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 30th and 31st days of January 2008, the 13th day of February 2008 and the 22nd day of August 2008, by the Coroner's Court of the said State, constituted of Anthony Ernest Schapel, Deputy State Coroner, into the death of Kenneth Morton Carter.

The said Court finds that Kenneth Morton Carter aged 76 years, late of 10 Sonar Court, Seaford Rise, South Australia died at Seaford Rise, South Australia on the 8th day of June 2005 as a result of ruptured abdominal aortic aneurysm. The said Court finds that the circumstances of his death were as follows:

1. Introduction and cause of death

1.1. Kenneth Morton Carter was a 76 year old man who lived with his wife. He died unexpectedly at his home address on 8 June 2005. Following his death, his remains were subjected to a post-mortem examination. The autopsy was performed by Professor Roger Byard who was the Chief Forensic Pathologist at Forensic Science SA. The cause of death was clearly established as a ruptured abdominal aortic aneurysm. I find this to be the cause of death. The aorta, which originates at the heart, is the body's principal blood vessel. An aortic aneurysm is represented by an area of stretching with associated weakness of the aortic wall, frequently the result of chronic high blood pressure (hypertension). An abdominal aortic aneurysm is also known by its initials AAA. When this expression appears in these findings it is used to describe an abdominal aortic aneurysm, not a ruptured abdominal aortic aneurysm. Not all AAAs are fatal. However, when a AAA ruptures, extensive haemorrhage is the usual result. If the blood flow is not arrested, death will follow. This is what

happened in respect of Mr Carter. The basis for the rupture in Mr Carter's case was marked atherosclerosis of the abdominal aorta.

1.2. Mr Carter had a significant medical history. He was a very big man with a weight of 136 kilograms and a height of approximately 188 centimetres. His weight would suggest that he was morbidly obese. I mention a number of facets of Mr Carter's medical history in a moment, but the salient feature is the fact that he had a previous AAA diagnosed and treated in 1992. This aneurysm had been the subject of successful surgical intervention. A gortex graft measuring 80 millimetres extending from the bifurcation of the common iliac arteries proximally had been put in place at that time. The fatal AAA began approximately 40 millimetres above the previous graft. It extended 100 millimetres in length and had a circumference of 100 millimetres. It did not involve the renal artery. The fatal aneurysm was associated with a 50 millimetre laceration on the right side with extensive haemorrhage into the upper retroperitoneal space, particularly on the right side. This AAA remained undiagnosed prior to it rupturing and causing Mr Carter's death. The evidence before me, which I accept, is that although it is unusual for a person to experience a second AAA during his or her lifetime, it is more common than in a person who never had aneurysmal disease in the first place. I addition, a history of aneurysms should alert one to the possibility of further aneurysms because aneurysmal disease is reflective of more generalised disease in the arteries of the body.

1.3. Other features of Mr Carter's medical history include chronic obstructive pulmonary disease, chronic hypertension (high blood pressure), for which he had been diagnosed and treated for some time, back pain, atrial fibrillation, for which he was being treated with Warfarin, a right total hip replacement and varicose vein removal. There were other features of his history that do not have any particular relevance. The Warfarin medication that had been utilised to control Mr Carter's atrial fibrillation, which is an abnormality of heart rhythm, may have contributed to Mr Carter's death insofar as it could have predisposed him to haemorrhage.

1.4. Shortly before his death on 8 July 2005, Mr Carter had been hospitalised on two occasions. There can be no doubt that on both of those occasions the AAA that would claim Mr Carter's life was in existence. Mr Carter's AAA had never been diagnosed either before, during or after any of his recent periods of hospitalisation. It is fair to say that Mr Carter had existed in complete ignorance of his AAA. This is not entirely

unusual as they are not necessarily symptomatic unless and until they begin to leak, dissect or indeed fatally rupture. However, as was plain in the course of the Inquest a AAA is usually detectable upon a CT scan of the abdomen. The CT might also pick up the fact that the AAA is leaking, should that be the case. The evidence before me suggested that an undiagnosed and untreated leaking AAA carries 100% mortality. An abdominal CT scan was not performed with respect to Mr Carter on either of the occasions he was hospitalised in the days leading up to his death.

2. Mr Carter's presentation at the Emergency Department of the FMC on 31 May 2005

2.1. Mr Carter's first episode of recent hospitalisation occurred on 31 May 2005. On that occasion he was at home hanging out some washing when he felt a sudden pain in his lumbar region. As a result, paramedics were called and Mr Carter was conveyed by ambulance to the FMC. The South Australian Ambulance Service (SAAS) report that is part of Exhibit C4, being the Flinders Medical Centre (FMC) file with respect to Mr Carter, records that Mr Carter had been hanging out the washing at 5pm and had felt sudden back pain of lumbar origin. The FMC Emergency Department progress notes, that also form part of Exhibit C4, record that Mr Carter provided a history of lower back pain, in particular pain associated with a 'slipped disc' that had resolved with physiotherapy. It recorded that Mr Carter had described an episode where he had been hanging washing on the line, had reached up towards the line and had 'twisted' his back. Following that, he had experienced pain to the mid to right-hand side of his back. On examination Mr Carter was 'distressed with pain and was tender upon palpation'. Mr Carter was to tell medical staff at the FMC, when he again presented the following day that during the clothes line incident he had felt his 'disc go' in his lower back. On 31 May Mr Carter was treated with analgesia, discharged and told to return if he experienced any further concerns. It was common ground during the course of this Inquest that Mr Carter's episode of back pain and consequent presentation at the Emergency Department of the FMC on 31 May was unlikely to have been associated with his AAA. There is no evidence to suggest that what Mr Carter experienced on this particular occasion was anything more than perhaps an exacerbation of his chronic back trouble that had undoubtedly existed as part of his clinical history. In particular, on this occasion there was no loss of consciousness or low blood pressure (hypotension). Accordingly, there is no suggestion that on 31

May 2005 there had been any compelling reason to conduct tests such as an abdominal CT scan that may have been diagnostic of the presence of a AAA.

3. The incident at the physiotherapist's rooms on 1 June 2005

3.1. The following day, 1 June 2005, Mr Carter sought relief from his back trouble at his physiotherapist. This attendance occurred in the afternoon. Mr Carter there experienced an episode that again resulted in the attendance of SAAS. He experienced short periods of loss of consciousness on two occasions when he sat up from a lying position. This episode was variously described by Mr Carter to SAAS officers and to medical staff at the FMC where he was again taken. The SAAS report, which forms part of Exhibit C4, describes the incident in these terms:

> 'Went to physio. Collapsed after Px attempting to sit up. Short period LOC. Some abdo+ back pain.'
> (Px is patient; LOC is loss of consciousness)

The Emergency Department patient record records the following quote:

> 'Uncons collapse at physio. Short LOC.
> Had back pain yesterday
> Strained at home
> Feeling light-headed and dizzy
> Nauseated. Has vomited.
> C- mild abdominal pain.' [1]

3.2. The FMC patient journey record[2] also sets out the details of Mr Carter's presentation and complaint. This document records that Mr Carter had woken up at 1:30pm that day and had taken Diazepam. He had experienced 'foggy' vision whilst in his car, presumably while en route to his physiotherapist. He visited the physiotherapist in the afternoon for massage and ultrasound therapy. He had sat up from a lying position and felt dizzy/unbalanced/had blurred vision, with a brief loss of consciousness of some seconds. This is described as having improved on lying down. He sat up again and the same thing happened. There was some nausea associated with this episode. Mr Carter appears to have been asked whether he had experienced any previous blackouts. He responded negatively. In the course of this examination Mr Carter

[1] Exhibit C4, page 6
[2] Exhibit C4, page 7

denied any chest or abdominal pain which contradicts what he apparently had said earlier at the FMC Emergency Department and indeed what he had originally described to the SAAS to whom he had complained of vertigo and nausea on standing with a small amount of back and abdominal pain in the umbilical region/lumbar back.

