he experienced an unconscious collapse. The emergency team was called and attended. Resuscitative efforts that appear to have been thorough were unsuccessful.

3. The evidence of Professor Cade

3.1. Mr Trengove's clinical management at St Andrews in the light of his undoubted cause of death has been reviewed by two independent experts, independent in the sense that neither expert had any involvement in Mr Trengove's treatment. Professor John Cade is the Principal Specialist in Intensive Care at the Royal Melbourne Hospital and a consultant physician with a particular interest in haematology and the delivery of intensive care. Professor Cade was engaged by counsel assisting the State Coroner to examine Mr Trengove's clinical management at St Andrews and to provide an expert opinion as to its appropriateness and effectiveness. In the first instance he prepared a report dated 21 August 2009[21]. In due course he was asked to prepare a further report dated 6 July 2010. This report dealt with the responses to his first report that were made by the medical practitioners involved in Mr Trengove's care [22]. Professor Cade also gave evidence in the Inquest. After he gave evidence in the Inquest Professor Cade prepared a further report dated 4 January 2011[23] which dealt with issues that in the meantime had been commented upon by the second expert in this case.

3.2. The second expert to examine this matter was engaged by counsel for Dr Lakshmanan, Mr John Homburg. This expert was Dr Michael Waters who is a general physician. Dr Waters works predominantly in private practice at a number of hospitals, mainly Calvary Wakefield Hospital but also St Andrews. Dr Waters report and curriculum vitae were tendered to the Inquest[24]. In his report Dr Waters makes it clear that he would regard Dr Lakshmanan as a medical colleague.

3.3. Speaking generally at this point, Professor Cade's essential criticism of Mr Trengove's management at St Andrews is that there was an unacceptable failure to diagnose Mr Trengove's fatal haemorrhage. In his view there were features of Mr

[21] Exhibit C14
[22] Exhibit C14a
[23] Exhibit C21a
[24] Exhibit C24a

Trengove's presentation that ought to have led clinicians to such a diagnosis. Professor Cade expressed the belief that a timely and accurate diagnosis of Mr Trengove's haemorrhage would have resulted in Mr Trengove receiving appropriate treatment and have meant that he would probably have survived.

3.4. Professor Cade's specific criticisms involve a number of matters. He points to the failure to perform routine blood tests upon Mr Trengove's admission to the St Andrews Emergency Department in the afternoon of 29 March 2008. In his view there was a failure, by way of blood testing, to establish Mr Trengove's excessive anticoagulation as reflected by an INR in excess of the therapeutic levels. There was a failure to admit Mr Trengove to a high dependency unit where his condition could have been more effectively monitored overnight and corrected. Professor Cade is critical of Dr Lakshmanan's diagnostic approach which he suggests was flawed and which, in his view, did not properly take certain aspects of Mr Trengove's then clinical presentation properly into account.

3.5. Before dealing with Professor Cade's evidence and opinions in detail, it is as well to mention the fact that one feature of Professor Cade's criticism, as expressed in his initial report[25] and in his evidence, proved to be factually unfounded. I have already referred to this issue. In his first report Professor Cade suggested that the absence of any urine upon Mr Trengove's catheterisation on the morning of 30 March 2008 should have given rise to an immediate suspicion of circulatory impairment due to either bleeding and/or an acute cardiac event. In Professor Cade's second report, Professor Cade was quite critical of Dr Lakshmanan's failure to check the urinary output and to have established that Mr Trengove had not produced any urine upon catheterisation, particularly in light of the fact that Dr Lakshmanan had indicated in his police interview that if he had known that the catheter had not released urine, he would have conducted a diagnostic CT scan of the pelvis. However, it is now established that there was some urinary output upon catheterisation. Notwithstanding that the factual basis for this aspect of Professor Cade's criticism of Dr Lakshmanan has fallen away, Professor Cade in his final report nevertheless remains critical of the fact that there was a failure to diagnose Mr Trengove's haemorrhage and to perform the necessary treatment.

