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 October 2012, the 1st day of November 2012, the 12th and 13th days of June 2013 and the 15th day of August 2013, by the Coroner's Court of the said State, constituted of Mark Frederick Johns, State Coroner, into the death of Anthony Douglas Stock.

The said Court finds that Anthony Douglas Stock aged 64 years, late of Lot 11 Yumali Road, Meningie, South Australia died at the Flinders Medical Centre, Flinders Drive, Bedford Park, South Australia on the 15th day of December 2009 as a result of hypoxic-ischaemic encephalopathy complicating cardiac arrest due to retroperitoneal and pelvic haemorrhage complicating pelvic fractures. The said Court finds that the circumstances of his death were as follows:

1. Introduction and cause of death

1.1. Anthony Douglas Stock died on 15 December 2009. He was 64 years of age. An autopsy was conducted by Dr John Gilbert who reported[1] that Mr Stock's cause of death was hypoxic-ischaemic encephalopathy complicating cardiac arrest due to retroperitoneal and pelvic haemorrhage complicating pelvic fractures, and I so find.

2. Background

2.1. At approximately 5pm on 9 December 2009 Mr Stock was working on his property at Meningie when he was thrown from a horse. He fell onto his right hip and, although he was alone at the time, he was able to make a call on his mobile phone to the

[1] Exhibit C2a

ambulance service. He was transported to the Meningie Hospital and from there to the Flinders Medical Centre by helicopter. He arrived at the Flinders Medical Centre Emergency Department shortly before midnight. A CT scan of his abdomen and pelvis was undertaken and it confirmed that he had a comminuted right pelvic fracture involving the acetabulum as well as the superior and inferior rami.

2.2. In the early hours of 10 December 2009 Mr Stock was examined by orthopaedic registrar, Dr Johnson. Dr Johnson noted that there was a fracture of the right acetabulum and the iliac wing of the pelvis. He said that there were many fracture lines. Mr Stock was wearing a pelvic binder which had been applied by the paramedics. Dr Johnson removed it as he did not think it was necessary, the fracture being a lateral compression injury not requiring such a device. Dr Johnson said that he had expected that there would be some bleeding as a result of the injuries suffered by Mr Stock. The CT revealed a small retroperitoneal haematoma. He explained that this is a bleed outside the abdominal cavity. Blood tests taken at the time reveal that Mr Stock's haemoglobin level was 126 grams per litre which Dr Johnson regarded as good following the type of injury that Mr Stock had sustained. As a result it was Dr Johnson's view that Mr Stock was haemodynamically stable and he arranged for Mr Stock to be admitted to the Orthopaedic Department on an orthopaedic ward. A urinary catheter was inserted and Dr Johnson ordered routine neurovascular observations to be performed regularly.

2.3. Mr Stock's first day on the orthopaedic ward was relatively uneventful. He was seen by the colorectal team and later by the orthopaedic team. He experienced several bouts of pain during the day when adjustments were being made to his traction but with morphine and endone the pain abated.

2.4. On 11 December 2009 Mr Stock was seen by the orthopaedic team on a ward round. Dr Shahrokhi was an orthopaedic registrar on Ward 5C[2] that day. He explained that the routine on the ward was that there would be a daily meeting called a trauma meeting which included all doctors from consultants to interns at which patients would be discussed and plans made for their treatment. Following that the ward rounds would occur. Typically, the ward rounds would include a registrar, a resident medical officer, an intern and an orthopaedic nurse. Dr Shahrokhi did not specifically remember the ward round of 11 December 2009 but he is recorded in Mr Stock's