3.3. A feature of Mr Carter's presentation on 1 June 2005 was the fact that the SAAS recorded a blood pressure of $^{90}/_{65}$ which is significantly low (hypotension) for an adult. It is to be borne in mind that Mr Carter suffered from chronic high blood pressure (hypertension), although it appears that this had been managed to a degree. It is of some interest that when Mr Carter had presented on the previous day, blood pressure readings appear to have been taken, including high systolic readings of 180, 170 and 160. There was also a reading apparently taken in the Emergency Department of 138 systolic. As will be seen, although not diagnostic, the blood pressure as recorded on 1 June 2005 by the SAAS, being $^{90}/_{65}$ to begin with, is quite hypotensive and is of some significance when it is remembered that Mr Carter experienced two periods of loss of consciousness at the physiotherapist. As it transpired, this low blood pressure recording does not appear to have been given any significant consideration in a diagnostic sense either at FMC or at the Flinders Private Hospital (Flinders Private) to which Mr Carter was ultimately transferred and admitted.

4. Mr Carter's presentation at the Emergency Department of the FMC on 1 June 2005

4.1. The Emergency Department patient record[3] reveals that Mr Carter's initial assessment took place at 4:30pm. A lying blood pressure of $^{101}/_{68}$ was there recorded. This was an improvement on the figure of 90 systolic, but is still hypotensive. A temperature of 36.5ºC was also recorded, as was a pulse rate of 108. It was here recorded that Mr Carter had earlier that day suffered the unconscious collapse at the physiotherapist, that he had felt light-headed and dizzy, was nauseated and had vomited. He complained of mild abdominal pain. At that time, as is clearly noted in the record, a previous medical history that included atrial fibrillation and a AAA in 1992, as well as a THR (total hip replacement), was identified as part of Mr Carter's medical history.

[3] Exhibit C4, page 6

4.2. At 6pm Mr Carter was seen by a doctor at the Emergency Department of FMC. The doctor has recorded the two episodes of brief loss of consciousness that afternoon. The doctor has also recorded the incident of the previous day where Mr Carter had said that he had felt his 'disc go' in his lower back, a problem that he had in the past. In fact, it is recorded here that Mr Carter had suffered a slipped disc three months previously. Upon Mr Carter's discharge from FMC on the previous day, he had been prescribed with Panadeine Forte and Diazepam. He had taken two Diazepam tablets prior to going to bed at 2am, had woken up at 1:30pm and had taken a further Diazepam tablet. He had experienced 20 minutes of foggy vision whilst in the car, and had then recounted the episode at the physiotherapist that had involved dizziness, the feeling of being unbalanced, blurred vision and brief losses of consciousness that had improved on lying down. There was also some nausea associated with that experience. Significantly, Mr Carter denied previous blackouts. He is recorded as also denying chest pain or abdominal pain at that point. As earlier observed, this latter denial to the examining doctor is to be contrasted with what he had said originally at the Emergency Department when he had complained of mild abdominal pain. It also contrasts with what had been recorded by the SAAS, namely that he had experienced a small amount of back and abdominal pain and that his abdomen was soft and tender, one assumes on palpation by ambulance personnel.

4.3. The examining doctor has recorded Mr Carter's previous medical history as including hypertension, chronic obstructive airways disease with 15% lung capacity, atrial fibrillation, for which he was on Warfarin, a gastro-intestinal bleed in September 2004 which had been secondary to anticoagulation/NSAIDS use, the slipped disc and the AAA repair in 1992 which is recorded as having involved an aneurysm of 13cm. Mr Carter evidently admitted to having a cigarette smoking habit of 30 cigarettes per day for 40 years, but that he had quit in 1992.

4.4. On examination, Mr Carter had a temperature of 37ºC, which is said to be at the upper limit of normal. His blood pressure was $^{130}/_{65}$. It was recorded that Mr Carter had an 'abdomen obese ++' that was soft and non-tender with no masses. The systolic blood pressure level of 130 represents an increase and is a normal reading.

4.5. As to Mr Carter's losses of consciousness, the following has been recorded:

'SA/Syncope - ? Underlying dehydration/Sepsis'

I take this to mean that at that point in time, dehydration and/or sepsis was considered to be a possible explanation for the losses of consciousness.

4.6. Certain tests appear to have been ordered at that time including blood tests, a chest X-ray, a urine dipstick and tests for postural blood pressure in both the lying and sitting positions.

4.7. It will be noted that although there appears to have been some consideration given to the possible underlying causes of Mr Carter's losses of consciousness, there does not appear to have been any consideration given to the possibility that it was connected with an existing AAA in this context. There is no notation of any such consideration having been given.

4.8. The blood pressure that had originally been recorded by the SAAS, namely $^{90}/_{65,}$ which is hypotensive, does not appear anywhere in the Emergency Department notes except in the SAAS patient report form, a copy of which has obviously been incorporated into the FMC notes.

4.9. A chest X-ray was performed on the evening of 1 June 2005 and the report is at page 14 of Exhibit C4. The 'exam date' is recorded as 1 June 2005 at 7:08pm. The report, insofar as is relevant, stated that there was some patchy atelectatic changes seen in both bases of the lungs. The upper lung fields appeared grossly clear. The report noted the existence of 'marked unfolding of the thoracic aorta'. The report also indicated that there was an impression of an extra density projected over the inferior aspect of the right hilar region and suggested that this abnormality should be correlated with any old chest X-rays and that a CT scan may be indicated to further evaluate this if there was no clinical resolution. The report went on to state 'it may be infective in nature given the clinical history'. This X-ray did not reveal anything about the state of the abdominal aorta.

4.10. The X-ray report spoke of the condition of the thoracic aorta. It will be noted that this section of the aorta is not that which was involved in the fatal rupture. I return in due course to the condition of the thoracic aorta as revealed by the X-ray. The reference to, and suggestion of, the performance of the CT scan is a reference to a possible CT scan of the chest region. Such a CT scan in the event was not performed, but it is clear that a CT scan of the chest would not have revealed any abnormality as far as

Mr Carter's abdominal aorta is concerned. I return to the significance of the chest X-rays in due course, particularly in the light of other findings in respect of Mr Carter.

4.11. At 9:30pm that evening FMC Emergency Department medical staff discussed Mr Carter's presentation with Dr Peter Allcroft who is a Consultant Physician at Flinders Private. A record of these discussions was made within FMC and the principal aspect of the initial discussion was whether Dr Allcroft would accept Mr Carter as a patient in Flinders Private. During the course of this conversation, which took place on the telephone, there was discussion between the two medical practitioners inter alia concerning C-reactive protein and the obtaining of blood cultures if Mr Carter's temperature were to spike overnight. The dominant feature of the discussion was the possibility that Mr Carter was suffering from an infective disorder. There was no discussion about the possibility of existence of a AAA.

4.12. At 10:30pm there was further discussion with Dr Allcroft in the light of the X-ray results.

4.13. Dr Allcroft agreed to accept Mr Carter as a patient at the Flinders Private and Mr Carter was transferred from the FMC ED that evening. The referral document that facilitated the transfer from the Emergency Department to Flinders Private referred to a diagnosis of syncope (loss of consciousness) for investigation/? (R) pneumonia. The document also listed among Mr Carter's several comorbidities a previous AAA repair as well as his atrial fibrillation, etc. The document also referred to the performance of blood cultures if Mr Carter's temperature were to rise above 30ºC and the need to compare Mr Carter's chest X-ray to previous X-rays with a view to further investigating the right hilar mass as revealed by the X-ray. Again, there is no reference to the possibility of an existing AAA. With Mr Carter's transfer to Flinders Private the involvement of FMC Emergency Department ceased. From that point Mr Carter was for the most part in the hands of Dr Allcroft until his discharge from Flinders Private on 6 June 2005 at approximately 6pm. Mr Carter died on the morning of 8 June 2005.