[25] Exhibit C14, paragraph 5

3.6. In his evidence during the Inquest Professor Cade suggested that Mr Trengove's excessive INR would have been established on his admission if an INR test had been performed. There was no dispute about this proposition and there was universal acceptance during the course of the Inquest, and in particular by Dr Miller and Dr Lakshmanan, that blood tests including INR ought to have been conducted that afternoon. Professor Cade suggested that the establishment of Mr Trengove's excessive INR at a level similar to what it was the following morning would properly have triggered a number of significant responses[26]. Firstly, there would have been a need to take into account the fact that the excessively high anticoagulation would predispose the patient to a risk of bleeding. Secondly, it would prompt the daily dose of warfarin which was administered that evening to have been withheld. Thirdly, any agent that would have aggravated bleeding, such as indocid which was administered during the course of the night, ought to have been avoided. Fourthly, there should have been a high index of suspicion that the patient was at risk of a bleeding complication such that an appropriate observational program ought to have been instituted. Professor Cade expressed the view in respect of that last matter that a high dependency unit would have been the appropriate place for such a program of observation to have been established.

3.7. In relation to the second of those measures, namely the withholding of Mr Trengove's daily dose of warfarin, there is no real suggestion that this dosage would have appreciably affected Mr Trengove's tendency to haemorrhage. The impact of a single dose would not be of immediate significance. Nonetheless, the fact that it was ordered and administered is reflective of a misunderstanding or a misappreciation of the risks presented by Mr Trengove's pelvic injury in conjunction with his excessive anticoagulation. The other matter that requires comment concerning the question of Mr Trengove's state of excessive anticoagulation is that Professor Cade agreed in his evidence that an INR level of 4.6 or thereabouts would not in itself dictate positive reversal of anticoagulation to a normal level unless there was evidence of bleeding[27]. In such circumstances Professor Cade suggested that the effects of the warfarin anticoagulation would be allowed to wear off by its own natural decline. However, he

[26] Transcript, page 385
[27] Transcript, page 422

suggested that there would still be a need for Mr Trengove to be under close observation with preparedness to intervene if the bleeding risk became reality[28].

3.8. Professor Cade did not suggest that there was a specific need during 29 March 2008, the day of Mr Trengove's admission, to perform a diagnostic CT scan to establish whether or not there was bleeding within the pelvis[29].

3.9. There is no evidence that the administration of warfarin overnight had any material affect on the outcome in terms of Mr Trengove's cause of death by way of haemorrhage. However, the fact still remains that the establishment of excessive anticoagulation during the course of the afternoon of Mr Trengove's admission ought to have given rise to a heightened appreciation of risk of haemorrhage that needed to be dealt with in a close observational setting. It was in respect of this aspect of Mr Trengove's management that Professor Cade was critical.

3.10. Regarding Mr Trengove's management overnight and his deterioration, Professor Cade was of the view that there were important observations during the course of the night that were not properly understood and taken into consideration. Professor Cade refers to the drop in blood pressure overnight from normal readings around 130 systolic to levels below 100. In particular, at 4:30am Mr Trengove's blood pressure had descended to 100/60. At 6am they had descended further to 90/60 and then to 80/60 at 7:30am. There was also the question of reported fluctuating bradycardia and descending oxygen saturation levels and the inability to void. Although Dr Lakshmanan in his evidence stated that if he had been aware of fluctuating bradycardia his management may well have been different, Professor Cade was of the view that the low blood pressure readings were of greater significance in any event. Professor Cade viewed the reading of 80/60 at 7:30am as a dangerous level[30] and explained as follows:

> 'Well the blood pressures are much lower than they were in the Emergency Department and then in the ward for a start. So there has been a significant decrease in blood pressure, and that is a concern regarding the patient's circulatory states of which there is a list of problems, the top of the list being bleeding.' [31]

[28] Transcript, page 423
[29] Transcript, page 424
[30] Transcript, page 435
[31] Transcript, page 436

It will be recalled that at 8am Nurse Edwards phoned Dr Lakshmanan to advise him of that reading of 80/60 and Mr Trengove's inability to void.

3.11. Professor Cade was also critical of Dr Lakshmanan's overnight order to nursing staff that Mr Trengove be administered indocid because this substance can adversely affect platelet function and therefore add to the already existing risk of haemorrhage caused by excessive warfarin anticoagulation. This presented as another instance of an overall failure to appreciate and deal with risk of haemorrhage.

3.12. As to the blood test results, Professor Cade expressed the view that these indicated a renal impairment[32]. As to the bicarbonate level that Dr Lakshmanan suggested was more in keeping with chronic renal failure, Professor Cade suggested that the bicarbonate level does not provide any message as far as acuteness or chronicity of renal failure is concerned. He suggested that the bicarbonate level of 19 was quite low and in accord with the other parameters relevant to renal impairment. Professor Cade suggested that in the circumstances, dehydration as an explanation for the renal impairment has to be considerable before renal impairment occurs because the body has very good compensatory abilities. In addition, the urea result in dehydration tends to be higher than the creatinine result and that this did not apply in Mr Trengove's case[33].