[2] The orthopaedic ward

notes as having led the ward round on that day[3]. The plan as noted after the ward round on that day states 'seeking advice from RAH surgeon re ? fixation'. Dr Shahrokhi explained that this suggested that the plan was to seek advice from an orthopaedic specialist at the Royal Adelaide Hospital as to whether it was necessary to operate or treat conservatively. The note went on to say 'anticoagulation as per pelvic protocol'. Dr Shahrokhi explained that there was a protocol for treatment for deep vein thrombosis (DVT) prophylaxis. He said that it had a specific provision dealing with pelvic fractures which required 48 hours of mechanical prophylaxis with no medication. Then, if the patient were haemodynamically stable, the patient would be treated with clexane followed by warfarin from day 7. Dr Shahrokhi said that protocols such as this are quite different from hospital to hospital. He explained that there was no consensus in the medical literature for appropriate DVT prophylaxis for this sort of injury. He acknowledged that it was probably he that gave the direction for commencing the prophylactic anticoagulation in accordance with the protocol. He was asked if he was aware that although a blood test had been taken for Mr Stock on admission, no further blood tests were taken until after Mr Stock's collapse on the ward on the evening of 12 December 2009. Dr Shahrokhi said that he would have expected that a further blood test or tests would have been taken on Friday morning, 11 December or Saturday morning, 12 December 2009 at the latest. Dr Shahrokhi did not see Mr Stock again prior to his collapse on the evening of 12 December 2009. Although he had said that he had no specific memory of that ward round, Dr Shahrokhi was asked what guidance he would give to an intern who had been instructed to apply the prophylactic DVT protocol. He responded by saying that he would ask them to calculate the patient's weight and provide an appropriate dose by reference to the protocol. He said:

> 'In hindsight it's easy to say. I may have suggested that a blood test be done but I don't have a clear recollection of whether I did that or not.' [4]

He said that the conduct of a blood test is 'such a routine investigation' and it is done as a matter of daily care for patients, even patients who are stable. He said that he would have expected that it would have been performed and would not have expected to have given any prompting. Dr Shahrokhi said that the protocol for prophylactic DVT treatment at that time implicitly required that there be blood tests[5]. He referred

[3] Exhibit C18, page 54
[4] Transcript, page 62
[5] Transcript, page 65-67

to that part of the protocol which requires the adjustment of the dosage of enoxaparin[6] if creatinine clearance is less than 30mls per minute, once daily. He explained that in order to make sure that you were giving the dose therefore, you need to perform the blood test to check the creatinine level[7]. Dr Shahrokhi was asked whose task it was to order the blood tests. He explained that it was the job of the intern[8]. Furthermore, he said that he would expect that the intern who was going to institute the protocol would.

2.5. Dr Alecia Macrow was the intern who was present on that ward round and it was she who made the entry in Mr Stock's hospital notes recording the outcome of that ward round. She is now a general practitioner registrar. She gave evidence that she had been on her orthopaedic rotation at Flinders Medical Centre for some 6 weeks when she became involved with Mr Stock. Dr Macrow presented as a very honest and forthright witness. She could recall some events quite clearly, although not full details of others. She gave evidence that she accompanied Dr Shahrokhi on that morning ward round on 11 December 2009. Dr Shahrokhi was leading the ward round. Dr Macrow said that Dr Shahrokhi asked her to start the protocol using clexane[9]. Dr Macrow said that she had no previous experience of this particular procedure. Dr Macrow said that she was 'quite anxious' because she had never seen or used this part of the protocol before and so she asked Dr Shahrokhi for some clarification on how he wanted her to write it up. She said that he just said 'quite bluntly' to get the protocol and follow it[10]. Following the ward round Dr Macrow obtained the protocol and tried to familiarise herself with it. Because she had not used it before and was a bit nervous about it she spoke to the ward pharmacist and checked her calculations with him to make sure that they got it right[11]. Following this she duly gave the necessary orders for the administration of enoxaparin correctly in accordance with the protocol[12]. Dr Macrow was asked about that part of the protocol which dealt with adjusting the dosage of enoxaparin by reference to creatinine clearance levels. She asked how she addressed that issue and explained:

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[6] The anticoagulant to be administered under the protocol
[7] Transcript, page 66
[8] Transcript, page 68
[9] Clexane is another name for enoxaparin
[10] Transcript, page 103
[11] Transcript, page 104
[12] Transcript, page 104-105

> 'Yes it was, with the pharmacist. Essentially that is to be flagged for any level of kidney impairment, that the dose would need to be adjusted, and it wasn't felt that he had any level of kidney impairment.' [13]

2.6. Dr Macrow referred to checking the blood test which had been taken the previous day which did include creatinine. It was apparent from this answer, and a further answer given in cross-examination[14], that Dr Macrow interpreted the protocol at the time as requiring only that the creatinine level be checked before institution of the regime. She did not understand that it was implicit in the protocol that there was a need to check the creatinine clearance level daily to ascertain whether there were any changes occurring, with a view to adjusting the dosage throughout the duration of the treatment. I should point out that the protocol does not specifically make it clear that the blood tests must be taken daily and the creatinine levels checked accordingly, and the enoxaparin dosage adjusted if necessary, all on a daily basis. Dr Macrow was at the time the most junior of doctors and it is entirely understandable that she would not have understood this even though to Dr Shahrokhi it was, to use his expression, implicit. Dr Macrow did not feel that she was adequately supervised in relation to this issue[15].