5. Issue upon Inquest

5.1. The existence of Mr Carter's AAA throughout the entirety of the period from 1 June 2005 to his death on 8 June 2005, which of course includes his period of hospitalisation at the FMC Emergency Department and Flinders Private until his

discharge on 6 June 2005, cannot be denied. The fact that Mr Carter also died from the ruptured AAA is also the inescapable conclusion from the post-mortem examination. What appears to be in contention, however, is whether or not any of the symptomatology that Mr Carter displayed during that same period can be assigned to his AAA and whether it should have been seen as heralding a rupture. An associated question of course is whether any catastrophic changes that were taking place in his AAA ought to have been identified while he was in hospital. Another issue involves whether or not Mr Carter was suffering from an infective disorder during that same period, and if so, whether the various symptoms displayed by Mr Carter, including those that he displayed at the physiotherapist on 1 June 2005, are to be assigned to an infective process. If that is the case, then the symptoms that might be assigned to an infective process, it is said, could have masked and confused the signs and symptoms being displayed by whatever was happening in Mr Carter's AAA. The central question in the Inquest is reduced to whether or not Mr Carter's AAA should have been diagnosed and, if it had been, whether anything in the way of medical or surgical intervention before its rupture could have saved his life.

5.2. I heard evidence from Dr Allcroft. Dr Allcroft's position is that Mr Carter's presentation during the period of time in which Mr Carter was in his care was totally in keeping with an infective disorder, possibly pneumonia, and that it was reasonable for Mr Carter's treatment to have been directed towards such a diagnosis. Dr Allcroft's position is that a possible diagnosis of a leaking or dissecting AAA was for him simply not on the table. No such provisional or differential diagnosis from anyone in the FMC Emergency Department was ever suggested or otherwise presented to him. The Emergency Department personnel presented the case to him very much on a basis of there being in existence an infective disorder, possibly pneumonia, which admittedly seems to have been the focus of inquiries within that Department as well. In any event, Mr Carter's presentation was, for Dr Allcroft, classically that of a man suffering from a serious infection. Dr Allcroft would also take issue with the proposition that Mr Carter's AAA was in any case not responsible for any of his symptomatology.

5.3. Complicating matters was the fact that two very experienced medical practitioners, Professor Jamieson and Dr Braund, both experts in their fields, could not agree in evidence whether or not the diagnosis of a leaking or dissected AAA ought to have

been made, and in particular by way of an abdominal CT scan. Professor Jamieson would advocate a CT scan as being an essential and integral diagnostic aid in the evaluation of any presentation such as Mr Carter's.

6. The evidence of Dr Allcroft

6.1. Dr Peter Allcroft gave evidence to the Inquest. He was the only clinician involved with Mr Carter's care who gave oral evidence. Dr Allcroft is a physician who received his specialist qualifications in 1996 and has been a Consultant Physician since 1997. Much of his work as a physician has been undertaken at Flinders Private. Dr Allcroft described the work of a consultant physician as dealing with complex medical issues. A consultant physician is asked to investigate, assess and manage a range of issues which include heart disease, lung disease, kidney disease and cerebrovascular disease. His role at Flinders Private involved the care of inpatients. The medicine that he practices there involves acute medical issues such as the care of people who have experienced heart failure, pneumonia, the exacerbation of an airways disease and an infective process.

6.2. Dr Allcroft told me that the treatment and management of an aortic aneurysm would usually reside in the hands of a vascular surgeon. This is true. It was not something that he would have managed routinely. In the previous ten years he had had no direct experience in treating a ruptured AAA because he suspected that patients suffering from the same were diagnosed in emergency settings and then referred to surgical specialists. It will be observed here that there had been no such diagnosis at the Emergency Department of FMC and that the case was referred to Dr Allcroft on the basis that it probably involved some kind of infective process such as pneumonia. AAA complications were not part of any differential diagnoses at FMC.

6.3. Dr Allcroft did not consider a diagnosis involving a AAA. Rather, Dr Allcroft's investigations were for the most part focussed on the possibility that Mr Carter was suffering from a sepsis. Supporting such a diagnosis in his mind was a markedly elevated white cell count. In addition, a C-reactive protein test that was initiated by Dr Allcroft also supported an infective scenario. The underlying infection was thought to be a respiratory tract infection. Pointing to that possible diagnosis was Mr Carter's longstanding severe airway disease, raised inflammatory markers including the white blood cell count and the C-reactive protein result. The chest X-ray that was

performed also suggested an infective process. To Dr Allcroft the earlier blood pressure results were also in keeping with that diagnosis. It is to be observed, however, that it appears that Dr Allcroft was not informed of the quite markedly low blood pressure that had been detected by the SAAS upon their attendance at Mr Carter's physiotherapist's rooms.

6.4. In the days following Mr Carter's admission to Flinders Private antibiotics that had been prescribed for him were believed to have accounted for a gradual improvement in his infective and septic condition. As well, Mr Carter's blood pressure improved and remained stable.

6.5. The one feature of Mr Carter's presentation that was indeed highly supportive of a diagnosis of an infective process at work was the fact that the white cell count was markedly raised but with toxic changes in the neutrophils. This feature of Mr Carter's presentation was said to have distinguished Mr Carter's complaint from, say, an inflammatory process that might have been the result of a leaking or dissecting AAA. Toxic changes, sometimes referred to as toxic granulation, are indicative of a severe infection as opposed to some other inflammatory process. Such an infection was in keeping with not only the elevated C-reactive protein, but with the fact that it was markedly elevated at a figure of 76 where normal is less than 6.

6.6. As to symptoms that would have been consistent with a leaking or dissecting AAA, Dr Allcroft saw no evidence of it. He did not conduct any tests that would specifically be diagnostic of the same. The symptom of lower back pain he considered may have been part of an infective discitis or an osteomyelitis. In addition, Mr Carter's back pain seemed to have been chronic, and had been recently exacerbated when he had experienced the clothes line incident on 31 May 2005. As to the loss or losses of consciousness that Mr Carter had experienced before he came into Dr Allcroft's care, he told me that he considered the probable causes to have arisen from a combination of the fact that the patient was septic and unwell, which could lower one's blood pressure, and that he had been a little dehydrated as evidenced by a renal function that had slightly deteriorated. Mr Carter had also taken benzodiazepines, had slept all day prior to his attendance at the physiotherapists and had then taken more Valium and Panadeine Forte. In addition, the pain that Mr Carter had experienced might also cause one to faint.

6.7. On 3 June 2005 Dr Allcroft saw Mr Carter at about 1pm after which he noted with question marks 'infection or degenerative' against the notation for pain. The following day, 4 June 2005, Dr Allcroft saw Mr Carter at about 8:30am. Again question marks were noted against notations in respect of Mr Carter's back pain, and again an acute injury or an infective process was queried as an explanation for that. Dr Allcroft noted on this occasion that he believed Mr Carter was suffering from 'presumed pneumonia, right lower lobe'.

6.8. On the same day at about 8:30pm, Mr Carter was seen by a Dr Tan. Dr Tan made certain entries into Mr Carter's clinical record. At the conclusion of Dr Tan's notes it is recorded that Dr Tan discussed the plan for Mr Carter's treatment with Dr Allcroft. Dr Allcroft acknowledged in his evidence that he had been in contact with Dr Tan at approximately 8:30pm on 4 June 2005. As Dr Tan has recorded in his note, Mr Carter had, for the first time, complained of some chest pain. This revelation was discussed with Dr Allcroft. It was felt that the pain may have been musculoskeletal and this was recorded in Dr Tan's notes to be its probable origin. The possibility of the existence of a dissecting thoracic aorta was at that point also considered as a possible source of the chest pain. Tests including a chest X-ray did not reveal the existence of the same and so that diagnosis, together with a possible diagnosis of a pulmonary embolus, were considered unlikely and were essentially ruled out. It is clear that Mr Carter did not have any such thoracic aortic dissection at that time. This chest X-ray would not have revealed the existence of a AAA which is situated in a different region of the aorta. The possibility of an abdominal aortic abnormality was not considered.

6.9. Dr Allcroft told me that in essence he had been reassured by Mr Carter's continuing improvement over this time. Among the improving features of his presentation were a fall in his C-reactive protein level, a normal haemoglobin reading which was consistent with not having bled since his admission, the return to levels approaching normality of the white cell count and the return to normal levels of parameters associated with renal function. Dr Allcroft summarised his thinking in these terms:

> 'It was just not on the radar at that time because everything had got better so it didn't point me in the direction of an aortic dissection, be it thoracic or abdominal at that time because it was all completely normal.' [4]

[4] Transcript, page 56

6.10. In fact the more recent chest X-ray of 4 June 2005 showed similar abnormalities to those that had been there previously, including basal consolidation. This was still in keeping with the diagnosis of an infective process that can take up to a month to resolve fully.