3.13. Professor Cade would not have regarded dehydration as a reasonable diagnosis, nor would he have regarded Dr Lakshmanan's other hypothesis of possible myocardial infarction as reasonable. Professor Cade suggests that a differential diagnosis of myocardial infarction is not consonant with Dr Lakshmanan's management plan. It will be remembered that Dr Lakshmanan, when he examined Mr Trengove on the morning of 30 March 2008, suggested and noted that cardiac enzyme tests be performed on the morning of the following day. Professor Cade suggested that such a plan was flawed insofar as one would regard such tests, including the well-known Troponin test, as immediately necessary in order to either diagnose or rule out a myocardial infarction[34]. Professor Cade suggests that if there had been an acute cardiac event during the night, the Troponin would definitely have been abnormal when the 8:45am blood test had been taken[35]. In the event that the result was

[32] Transcript, page 391
[33] Transcript, page 392
[34] Transcript, page 394
[35] Transcript, page 394

abnormal, one would still need to perform a repeat Troponin test in 4 hours. None of that was contemplated. In this regard it is known that there is no evidence at post-mortem that Mr Trengove had suffered an acute myocardial infarction.

3.14. As to the haemoglobin result of 103, Professor Cade expressed the view that that would not in and of itself have dictated a blood transfusion, but would have required an assessment as to whether the haemoglobin in the patient was stable or whether the patient was actively bleeding, especially where the patient has co-morbidities[36]. Dr Gilbert's evidence[37] suggested that one matter that would require evaluation in this regard is a comparison between the haemoglobin level established at 8:45am and Mr Trengove's level if blood tests had been taken the day before. A fall in haemoglobin level from normality to a level of 103, which is below normal, might have also provided evidence of haemorrhage having taken place in the intervening period. It is difficult to assail the logic of that proposition. In the event, Professor Cade was critical of Dr Lakshmanan's plan to infuse Mr Trengove with saline which he believed had been administered at an inadequate rate.

3.15. Professor Cade expressed the view in his first report and his evidence at the Inquest that if his haemorrhage had been identified, Mr Trengove would have been very likely '*rescuable*' until quite late in the morning[38]. Professor Cade suggested that upon the identification of Mr Trengove's haemorrhage by way of a CT scan, two things would inevitably have taken place. Firstly, an attempt would have been made to restore the circulation, and in this setting at 9:30am there would possibly not have been an urgent need for a blood transfusion. Resuscitation might have been effected by way of the administration of albumin. This would have resulted in improved blood pressure and urinary output. Secondly, there would be a need by way of a CT of the pelvis to identify the extent of the bleeding and then put measures in place to control it. One measure that would need to be addressed was Mr Trengove's anticoagulation. It would need to be reversed[39].

3.16. Professor Cade explained his opinion as to Mr Trengove's chances of survival by reference to the fact that declines in the elderly tend to be abrupt rather than gradual. The terminal event is usually quite sudden. This means that the chance of such a

[36] Transcript, page 402
[37] Transcript, page 205, line 32
[38] Transcript, page 401
[39] Transcript, pages 402-403

patient being rescued exists until a quite late stage in the clinical course[40]. Professor Cade expressed the view that ideally the timeframe for appropriate investigation of Mr Trengove's decline would have commenced at approximately 4:30am.

3.17. The other salient feature of Professor Cade's evidence was that only limited comfort ought to have been derived from a picture of haemodynamic stability that existed in the course of the afternoon and early evening of the day of Mr Trengove's admission. In short, a picture of haemodynamic stability would not necessarily preclude a later significant deterioration caused by bleeding.

4. The evidence of Dr Waters

4.1. Dr Waters expressed the view that because of Mr Trengove's clinical stability, he would not have considered it necessary or desirable to admit Mr Trengove to the hospital's high dependency unit at or prior to 7pm. He regarded transfer to the medical ward as appropriate. To my mind nothing of significance turns on whether Mr Trengove should have been transferred to a high dependency unit. Wherever he was accommodated, the issue seems to be one of close monitoring and observation rather than his geographical situation within the hospital. As I understood Professor Cade's criticism, there was inadequate monitoring and response to monitoring and a lack of medical supervision that a high dependency unit could have provided. I make no finding as to whether or not it would have been more appropriate for Mr Trengove to have been accommodated in a high dependency unit.