2.7. Dr Macrow had one further interaction with Mr Stock on 12 December 2009. She explained this in her evidence. The ward round for that day was again recorded by Dr Macrow[16]. However, Dr Macrow had not actually attended the ward round that day. She explained the circumstances in which she came to make the note in her evidence. She explained that she was working with another intern by the name of Dr Thomson and she was assigned what she described as the outlying patients while Dr Thomson was assigned the ward patients including Mr Stock. Dr Thomson accordingly was present on the ward round. Because Dr Macrow's round was finished first she approached Dr Thomson and asked if she could assist her completing her duties. Dr Thomson accepted her assistance and as a result it was Dr Macrow who made the record of what had transpired at Dr Thomson's ward round. Dr Thomson explained to Dr Macrow that everyone on the ward was well and that the registrar, Dr Lars Schmitt, had seen them with her. Dr Macrow then went to see Mr Stock herself and wrote in his notes. She said that she remembered that occurrence pretty well and that

[13] Transcript, page 104
[14] Transcript, page 123-124
[15] Transcript, page 125
[16] Exhibit C18, page 56

she thought that Mr Stock looked well when she made the note at 10:30am on 12 December 2009. She said that Mr Stock talked to her and made eye contact. They discussed whether he was in pain and he said he had been put on a bed pan in order to attempt to use his bowels. As a result of this there had been increased pain but he was more comfortable when lying still. Dr Macrow looked at Mr Stock's charts which she thought looked okay. She listened to his chest and discussed his pain. That was her last involvement with Mr Stock. Dr Macrow said that Mr Stock's family were not present when she saw him on this occasion.

2.8. Dr Lars Schmitt gave evidence at the Inquest. He is an Orthopaedic Fellow at the Flinders Medical Centre and was one of the senior registrars in December 2009. Dr Schmitt had no memory of Mr Stock whatsoever and was dependent on Mr Stock's medical notes for any present appreciation of the events of his case. He had checked his records with the pay office at Flinders Medical Centre and established that he was on leave until 10 December 2009. He said that he was on-call on 11 December 2009 and that if he had been called in he would have participated in the handover on the morning of 12 December 2009. However, the handover would not have involved any patient contact. Furthermore, it was his evidence that had he been involved with Mr Stock on that day, it was his belief that on returning to work the following week the cardiac arrest that struck Mr Stock on the evening of 12 February 2009 would most certainly have been mentioned, and would have been an event that would have stood out in his mind. The implication of his evidence was that he certainly was not convinced that he was involved in Mr Stock's treatment. However, he was not prepared to rule out the possibility that he was.

2.9. It will be apparent from the narrative so far that the medical records for Mr Stock do not list Dr Schmitt as being present at the ward round on 12 December 2009. They only refer to Dr Macrow who was not present. It was Dr Macrow's evidence however that she believed from a conversation with Dr Thomson that Dr Thomson had attended on that ward round. She also believed from what Dr Thomson conveyed to her that Dr Schmitt was the registrar who led the ward round on that day. However, Dr Macrow had no personal knowledge of Dr Schmitt's presence on the ward round that day. I will come to Dr Thomson's evidence shortly. As I say, Dr Schmitt would not go so far as to rule out the possibility that he was present on the ward round that

day[17]. Dr Schmitt's evidence was that it would have been desirable for another blood test to have been taken to check Mr Stock's haemoglobin levels[18].

2.10. Dr Schmitt's evidence raised another matter which I will return to later in this finding.

2.11. Dr Thomson gave evidence. She is now an acting registrar at the Flinders Medical Centre. Dr Thomson confirmed that she was an intern in the Orthopaedic Department in late 2009. She did recall Mr Stock. She was present on the ward round on 10 December 2009 which was the first day Mr Stock was on the ward. As I have already said, nothing particularly significant occurred on that day and Mr Stock's prophylactic DVT treatment had not commenced at that point. Dr Thomson said that the notes did not disclose that she had any further involvement with Mr Stock's care. She said that although she had no recollection of this having occurred, she thought that it was entirely possible that Dr Macrow's version of the events was correct. In other words, that she, Dr Thomson, had been present on the ward round that day. She also accepted that it was possible that the senior person on that ward round was Dr Schmitt, although she had no recollection. She said that interns never did ward rounds by themselves and that there would have always been a registrar or more senior person than that present on the ward round[19].