6.11. Dr Allcroft again saw Mr Carter on 5 June 2005 at about 12:30pm. As a result of Dr Allcroft's examination on this occasion, his belief that Mr Carter's chest pain was a result of a musculoskeletal was in his mind confirmed.

6.12. On 6 June 2005 Dr Allcroft saw Mr Carter at about 3:30pm. Mr Carter indicated that his back pain was easing and that he felt safe for discharge. On this particular occasion Dr Allcroft said that clinically, and on investigation, everything had returned to normal and was settling. The white cell count was resolving but not yet completely resolved because tissue repair can take up to a month to fully recover. However, Dr Allcroft formed the clear opinion that the infective condition had shown improvement as one would expect given Mr Carter's placement on an antibiotic regime.

6.13. Dr Allcroft went as far as to suggest that there had been negative indicators of a leaking or otherwise problematic AAA while Mr Carter was in his care. He told me that Mr Carter's abdominal examination that had been performed on a number of occasions was normal, that there had been no abdominal pain, that there had been no bruising or bleeding, and in particular no bruising seen along Mr Carter's flanks. As well, Dr Allcroft said that he would have expected Mr Carter's blood pressure to be falling over the period of time he was in the hospital, seeing a rise in his heart rate, as well as a general deterioration in his wellbeing if a leaking AAA had been present. He also said that if Mr Carter had been bleeding from a AAA, he would have seen his haemoglobin level fall as well as his blood pressure and renal function. Although Dr Allcroft was prepared to concede that clearly Mr Carter did have a AAA during the time that he was in hospital, Dr Allcroft did not believe that it had been bleeding and that the AAA was asymptomatic. Other indicia of the existence of a dissecting AAA would, according to Dr Allcroft, have included worsening pain, possible impaired blood supply to the intestines and abdominal pain. Many of these symptoms, I find, would not necessarily have been present if Mr Carter had been suffering from a leaking or dissecting AAA whilst in hospital. Given Mr Carter's level of obesity, it is unlikely that any manual manipulation would have detected a pulsatile mass. I did not understand Dr Allcroft in any event to be looking for one when he examined Mr

Carter's abdomen. Bruising would not necessarily have been evident, again because of the thickness of Mr Carter's abdominal wall. As to other clinical signs, I am persuaded that there would not necessarily have been any significant change in haemoglobin levels or renal function if the bleeding from the aneurysm had not been considerable. As far as abdominal pain was concerned, there does not appear to be any evidence that Mr Carter was experiencing the same whilst in hospital but it will be remembered that Mr Carter had reported abdominal pain to SAAS upon their attendance at the physiotherapist. The ambulance report reveals that Mr Carter's abdomen was found to be soft and tender. When Mr Carter first presented at the FMC Emergency Department it is also recorded that he complained of mild abdominal pain. However it will be remembered also that later at the Emergency Department he had denied chest and abdominal pain. This denial seems to have been what Dr Allcroft was informed of, or at least what he took on board. I would make the observation here that a denial of abdominal pain at a certain point in time could hardly be seen as refuting its earlier existence.

6.14. Although I take Dr Allcroft's views into consideration, his observations about what he believes were negative indicators of a leaking or dissecting AAA are made with the benefit of hindsight. The fact of the matter was that Dr Allcroft never performed any specific examination of Mr Carter with a possible AAA in mind. It simply did not occur to him that this was a possible diagnosis.

6.15. Dr Allcroft rejected suggestions made during the course of the Inquest, particularly those voiced by Professor Jamieson whose evidence I will come to in a moment, that a leaking or dissecting AAA should have been identified while Mr Carter was in Dr Allcroft's care. The reasons why Dr Allcroft would reject that suggestion I have already alluded to for the most part. The diagnosis that was on the table as far as Dr Allcroft was concerned, namely one of sepsis, was in Dr Allcroft's mind clearly supported by Mr Carter's clinical presentation, including of course the very significant raised neutrophil level with toxic changes indicating a severe infection. Dr Allcroft also pointed to the fact that Mr Carter was an elderly person with many comorbidities within which the presence of a AAA that was about to rupture would not have been obvious. As Dr Allcroft said 'there was a lot going on in this man'[5]. As far as Mr Carter's previous AAA was concerned, Dr Allcroft did not regard that as

[5] Transcript, page 74

a significant factor and it is true that a person who has experienced a AAA in the past does not necessarily retain a propensity to develop AAAs in the future. On the other hand, when asked in cross-examination whether he thought that Mr Carter's previous history of aortic aneurysm disease made it more likely that Mr Carter might experience another AAA in his lifetime, Dr Allcroft's response was 'there is the potential that it is more likely'[6].

6.16. Also in cross examination Dr Allcroft acknowledged the existence of certain symptoms that objectively might have been ascribed to a leaking or dissecting AAA, namely hypotension in the first instance. However, Dr Allcroft suggested that he would have expected a continual downward trend in Mr Carter's blood pressure that would not have corrected itself. The evidence before me suggests strongly that this in fact is not a feature of a leaking AAA, but that the drop in blood pressure can be transient and can return to normal levels. The deterioration in renal function that had also been detected in the first instance, whilst again a possible symptom of leaking or dissecting AAA, had to be seen, according to Dr Allcroft, in the context of other possible causes, including his cardiac function being impaired by sepsis. He was asked by counsel assisting, Dr Gray:

> 'Q. Those two factors combined with his history of an abdominal aortic aneurysm disease; was taking those factors combined together, not significant enough to raise in your mind at least the possibility of him having a ruptured or a leaking abdominal aortic aneurysm.
>
> A. Not at that time in that setting, given the other features.' [7]

6.17. Dr Allcroft agreed that the blood pressure of $^{90}/_{65}$ that had originally been measured by SAAS was hypotensive if it was a new thing for that person, bearing in mind that Mr Carter was normally hypertensive. The fact of the matter was that by any standard a figure of 90 was hypotensive even allowing for the fact that Mr Carter had controlled hypertension. However, there is no evidence that Dr Allcroft was ever made aware of that figure. The hypotensive episodes of which Dr Allcroft had been aware involved BP figures that were higher than that recorded by SAAS.

6.18. In cross examination Dr Allcroft agreed that although a thoracic aortic aneurysm had been excluded, there was some evidence of an abnormality as shown by the X-ray in respect of Mr Carter's thoracic aorta. He conceded that what had underlay Mr

[6] Transcript, page 88
[7] Transcript, page 95

Carter's original difficulty with his aorta in 1992 could still have been in existence in June 2005. This was to my mind a feature of Mr Carter's history and anatomical presentation that was of some significance. Although a second AAA in a person's lifetime is not the norm, the fact that he had a previous AAA in his lifetime, and that his thoracic aorta had a detected abnormality, were matters that, in assessing whether Mr Carter now had an aortic aneurysm, could hardly be put to one side. The difficulty was that the question as to whether Mr Carter currently had a AAA, or whether any of his symptoms on 1 June 2005 or later could be attributed to a AAA, was never asked.

6.19. Dr Allcroft rejected the notion that he should have performed a CT scan in relation to Mr Carter's abdomen. Although an abdominal CT scan on the evidence may well have been diagnostic of not only the presence of a AAA but of a leaking or dissection, Dr Allcroft said that an abdominal CT scan was simply not indicated at that time because there were no pointers to abdominal pathology. He went so far as to suggest that the administration of a CT scan may actually have worsened Mr Carter's condition because the contrast dye that is required for such a scan might have exacerbated Mr Carter's renal difficulties. Other evidence would suggest that this would not necessarily have been a real concern.

7. The autopsy and the evidence of Professor Roger Byard

7.1. Professor Byard performed the post-mortem examination of the deceased. The autopsy revealed a clear cause of death, namely the ruptured AAA. Professor Byard was of the view that the symptoms or indications that a AAA is about to rupture are sudden onset of severe abdominal or back pain, low blood pressure and collapse. Sometimes those symptoms can stabilise and indeed go away. Professor Byard was of the view that what had happened in Mr Carter's case was that there had been a small bleed before the fatal major bleed. Sometimes people can complain of chest pain or back pain or abdominal pain. The symptoms could vary as could the presentations of different patients.