4.2. Dr Waters acknowledges that indocid belongs to a class of medications that can increase the risk of bleeding. For these reasons he states that he would avoid using an agent such as this in a patient such as Mr Trengove who is on warfarin and who has other co-morbidities. However, while he acknowledges that the administration of indocid was unwise, he doubts whether a single dose of indocid would have had any significant effect on the haemorrhage suffered by Mr Trengove.

4.3. As to the question of reasonable diagnosis, Dr Waters was presented with a number of factual scenarios. These scenarios included the state of affairs as revealed in the 8am phone call between Nurse Edwards and Dr Lakshmanan, the position as it was between 9am and approximately 10:15am when Dr Lakshmanan reviewed Mr

[40] Transcript, page 404

Trengove (and on the assumption that urine had been detected after catheritisation) and, thirdly, in the light of the blood and general chemistry results that Dr Lakshmanan became aware of shortly after his examination of Mr Trengove. In each of the scenarios posed, Dr Waters has placed 'pelvic or other bleeding' at or near the forefront of differential diagnoses. For example, differential diagnoses during the 8am phone call that would have presented themselves to Dr Lakshmanan would have included:

'(a) Urinary retention with vagal effect on blood pressure and heart rate

(b) Pelvic or other bleeding' [41]

In relation to the scenario between 9am and 10:15am, Dr Waters would place pelvic or other bleeding at the top of the list of differential diagnoses. In relation to the time at which the blood results became available, Dr Waters has placed pelvic or other bleeding at the top of the list of differential diagnoses. Dr Waters was asked to comment upon Dr Lakshmanan's diagnosis of dehydration. In light of the fact that the blood results would have been available by approximately 9:15am, and that Dr Lakshmanan reviewed those blood results not long after, the reasonableness of any earlier diagnosis, particularly during a phone call at 8am, is somewhat beside the point. Regarding the period between 9am and 10:15am, Dr Waters suggests that dehydration alone would not have been a good explanation for Mr Trengove's symptoms and signs at this point. An acute myocardial infarction was possible. I have already referred to the fact that regardless of whether a myocardial infarction was reasonably suspected or not, tests to confirm or rule out myocardial infarction were never performed and were only ordered to take place the following day. When the blood chemistry results are borne in mind, Dr Waters has indicated as follows:

'Q. Do you consider that the general chemistry results are consistent with Mr Trengove being dehydrated and suffering from acute on chronic renal failure?

A. The likely cause of Mr Trengove's acute on chronic renal failure was hypotension with resultant reduced renal perfusion. In retrospect, haemorrhage was the likely cause of the hypotension. Again, dehydration was a possible contributor, but unlikely to be the primary cause.' [42]

Dr Waters again places pelvic or other bleeding at the top of the list of differential diagnoses in this scenario. Nevertheless, Dr Waters suggests that Dr Lakshmanan's

[41] Exhibit C24a. page 3
[42] Exhibit C24a, page 4

view that Mr Trengove's symptoms were explicable by dehydration and/or acute myocardial infarction remained as 'reasonable thoughts, but seemed less likely than haemorrhage in the clinical context'. Dr Waters' views about the reasonableness or otherwise of Dr Lakshmanan's diagnostic approach can be gleaned from what he suggests ought to have taken place at this point in time. Dr Waters suggests that once the blood chemistry results were known, anticoagulation should have been rapidly reversed, urgent investigations should have been undertaken including a CT scan of the abdomen and pelvis looking for haemorrhage and blood and urine cultures ought to have been conducted. In addition, an urgent cross-match for blood would need to be performed with a view to transfusion should the haemoglobin level drop further. As well, consideration might have been given to contact involving an Intensive Care Unit team. It is clear from that description that Dr Waters is of the opinion that steps should have been taken to diagnose and treat Mr Trengove's haemorrhage. That much is identical with the view expressed by Professor Cade.

4.4. I find that the measures that both Professor Cade and Dr Waters have advocated undoubtedly should have been put in place once the blood results were known.

4.5. As to Mr Trengove's prognosis, Dr Waters feels that his prognosis was grave at the time he began to deteriorate in the early hours of 30 March 2008. He expresses the view that his condition was irretrievable at the time he ultimately went into cardiac arrest at 12:10pm that day. I do not understand Dr Waters to express a contrary view to Professor Cade's opinion that Mr Trengove might have been rescued at a very late stage prior to his eventual cardiac arrest if proper measures had been put in place.