2.12. The evidence in relation to the ward round of 12 December 2009 is unsatisfactory. The notes for that day were taken by a doctor who was not present on the ward round, and not present to observe the leader of the ward round and the person who must therefore ultimately bear responsibility for what occurred on that ward round. The person who made the note, Dr Macrow[20], made the note on behalf of Dr Thomson. However, Dr Thomson now has no recollection of who the registrar on that ward round was. It was Dr Macrow's recollection that Dr Thomson informed her that it was Dr Schmitt. However, Dr Schmitt now has no recollection of having been present and indeed thought that in light of what happened to Mr Stock, he would have had a clear recollection, but he was unable to rule out that it was indeed he who was present. All in all this is a very poor state of affairs and leads to a lack of accountability for the medical treatment of Mr Stock on that occasion.

[17] Transcript, page 141-142
[18] Transcript, page 137
[19] Transcript, page 215
[20] Of whom I make no criticism for this

2.13. It would appear that although Mr Stock collapsed later that night, at the time of the ward round on the morning of 12 December 2009 it was not possible from his clinical appearance to predict what was likely to occur that night. Nevertheless, it would have been evident on the morning of 12 December 2009 that no creatinine clearance information was available in relation to Mr Stock and that therefore it would not have been possible to adjust his enoxaparin dosage by reference to creatinine clearance rates. Furthermore, no blood test had been undertaken with a view to measuring his haemoglobins, and it was the evidence of both Dr Schmitt and Dr Shahrokhi that blood tests would be a routine event. Therefore an opportunity was missed at the ward round of 12 December 2009 for a senior doctor to detect that routine bloods had not been taken and as a result creatinine clearance levels could not be detected (determined) and haemoglobin levels could not be measured. It was possible, had that issue been addressed on that morning, that a blood test might have revealed something that would have indicated what was going to transpire that night. However, it is not now possible to determine what any such blood test might have revealed. It is clear however that an opportunity that may have benefited Mr Stock, and may have prevented the tragic outcome, was indeed missed.

2.14. This Inquest has considered the possibility that high levels of enoxaparin in Mr Stock's bloodstream may have contributed to his bleeding which in turn led to a hypovolaemic state, which caused his cardiac arrest. Certainly, that was the opinion of Professor Cade[21] who said:

> 'His excessive anticoagulation was misguided and almost certainly contributed substantially to his fatal bleeding.'

However, Dr Schmitt proffered another explanation. Dr Schmitt offered the theory that the bleeding that led to Mr Stock's hypovolaemia was arterial bleeding from an internal iliac artery which would have led to very rapid blood loss[22]. He said that it could also have occurred a few days later[23]. He said that would have explained why the deterioration happened so suddenly[24]. Dr Schmitt explained further that if this theory were correct, the enoxaparin anticoagulant would not have made a significant contribution to the event. He said that it was possible that Mr Stock might have been tolerating the enoxaparin quite well and that the arterial bleed was simply a result of

[21] Exhibit C30
[22] Transcript, page 135
[23] Transcript, page 135
[24] Transcript, page 135

the original injury[25]. Dr Schmitt postulated that fracture fragments may have shifted and that they could have either caused arterial damage at the time of the original accident which might have then clotted but moved with a vasospasm, or may have been affected by movement on the ward or in the bed on the ward and may have dislodged and caused the arterial bleeding[26].

2.15. I turn now to consider the charted observations for Mr Stock on 12 December 2009. These are to be found at page 102 of the notes[27]. Observations were recorded for 0030, 0625, 1100 and 1600 hours on that day. The observations for the first three times were steady with acceptable levels of respiration, blood pressure and oxygen saturations of 95% or higher. The observations at 1600 hours that day show that the respiratory rate increased to 24 breaths per minute, that the blood pressure has increased from around $^{138}/_{70}$ earlier in the day to $^{154}/_{87}$. Furthermore, the saturations of oxygen on room air had dropped to 92% at that time. The fluid balance chart demonstrates that Mr Stock was continuing to pass urine and take fluids throughout the day, certainly until 1700 hours[28]. The evidence of the nursing staff who saw him that day was that apart from complaining of pain, they did not note any matters of concern. It was their evidence that severe pain when traction changes occurred or when the patient was moved for any other reason, were not out of the ordinary in pelvic fracture patients[29].