7.2. Professor Byard suggested that the appearance of Mr Carter's aneurysm was that of a typical atherosclerotic aneurysm. He did not believe it to be an infective aneurysm and found no evidence of infection such as pus when he examined it. While acknowledging that there had been a clinical history of sepsis, that is to say Dr Allcroft's diagnosis of it, Professor Byard did not find any significant sepsis at

autopsy. In particular, there was no obvious pneumonia. Professor Byard suggested that he would have been surprised if there had been a significant infection, although he acknowledged that treatment with antibiotics can cause a very quick and favourable response to an infection. He also acknowledged the possibility that Mr Carter may have had a heavy bronchitis and pneumonia when he first came to the hospital. He said:

> 'I suppose that's possible but I think the clinical assessment would be the correct way to look at that and the chest X-ray to see if there was pneumonia on the chest X-rays.' [8]

7.3. Professor Byard told me that symptoms and signs associated with cardiovascular disease can come and go.

7.4. Professor Byard's evidence would support the suggestion that the symptoms associated with a leaking AAA might be transitory, and may well in Mr Carter's case have reflected an early leak that then stabilised before finally rupturing. This in turn could support the suggestion that on 1 June at the physiotherapist Mr Carter was indeed experiencing a leaking AAA. Professor Byard's evidence was also supportive of the proposition that there may have been an infective process at some stage, but tended to refute the likelihood that a severe infection such as pneumonia or sepsis had been present. However, Professor Byard's evidence did not dispose of the contention that while Mr Carter had been in Dr Allcroft's care there had been clinical signs and symptoms of an infective process at work.

8. The evidence of the experts

8.1. I received the reports of, and heard evidence from, two experienced medical practitioners whom I regarded as experts in their fields. Those practitioners were Professor Glyn Jamieson and Dr Wilson Braund. Professor Jamieson is currently an Oesophageal Surgeon. He has been a consultant surgeon since 1974 and practices at the Royal Adelaide Hospital. He was a full-time vascular surgeon until 1990. It is in respect of his qualifications and experiences as a vascular surgeon that Professor Jamieson's opinions have been expressed. Dr Braund is a physician in both the general and endocrine areas. As a general physician he has encountered acutely ill patients with aortic aneurysms and because of his endocrine/diabetes practice he has encountered patients with vascular disease. From time to time he also acts as the duty

[8] Transcript, page 299

doctor at the Ashford Hospital for all emergency general medical admissions. He obtained his specialist qualifications as a physician in 1985.

8.2. As earlier foreshadowed, there was some marked difference in opinion between Professor Jamieson and Dr Braund. The difference can be summarised by stating that Professor Jamieson was firmly of the view that Mr Carter's presentation was indicative of a problematic AAA. Professor Jamieson expressed the opinion that Mr Carter was suffering from a leaking AAA at the time of his episode at the physiotherapists and that his symptomology reflected this. Professor Jamieson was of the view that a leaking AAA or a stretching AAA was a differential diagnosis that was on the table and should have been considered and at the very least an abdominal CT scan should have been administered to explore that possibility. It is fairly clear that an abdominal CT scan would have been diagnostic of the existence of a AAA. Professor Jamieson was of the belief that Mr Carter's symptoms, particularly the back pain and hypotension, were symptomatic of the AAA.

8.3. On the other hand, Dr Braund told me that although he agreed that an aneurysm was no doubt present, a AAA would not have been high on his differential diagnosis list. He was of the view that Mr Carter's signs and symptoms were all in keeping with explanations other than a AAA. Dr Braund expressed the view in his report[9] that he did not think Mr Carter had been suffering a subacute rupture of the aorta or a leak at the time of his Emergency Department presentation on 31 May 2005 and at the time of his hospital admission and discharge. He believed that the terminal event of the aortic rupture was extremely sudden and was unrelated to his recent hospital presentation with sepsis – even though the process of rupture must having been taking place at that time. Dr Braund suggested that if he had been responsible for Mr Carter's management it would have mirrored that of Dr Allcroft.

9. <u>The evidence of Professor Jamieson</u>

9.1. Professor Jamieson provided two reports to the Inquest[10]. In his first report of 10 October 2005 he expressed the view that there were enough pointers in the case of Mr Carter to suggest that a CT scan should have been carried out. Those pointers were his known aortic aneurysmal disease, his back pain associated with a collapse, his hypotension and tachycardia on admission and a disordered renal function.

[9] Exhibit C7f
[10] Exhibits C8 and C8a

9.2. In particular, Professor Jamieson stated in his report and indeed in his evidence that he has worked off an adage, which he passes on to his medical students, that a patient with back pain and hypotension has a leaking aneurysm or a dissecting aortic aneurysm until proven otherwise. In other words, until a patient has had an abdominal CT scan to exclude those conditions, their existence should be assumed out of an abundance of caution. In his second report dated 28 July 2006, in which he was asked to comment upon the statement made by Dr Allcroft to the investigating police officers, Professor Jamieson expressed the view that given the setting of a patient with known aneurysmal disease, two episodes of fainting and an admission to hospital with back pain, a recorded hypotension and tachycardia, the likelihood was very high that the aneurysm was stretching or leaking at the time Mr Carter was admitted to FMC. Pain of that nature is quite common in the days and weeks leading to an aneurysm actually rupturing. A CT scan may have revealed no abnormality other than the AAA itself, but it might also have revealed a leakage that had already occurred. It is worth observing here that even if a CT scan had revealed the existence of a AAA, but not necessarily a leaking AAA, the very existence of the aneurysm could have led to suspicion that Mr Carter's symptomatology, or some of it, was ascribable to the aneurysm. If the leak had been actually seen on the CT scan it is certain that Mr Carter would have been referred to a vascular surgeon. I deal separately with the question as to whether or not such referral and any surgery undertaken by a vascular surgeon would have been successful.

9.3. In his evidence Professor Jamieson elaborated upon his opinions. He added that one factor that had to be seriously considered was the importance of excluding the most serious possible cause of a medical problem, and in particular, causes which might lead to death. This meant that even though the pre-test probability of a diagnosis may be low, it is, in modern times, a relatively simple matter to obtain a CT scan. In these circumstances, pre-test probability does not have particular relevance in Professor Jamieson's view. I can readily understand Professor Jamieson's line of thinking. Even though there may be a low pre-test probability of a potential diagnosis, this has to be weighed against the seriousness of the potential diagnosis and its possible consequences in terms of death or serious illness. I accept Professor Jamieson's evidence that low pre-test probability is not the be-all and end-all of a diagnostic exercise.

9.4. In his evidence Professor Jamieson also placed into proper context Mr Carter's previous history of aortic aneurysm. Whilst it is true to say that it is unusual for someone to have a second aneurysm in his or her life, it would be more common for that to occur than in a person that had never had one in the first place and that this is a matter to be taken into consideration as well.

9.5. Professor Jamieson expressed the belief that Mr Carter's collapse at the physiotherapist's rooms was most likely caused by a leaking AAA, even allowing for the fact that perhaps dehydration, his consumption of diazepam, his age and comorbidities could have been a cause. Nevertheless, Professor Jamieson said it was important to have identified the cause for his low blood pressure. He said 'when you're hypertensive, whatever the cause, you need to exclude the most serious cause'[11].