4.6. Professor Cade's third report dealt with the views expressed in Dr Waters' report. Professor Cade agrees that pelvic or other bleeding should have been towards the top of the list in any diagnostic scenario. Professor Cade does emphasise in his third report that Mr Trengove's differential diagnoses, even at 8am, should have taken into account the fact that the patient had a known fractured pelvis and that a complication of that fracture should have been foremost among the diagnoses, even at that time. He disagrees with any suggestion that Dr Lakshmanan's diagnosis and treatment for dehydration was reasonable. He agrees with Dr Waters that the patient should have been medically reviewed overnight.

5. Conclusions

5.1. Mr Trengove should have been subjected to blood testing upon his arrival at the St Andrews Emergency Department. Specifically, a test for INR anticoagulation should have been conducted. This would have revealed an INR level approximating the INR level that was established the following morning, namely 4.6. The possibility that Mr Trengove might copiously bleed from his pelvic injuries was not adequately assessed at St Andrews. Specifically, his management plan did not sufficiently take into account what could easily have been established at the outset, namely that he was excessively anticoagulated.

5.2. Mr Trengove's deterioration overnight should have been the subject of examination by a medical practitioner during the course of that night.

5.3. On the morning of Sunday 30 March 2008 Dr Lakshmanan entertained possible diagnoses in respect of Mr Trengove of dehydration and a myocardial infarction. I conclude that there was in existence a more likely diagnosis that ought to have been considered and that is that Mr Trengove's presentation was consistent with his having bled from his pelvic injuries. The index of suspicion in relation to such a diagnosis was sufficiently high to have warranted an immediate CT scan of his pelvis. A CT scan of his pelvis probably would have revealed the bleeding within Mr Trengove's pelvis.

5.4. Mr Trengove's bleeding into his pelvis ought to have been identified no later than the time at which Dr Lakshmanan became aware of the blood results which indicated an acute deterioration in Mr Trengove's renal function.

5.5. I find on the balance of probabilities that if Mr Trengove's bleeding in his pelvis had been identified during the morning of 30 March 2008, his chances of survival would have been significantly improved. It is impossible to be absolutely certain that he would have survived. It is also impossible to be absolutely certain of the point in time at which Mr Trengove could not have been retrieved.

6. Recommendations

6.1. Pursuant to Section 25(2) of the Coroners Act 2003 I am empowered to make recommendations that in the opinion of the Court might prevent, or reduce the likelihood of, a recurrence of an event similar to the event that was the subject of the Inquest.

6.2. Mr Coppola of counsel appeared for an on behalf of St Andrews Hospital. Mr Coppola produced documentary evidence relating to changes that have been made in relation to procedures within St Andrews. There is now in existence a protocol[43] for warfarin over-anticoagulation that includes reference to the fact that a patient's current INR will usually impact upon their management plan. The document requires an INR test to be performed as part of an emergency service assessment for all presenting patients receiving anticoagulation therapy. The document, in bold, urges clinicians to be aware of occult or concealed bleeding risks in even minor trauma to the head, thorax, abdomen or pelvis and to investigate or closely observe as appropriate. The document also sets out strategies for treatment of warfarin over-anticoagulation.

6.3. An updated Nursing Practice Manual was also produced by Mr Coppola[44]. The subject of the extract from the manual in question involves management of the deteriorating patient. The manual requires a clear management plan to be devised. The plan must include a description of the action to be taken if there is no improvement or the patient's condition continues to deteriorate. It should govern such things as the circumstances in which the patient's medical specialist should be called or, indeed, recalled. The document also deals with improved frequency of observations.

6.4. In the event it has not been necessary for the Court to make any recommendations concerning the taking of blood samples upon admission to St Andrews Hospital or in relation to improving the level of observation of patients such as Mr Trengove.

6.5. Mr Homburg, who appeared for Dr Lakshmanan, has made other suggestions for positive change. In particular Mr Homburg urges me to consider making a

[43] Exhibit C18
[44] Exhibit C23

recommendation in relation to the St Andrews Hospital's practices regarding fluid balance observations and records.

6.6. I make the following general recommendation.

1) That St Andrews Hospital ensure that patients with pelvic fractures who present in an anticoagulated or over-anticoagulated state be subject to the closest observation possible. This should include regular monitoring of the patient's vital signs, regular observation of the person's renal function, fluid balance observations and recording, regular testing of a person's haemoglobin and state of anti-coagulation and constant observation as to the patient's clinical presentation.

Key Words: Anticoagulation; Warfarin; Hospital Treatment

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 22nd day of June, 2011.

Deputy State Coroner