2.16. On the other hand, Mr Stock's wife said that at approximately 3:30pm that day when she arrived to see her husband, he looked 'terrible'. She said that he was pale and perspiring and that his skin smelt acidic. She said he did not look good and that he was in a lot of pain. Furthermore his stomach had bloated and the bloating was quite hard[30]. Mrs Stock said that she raised this matter with a nurse who did not appear to be concerned and attributed the bloating to the high dose of morphine and the fact that he was lying on his back. Mrs Stock also gave evidence that she went to the nurses' station and asked when a doctor would be coming. She said that the nurse at the station informed her that there was nothing they could do because Mr Stock was already on the highest level of morphine and that 'his doctor was on an emergency

[25] Transcript, page 147
[26] Transcript, page 160
[27] Exhibit C18
[28] Exhibit C18, page 98
[29] See the evidence of Nurse Collis
[30] Transcript, page 169-170

call'[31]. In correspondence sent by Mrs Stock to the Patient Safety Coordinator at Flinders Medical Centre in June 2010 the following is recorded:

> 'As my daughter, Katie, passed the nurses' station, on her way out of the hospital (she stood at the desk for quite some time, before she was even acknowledged), she said to the nurse that Doug's call bell had been rung and that he was in excruciating pain. Why did the nurse then look back at her monitor and say "oh yes, I'll get to that in a minute" and carry on at the computer? Katie left the hospital, disgusted with the treatment her father was getting.'

And further:

> 'He was extremely pale, perspiring profusely (cool cloths were being placed on his forehead!!) felt clammy, his skin smelt acidic, his stomach was bloated, he was in extreme pain and was very agitated.'

And further:

> 'Doug had asked, several times that day, to see a doctor (as did I when I arrived) why did one NOT come?' [32]

2.17. Mrs Stock had also written to the Director of Nursing on 27 January 2010 as follows. She said that when she arrived at 4pm she was shocked at Mr Stock's condition. She said:

> 'His colour was ghostly white – he was normally a suntanned farmer. He felt clammy … he was perspiring profusely … cool cloths on his forehead to cool him down.'

And further:

> 'He had asked earlier for a doctor to see him but was told that his doctor had been called out on an emergency. About 4:30 I asked the nurse at the desk again if a doctor was coming and that Doug had asked earlier, but I was given the same answer. I retorted with "and there is only one" but she just seemed to ignore that.' [33]

2.18. Mrs Stock said in her evidence that she left the hospital at approximately 5pm because Mr Stock had said to her that he was in too much pain to talk to her and he would try and handle the pain by himself.

2.19. None of the nursing staff recalled this interaction. In fact, it was the evidence of the nursing staff that had these symptoms been reported to them, they would have recorded them in the notes. No such notes were recorded. In summary, apart from

[31] Transcript, page 171
[32] Exhibit C31
[33] Exhibit C31

the observations made by Mr Stock's family members, it would appear that there were no particular concerns by any of the nursing staff that afternoon. The evidence does seem to suggest that Mr Stock was reasonably well in the morning and this is supported by the helpful recollections and convincing sincerity of Dr Macrow's evidence. I am concerned about the suggestions of Mr Stock's family that his condition may have been deteriorating in the afternoon and that there is no objective evidence to support this other than the beginning of a possible deterioration in the nursing observations for 1600 hours that afternoon.

2.20. Mrs Stock's evidence was not challenged by counsel for Flinders Medical Centre and the nursing staff. I have no reason to doubt the truth of her claims. Unfortunately, it is now not possible to identify with confidence the particular staff members with whom Mrs Stock and her daughter interacted that afternoon.

2.21. What is known is that Mr Stock suffered a cardiac arrest sometime before an MET call was made at 2205 hours on the night of 12 December 2009. On the arrival of the MET he was noted to be in asystolic cardiac arrest and was not resuscitated until he had suffered 20 minutes of downtime. Furthermore, he was not seen by a doctor at any time following Mrs Stock's requests that he be seen by one that afternoon and prior to his cardiac arrest. Indeed, the last time he was seen by a doctor prior to his MET call was at approximately 10:30 that morning when he was seen by Dr Macrow. I accept Dr Macrow's evidence that at that time he was in no discomfort.

2.22. On the basis of the observations made by Mrs Stock and her daughter, I find that Mr Stock was pale and clammy and perspiring and in excruciating pain at sometime between 3:30pm and 4pm. I find that Mr Stock and Mrs Stock separately made requests of staff that a doctor be called but that this did not happen. I find that Mr Stock's daughter, Katie, did approach a member of the nursing staff at the nurses' station to enquire why Mr Stock's call bell had gone unanswered as described in Exhibit C31.