9.6. The fact that Mr Carter's blood pressure returned to normal levels was not necessarily indicative that the original drop in blood pressure had not been as a result of a leaking AAA. There are reasons why low blood pressure could return to normal following a leak. Professor Jamieson thought that the low blood pressure of $^{90}/_{65}$ recorded by SAAS was consistent with a leak. Professor Jamieson said:

> 'Well I guess I come from my vascular background. I acknowledge that. But nevertheless to me it is a very typical scenario for the way a leaking aneurysm presents and to me looking at that when you look at the ambulance notes there, the man has had some back pain and abdominal pain they note. He's hypotensive. He's had previous aortic aneurysmal disease. Its something that should have been thought of I think.' [12]

9.7. Professor Jamieson agreed with the proposition that in coming to any diagnosis one needed to take into account the observations recorded in the ambulance record. To him all of what had been recorded here was obvious relevant history that would need to be considered. It is worthwhile observing that much of Mr Carter's presentation as observed and recorded by SAAS on Professor Jamieson's analysis would be classical symptomology of a leaking AAA. There was a significant hypotension of $^{90}/_{65,}$ two episodes of loss of consciousness together with abdominal pain and back pain. One would have thought that this, together with his previous history of AAA, would have at least raised the possibility of a leaking AAA. The difficulty of course was that this scenario was not presented to Dr Allcroft. What was presented to Dr Allcroft by the

[11] Transcript, page 130
[12] Transcript, page 139

FMC Emergency Department was a picture of an infective illness. But in reality there had been no such suggestion upon Mr Carter's presentation to the FMC Emergency Department in the first instance. What was in existence at that time was a fairly strong picture of a possible AAA. It may well be that that was the point in time at which the possible diagnosis of a AAA should have been actively considered. As Professor Jamieson said, it was a very typical scenario for the way a leaking aneurysm presents. It is for my part difficult to see how at that point in time the possibility of a leaking AAA could have been overlooked, or at least not placed among a list of differential diagnoses. It was a possible diagnosis that ought to have been explored at that stage if for no other reason than to eliminate it. As it transpired, a CT scan would undoubtedly have shown, at the very least, the existence of a AAA.

9.8. However, Professor Jamieson added this:

> 'I think it's certainly true that he had been assessed, the patient had been assessed, in the emergency department and by the time Dr Allcroft was called in I guess the acuteness of the situation had diminished somewhat because he's gone through the emergency area. So I think that's probably true.' [13]

9.9. Professor Jamieson disagreed with Dr Braund's assessment that Mr Carter's terminal event of an aortic rupture had been extremely sudden and unrelated to his recent hospital presentation with sepsis. He was of the view that the clinical scenario had been very typical of a leaking aortic aneurysm that finally ruptured.

9.10. As to the suggestion made in the evidence that a CT scan using contrast dye may have pushed Mr Carter over the edge into overt renal failure, Professor Jamieson agreed with the proposition that where there is a need for a diagnostic procedure, a judgment has to be made weighing the possibilities of adverse consequences of that procedure against the need for the diagnostic procedure to be carried out. He added that a CT scan could be administered without the administration of contrast dye and that it would have at least picked up the existence of the AAA in any event. Although without the dye it is more difficult to detect whether there is a leak involved, it would still have indicated the existence of the aneurysm, an important diagnostic fact in itself given Mr Carter's presentation. In any event Professor Jamieson was of the view that given the degree of renal dysfunction that Mr Carter had exhibited, he believed that the radiologists probably would still have administered the contrast dye.

[13] Transcript, page 142

9.11. Professor Jamieson conceded that Mr Carter's back pain may have been the result of a back condition, but expressed the view that it was more likely that the symptoms were associated with the AAA[14].

9.12. Professor Jamieson expressed the view that was not challenged that there is a 100% mortality rate with a leaking aneurysm.

9.13. Professor Jamieson told me that he thought Mr Carter may well have had an upper respiratory tract infection at the same time. He said that the acuity of Mr Carter's difficulty with his AAA had diminished somewhat by the time that Dr Allcroft had come on the scene. There had been by then a possible diagnosis of an infective illness, Mr Carter's blood pressure had recovered and the pain seemed to have dissipated. The possibility of the existence of the AAA as accounting for Mr Carter's presentation was more evident at the Emergency Department than it was when Mr Carter's case was presented to Dr Allcroft.

9.14. Professor Jamieson dealt with the possibility of the infective process and the fact that there had been indications of the same when the case was being considered by Dr Allcroft. Professor Jamieson thought that the elevated white cell count was consistent with the leaking AAA, although it is also indicative of a very wide range of conditions. However, the observed toxic changes in the elevated white cell count he agreed certainly pointed more towards 'an infective type of situation'[15]. He suspected however that toxic granulation could also be explained by inflammatory conditions rather than infective conditions. I inferred from this answer that Professor Jamieson was by no means sure about that and I would prefer the evidence of other experts where they might conflict with Professor Jamieson's on that issue. Indeed, the existence of toxic changes in addition to the elevated white cell count to my mind was highly consistent with an infective process at work and in those circumstances it is not surprising that Dr Allcroft viewed them accordingly.

9.15. Professor Jamieson in cross examination on behalf of Dr Allcroft was not entirely convinced that Mr Carter had in fact been suffering from a significant infection. He believed that many of the findings which were regarded as infective could have been due to the aneurysm. He conceded however that the toxic granulation within the white cell count was, to his mind, 'a little unusual'. Later in his cross examination,

[14] Transcript, page 157
[15] Transcript, page 168

however, Professor Jamieson indicated that he was prepared to agree from reading the hospital records that Mr Carter did indeed have a septic illness. He was not prepared to agree that it was necessarily serious, but agreed that there appeared to have been some improvement in the septic illness in the course of Mr Carter's hospitalisation. Further, Professor Jamieson was prepared to accept that Mr Carter probably had an illness within his airways. He suggested that pneumonia was too strong a diagnosis, but conceded that he had infective signs within his chest.

9.16. In response to the suggestions that the episode of loss of consciousness at the physiotherapist's rooms could have been the result of underlying dehydration or sepsis, Professor Jamieson expressed the view that while both scenarios were possible, they were unlikely. In support of that opinion Professor Jamieson suggested that there was no good reason to suppose, given Mr Carter's lifestyle, that he would have been dehydrated. I add here that there appears to be no evidence of any dehydration as such and the suggestion of dehydration does seem to be based very much on supposition as Professor Jamieson would suggest. As to sepsis being the possible cause of the syncopal episode, Professor Jamieson said that it would have to be a very severe degree of sepsis to cause that. Professor Jamieson was of the view that there was no evidence to suggest that there was very severe sepsis. Rather he had a slight elevation in his white cell count which was in keeping with the pathology revealed in his lungs. However, it will be remembered that it was not simply a slight elevation of the white cell count which existed in Mr Carter's case. There was evidence of toxic changes which in fact did rather suggest a septic illness or an infection of some severity. On the other hand, as already seen, Professor Byard could find no evidence of any severe septic or infective pathology.

9.17. Professor Jamieson's view about the matter can be summarised as follows. While he was prepared to agree that Mr Carter's case was difficult to assess and treat[16], and that with his medical history and presentation it would have been difficult to arrive at a conclusion that a AAA was present[17], he was of the belief that people who have back pain and hypotension always should be investigated for aneurysmal disease regardless of what their history has been[18]. He suggested that Mr Carter's experiences and

[16] Transcript, page 190
[17] Transcript, page 188
[18] Transcript, page 180

presentation were typical of the majority of people who ultimately die from a AAA. Professor Jamieson was asked this question by me:

> 'Q. Can I ask you this: if you were in the emergency department or in any of the shoes of these practitioners who saw him on 1 June and you suspected a triple A, or even suspected a leaking triple A, and even if you didn't think that that was one of the differential diagnoses but you suspected it, you had a feeling about that because of certain signs and symptoms whilst possibly being consistent with some other pathology, were also consistent with a triple A, leaking or otherwise, how would - in the circumstances of this patient, given his obesity - how would you lay those suspicions or your unease about triple A to rest.
>
> A. A CT scan.' [19]

Professor Jamieson was clearly of the view that there ought to have been a sense of unease about Mr Carter's presentation on 1 June 2005, particularly in the Emergency Department. Even if a AAA was not part of the differential diagnosis but was merely suspected, he agreed that those concerns should be laid to rest and the appropriate method of so doing would be to conduct a CT scan. The difficulty with this however, was that there is no evidence that anyone in the Emergency Department at the FMC considered the possibility of a AAA pathology and by the time the case was presented to Dr Allcroft it was a case of a different complexion altogether such that a diagnosis of AAA did not enter Dr Allcroft's head at any time.

10. The evidence of Dr Braund

10.1. In his oral evidence, Dr Braund elaborated upon his reports. There were two matters which together, in his opinion, pointed strongly to a significant inflammation and indeed sepsis in Mr Carter. The strikingly high white cell count, in the vicinity of 18,000 which in itself Dr Braund said would indicate a very severe infection was one matter. As well, the C-reactive protein level represented 'statistically an enormous deviation from normal'[20]. Added to those two factors was the fact that, according to the laboratory test of 2 June 2005, toxic changes were observed. Dr Braund told me that these changes were seen only in the most severe cases of inflammation and was evidence of a severe illness, almost certainly sepsis. This assertion remained for the most part unchallenged during the course of the Inquest. I did not understand any of the medical practitioners who gave evidence to seriously dispute Dr Braund's proposition, based as it was upon the existence of toxic granulation within the

[19] Transcript, page 201
[20] Transcript, page 235

neutrophils. In addition, there was no evidence, or at least no persuasive evidence, that toxic granulation would be seen as a feature of the inflammation that could be explained by the existence of a leaking or dissecting AAA. Indeed, Dr Braund went so far as to say that the proposition that an elevated white cell count and C-reactive protein level during the rupture of an aneurysm, whilst a well documented reaction, was somewhat of a revelation of Dr Braund. However, Dr Braund pointed out that when one encounters a very high white cell count, as was the case here, the prospect of it being attributed to some disease other than sepsis diminishes. Very high white cell counts he said were almost invariably reflective of a bacterial sepsis and in this context he repeatedly emphasised that toxic granulation in his experience was reported only in the most extreme cases of bacterial sepsis, as I say, a proposition not seriously challenged. It will be remembered that Professor Jamieson's assertion that one might encounter toxic granulation even in inflammatory conditions other than infective conditions was based on suspicion only. Be that as it may, the fact of the matter was that by the time Dr Allcroft came into Mr Carter's case, Mr Carter's presentation by then was very much in keeping with his having an infection. In addition, in attributing the toxic granulation to an infective process or an inflammatory process, there was also the fact that the X-ray report of 1 June 2005 described a lung pathology that was possibly infective in nature especially given the clinical history.

10.2. Dr Braund would not necessarily have attributed Mr Carter's signs and symptoms to the existence of a leaking or dissecting aortic aneurysm. For example, he would have regarded the back pain as a matter that would be difficult to regard as diagnostic. It was chronic pain that had been going on for a number of months and he had recently had an acute episode of pain when hanging out the washing. On the other hand, he agreed that the abdominal pain may have supported a conclusion that there was some rupturing process at work during his hospitalisation. Dr Braund would have regarded dehydration as a reasonable explanation for the low blood pressure. As well, there was the sepsis, Mr Carter's age and his anti-hypertensive treatments possibly at work. Dr Braund also thought that Mr Carter's earlier symptoms may have been connected with the taking of diazepam. He said that in Mr Carter's age group, bacterial sepsis is almost invariably accompanied by low blood pressure, or by lowering of their normal blood pressure. However, I did not understand Dr Braund to be disputing the

suggestion that many of Mr Carter's symptoms could be ascribed to a leaking or dissecting AAA.

10.3. Dr Braund was of the view that the difficulty with the AAA only appeared in hindsight and there was no point at which its existence was becoming obvious. One factor involved in that assessment was the fact that Mr Carter's laboratory signs and clinical appearance improved. Nevertheless, and importantly in my opinion, Dr Braund said that the possibility of a AAA would fit 'in every good doctor's differential diagnosis of any severely ill patient'[21], although in this case it would have been well down in his list. Dr Braund seems to have regarded the possibility of a AAA as low if not trivial[22]. He described it as a 'possibility with low absolute risk'[23]. A diagnosis of sepsis had come to the fore very quickly because of the test results, but I did not understand Dr Braund to be saying that the possibility of a AAA would be entirely ignored. If that is Dr Braund's position then I would reject that. I would also reject the suggestion that the possibility of a AAA was trivial. The fact that it could be regarded as a possibility at all, given its possible serious consequences, was a matter that in my opinion could have been given serious consideration, particularly at the Emergency Department level. On this aspect of the matter I prefer the evidence of Professor Jamieson that the question of pre-test probability is a factor of relative unimportance when the consequences of a misdiagnosis are to be considered. When asked in cross examination whether it would have been an error for the possibility of a AAA not to be included somewhere in the differential diagnosis when Mr Carter was at Flinders Private, Dr Braund said:

> 'It should be somewhere in the list, but Malaria can cause back pain and abdominal pain and a low blood pressure and a low fever. You have to put your bets in the right spot.' [24]

10.4. Dr Braund seems to be suggesting there, and elsewhere, that it would have been dangerous to:

> 'Start putting bets on a diagnosis connected with an aortic aneurysm because to do so would possible involve a distraction from proper diagnostic procedures.' [25]

10.5. For my part, it is difficult to support a lot of what Dr Braund is advocating here. Once a potential diagnosis of a leaking or dissecting AAA forms part of the differential

[21] Transcript, page 273
[22] Transcript, page 273
[23] Transcript, page 290
[24] Transcript, page 273
[25] Transcript, page 242

diagnosis, the preferred line of thinking would have to be that of Professor Jamieson, namely that because the consequences of a missed diagnosis are so serious, the possibility of a leaking AAA needs to be explored by proper diagnostic means which would include an abdominal CT scan.

10.6. Dr Braund rejected the notion that a CT scan should have been administered in Mr Carter's case. He also rejected as a universal proposition Professor Jamieson's adage that a AAA should be presumed in a person presenting with back pain and hypotension, especially where the presenting symptoms pointed in entirely different direction as they did in this case in his opinion.

11. Conclusions as to whether the AAA should have been diagnosed

11.1. There is no question but that Mr Carter had a AAA on 1 June 2005. While I do not think that Mr Carter was experiencing any symptoms of his AAA on 31 May 2005 when he was hanging out the washing, in my view it is more probable than not, and I so find, that on 1 June 2005 at his physiotherapist's rooms he experienced an episode that was symptomatic of his AAA. In my view, the evidence of Professor Jamieson that this was so symptomatic is to be preferred. Mr Carter was recorded by SAAS personnel as experiencing both back and abdominal pain – his abdomen was soft and tender. There was much debate during the evidence as to whether a leaking AAA might cause back or abdominal pain. The fact of the matter was that at that stage Mr Carter had both. There were his two episodes of syncope that were experienced virtually simultaneously with pronounced hypotension. The hypotension proved to be transient which in Dr Jamieson's opinion is in keeping with the fact of a leak. He told me, and I accept his evidence in this regard, that when patients leak with their aneurysm they often experience fainting episodes caused by a sudden drop in blood pressure which then recovers. In my opinion the evidence demonstrates that these symptoms were more likely than not the result of a leaking AAA. I am impressed with Professor Jamieson's evidence that this constellation of signs and symptoms is typical of what might be experienced by someone suffering a AAA at some time prior to a fatal rupture. He told me, and he was unchallenged on this, that the majority of aneurysms go through the scenario that Mr Carter experienced. I accept all of that evidence. It is unlikely, therefore, that Mr Carter before his death would have experienced no signs or symptoms that were connected to his AAA. Dr Allcroft and Dr Braund would have me conclude that Mr Carter's symptoms, at whatever time

they were experienced, were attributable to an infective illness in their entirety and that Mr Carter prior to his death experienced no symptoms of significance that were related to his AAA. I reject that unlikely scenario. At some point in time it is more likely than not that Mr Carter would have experienced some heraldic signs of his AAA rupturing. The question would therefore need to be asked, when did Mr Carter experience those symptoms? The answer in my view lies in the events of 1 June at the physiotherapist's rooms. To my mind, Professor Byard's evidence also supports the notion that Mr Carter's symptoms were, at least initially, due to a leaking AAA. I reject the contention that the lack of post mortem evidence that might have revealed an earlier leak meant that there had been no leak. I am satisfied that any such evidence would likely have been obscured by the later more copious bleeding that undoubtedly took place upon rupture. In the event, the only aspect of Mr Carter's clinical presentation that the autopsy would bring into question would be the existence of a severe infection or sepsis prior to his death.

11.2. When Mr Carter arrived at the FMC Emergency Department it is likely that at that time he was still experiencing symptoms connected with his AAA. It may well be that in the ensuing days the leak resolved and as a consequence, some of the symptoms associated with the leaking AAA subsided. I include here the resolution of his hypotension. There can be little doubt that while Mr Carter was a patient in Flinders Private he also had an infective illness that was to become the focal point of his treatment.

11.3. There is no documented evidence that within the FMC Emergency Department consideration was given to the possible existence of a leaking or dissecting AAA. Certainly no diagnostic measures were undertaken in order to confirm or refute that kind of pathology. There is no note in any of the FMC Emergency Department records of any possible diagnosis connected with a AAA.

11.4. It was unfortunate that a CT scan was not conducted with respect to Mr Carter in order to explore the possibility of a AAA. It would certainly have revealed the AAA and it very possibly would have revealed a leak. However, in my view for reasons that follow it cannot be said that any omission is to be sheeted home to Dr Allcroft.

11.5. Mr Carter was placed in the care of Dr Allcroft who was a physician. Mr Carter was transferred to the Flinders Private where he remained until his discharge on 6 June

2005. I find that at no time during the period between his admission and his discharge from Flinders Private did Dr Allcroft consider the possibility that Mr Carter was experiencing signs or symptoms of AAA pathology. In short, the case was for the most part presented to Dr Allcroft as one involving possible infection, namely possible pneumonia. This was very much the flavour of discussions that were held between Emergency Department personnel and Dr Allcroft when consideration was being given to Dr Allcroft's taking over Mr Carter's clinical management. However, based upon the evidence of Dr Braund, in my view this fact did not absolve Dr Allcroft of all diagnostic responsibility. The fact of the matter was that Flinders Private was placed in possession of all of the written material that had been gathered in respect of Mr Carter since 1 June 2005. This included the SAAS notes. The clinical picture revealed in the SAAS notes, as well as the notes of Mr Carter's first examination at the FMC Emergency Department, was suggestive of AAA pathology. However, the fact remains that there were very strong indications upon which Dr Allcroft, by the time he came to be involved in Mr Carter's management, could reasonably make a diagnosis of an infective illness. In particular there was the high white cell count, the high C-reactive protein count and above all the existence of toxic granulation within the neutrophils. The toxic granulation was suggestive of a severe inflammation if not infection. Although Dr Allcroft did not give consideration to the existence of a AAA, based as it might have been upon an assessment of Mr Carter's earlier symptomatology as described to SAAS, it was not unreasonable for Dr Allcroft to focus his attention on a possible infective illness. When the evidence is properly evaluated, in my view no criticism attaches to Dr Allcroft and his management of the deceased. I accept Dr Allcroft's evidence, as supported by Dr Braund, that there was a strong clinical picture of an infective process at work. Although the autopsy did not reveal any evidence of a severe infective illness or sepsis, I do not understand the post mortem evidence to exclude the possibility that at some point in time there had been a severe illness. The toxic granulation within Mr Carter's neutrophils to my mind is strong evidence that there had been an infective or inflammatory process that, upon a proper diagnostic evaluation, would not have been ascribed to a leaking or dissecting AAA. It was more in keeping with an infective or inflammatory illness. The fact that Mr Carter improved in the next few days was also indicative of the fact that there was an infective illness that was improving with antibiotic treatment.

12. <u>Would Mr Carter have survived if his AAA had been diagnosed</u>?

12.1. Mr Carter was not subjected to an abdominal CT examination. If he had been, there is little doubt that the AAA would have been revealed. In those circumstances, it is almost unthinkable that the existence of the AAA would not have been thought to be associated with Mr Carter's earlier symptoms such as his abdominal pain, syncopal episodes and low blood pressure. It is difficult to envisage the existence and diagnosis of a AAA being ignored in these circumstances. Certainly if the CT had revealed a leaking AAA, and that is a distinct possibility, the primary focus upon Mr Carter's management would have been that leaking AAA. I accept the evidence that was unchallenged during the course of the Inquest that a leaking AAA will inevitably, in time, lead to a rupture and death. Either way, it seems clear that Mr Carter would have been referred to a vascular surgeon for evaluation.

12.2. What were Mr Carter's chances of survival? Professor Jamieson, a former vascular surgeon, told me that surgery designed to repair the leaking aneurysm would almost certainly have led to his death. He expressed a number of reasons for so saying. Among them was the fact that for the entire duration of any such surgery, Mr Carter's kidneys would not be receiving a blood supply. In Professor Jamieson's view the chances of his kidneys recovering from such a sustained insult were low. In addition, Mr Carter's general poor health and comorbidities would not have augured well for a successful outcome. If Mr Carter had been afflicted with an infective process as well, this would be an additional factor weighing against a satisfactory outcome.

12.3. Professor Jamieson referred to the possibility of stenting as an alternative to surgery. Professor Jamieson had little or no practical experience himself of such a procedure. However, he expressed the view that an attempt at stenting would probably have been preferred over surgery because of an extremely poor surgical prognosis. Professor Jamieson summed up the matter by saying that an attempt at stenting might have presented Mr Carter with some chance of survival whereas with a leaking aneurysm for which nothing is done, there is no chance of survival.

12.4. Dr Braund shared Professor Jamieson's view that an elective repair of the aneurysm involved a high chance of Mr Carter dying. Dr Braund told met that for him the biggest indicator that Mr Carter would probably not survive surgery was his chronic obstructive airways disease.

12.5. As far as stenting is concerned, Dr Braund was quite pessimistic as to whether or not this procedure would work in Mr Carter's case. Dr Braund referred to the fact that Mr Carter had a previous repair to his aorta. Any stenting that would take place would have to be delivered through the repaired section. This would probably give rise to difficulty. In addition, Dr Braund was not convinced that there would be in any case enough room to enable a stent to be anchored within the aorta.

12.6. Dr Braund also referred to the fact that if patients were considered too ill for open repair, such that the surgeon would not contemplate it, the patient could well be in the situation where they would also not be suitable for a stent procedure either. Nevertheless, Dr Braund suspected that in all of Mr Carter's circumstances if a leaking aneurysm had been detected, a vascular surgeon may well have 'gone for broke' with an open aneurysm repair[26].

12.7. The evidence would suggest that Mr Carter's chances of surviving an attempt to surgically repair a leaking AAA would have been poor. In addition, it can never be known whether or not stenting would in any sense have been feasible as an alternative to repair. It will be remembered that Mr Carter had general health problems and a number of comorbidities that would not have helped his situation. Thus it is that if diagnostic measures had been undertaken to detect a AAA that was leaking, or an aneurysm that had leaked, a satisfactory surgical outcome for Mr Carter was not guaranteed.

13. Recommendations

13.1. Pursuant to section 25(2) of the Coroner's 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.

13.2. As seen, this was quite a difficult case. Dr Braund pointed out that several medical practitioners whose competence would not be brought into question did not undertake any diagnostic measures in respect of a AAA. Further, it would appear that nobody even considered it as a possible diagnosis. And yet, as I find, at one point in time there had been in existence a leaking AAA in respect of which there had been a number of significant pointers. When Mr Carter was seen by SAAS at his physiotherapist's rooms and was first seen in the FMC Emergency Department on 1

[26] Exhibit C254

June 2005, the signs and symptoms of a possible leaking AAA were less ambiguous than they became later on. To my mind, this is a case that the medical profession worthy of close study. It may well be that even experienced Emergency Department personnel and even physicians require some further education in relation to the signs and symptoms of AAA pathology where there are other competing diagnoses available. It would seem to me that once a leaking or dissecting AAA is suspected, and once it is to be accepted that it will inevitably be fatal, it is a diagnosis that must be very firmly towards the top of the list, and not the bottom of the list of possible diagnoses. In that event, medical practitioners would be well advised to take the necessary diagnostic steps if for no other reason than to eliminate AAA pathology.

13.3. I would recommend the following:

1) That the Department of Health and the Medical Board of South Australia bring the details of this case to the attention of the medical profession, especially those who perform diagnostic tasks such as Emergency Department personnel and physicians;

2) That the Department of Health and the Medical Board of South Australia undertake measures to educate the medical profession in respect of the signs and symptoms relevant to a diagnosis of leaking or dissecting AAA and to impress upon the medical profession the need to undertake the necessary diagnostic measures in respect of a suspected AAA.

Key Words: Hospital Treatment; Misdiagnosis; Emergency Departments

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 22nd day of August, 2008.

Deputy State Coroner