3. The significance of the nursing note at 2000 hours

3.1. A nursing note at 2000 hours on 12 December 2009 contains the following:

> 'Foot pumps in situ c ✔ w ✔ m ✔ s R) foot.[34]'

Nurse Collis interpreted the note in her evidence. She said it indicated that neurovascular observations had been done in respect of the right foot, namely that colour, warmth, movement and sensation were all present[35]. Reference was also made to this observation in the evidence of Nurse Gobin[36] but she was vague and uncertain about the significance. I prefer the evidence of Nurse Collis.

3.2. It had occurred to me that this note may have tended to contradict Mrs Stock's observation that Mr Stock was pale, referring as it does to normal colour by means of the letter C with a tick immediately after it. However, it is clear from the passage of evidence of Nurse Collis referred to above that the observation was made in relation to his right foot only. Furthermore, it was never suggested by any other witness, expert or otherwise, or in submissions, that the observation related to his general appearance rather than specifically to his foot.

4. Dr Sandhu's evidence

4.1. The Court obtained an expert report from Dr Avinder Sandhu[37]. Dr Sandhu is a consultant radiologist with the Royal Adelaide Hospital. Significantly, he set up the Interventional Radiology Unit at the Royal Adelaide Hospital in the 1980s. That unit is a unit which performs angiography with a view to embolising bleeding points without the need to perform surgery. Indeed, angiography was performed upon Mr Stock immediately after the CT scan in the early hours of 13 December 2009 following his collapse, but was not successful in locating the site of the bleed.

4.2. Dr Sandhu very helpfully explained to the Court the significance of the differences between the first CT scans done upon Mr Stock on 10 December 2009 and those which were done in the early hours of 13 December 2009 following his collapse. Dr Sandhu noted in the first set of scans that he had suffered a comminuted fracture and

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[34] Exhibit C18, page 57
[35] Transcript, page 83
[36] Transcript, pages 198-199
[37] Exhibit C28

that the whole of the acetabulum had shattered[38]. He contrasted this scan with the second set of scans. The first of those was done without contrast. That scan revealed what Dr Sandhu described as a 'collection' sitting in the retroperitoneal region and extending all the way down to the pelvis, an abnormality that was not there two days before[39]. He said that the collection was probably blood and that he would call it a haematoma or a blood clot[40]. He said that it conservatively was measured in the pelvis at 13.5cm in the sagittal plane and 8cm in the coronal plane[41]. Following that scan a further scan was done using contrast. Dr Sandhu said this revealed:

> '… you can see a trickle of contrast is extending straight through into that large cavity. That is actively bleeding at the time of the CT scan.' [42]

Dr Sandhu explained that the 'trickle of contrast' indicated active bleeding into the collection which he had early described.

4.3. Dr Sandhu helpfully showed the Court a comparison of the CT scans side by side using the Court's audiovisual display. He showed the scan of 10 December 2009 and the scan of 13 December 2009 side by side and commented:

> 'In essence the bit of bone that has moved in between those two sides is this large segment of bone at the back here. This bone has now moved away from the side of the hip and lies very close to the area of bleeding that I showed you just now.' [43]

And:

> 'In those two days … since the last scan … one bit of bone … has now tilted and is medially displaced. So its sharp edge here now lies intimately close to this large haematoma.' [44]

He described it as a bit of bone that had tilted and had a triangular sharp pyramidal edge which lies close to the large haematoma at the site where the area of bleeding was occurring. Dr Sandhu said that the displacement of the piece of bone was a distance of 13mm into the pelvis[45]. Dr Sandhu said that from this he concluded that an artery had been damaged by the movement of the bone[46]. Dr Sandhu agreed with

[38] Transcript, page 234
[39] Transcript, page 236
[40] Transcript, page 237
[41] Transcript, page 238
[42] Transcript, page 239
[43] Transcript, page 240
[44] Transcript, page 240-241
[45] Transcript, page 241
[46] Transcript, page 241

the opinion expressed by Dr Gilbert in the post-mortem report[47] that the bleeding was most likely from a branch of the right internal iliac artery[48].

4.4. Dr Sandhu noted that by the time of the autopsy the collection which he had noted in the second of the CT scans had reached the level of the diaphragm. However, at the time of the second CT scan on 13 December 2009 it had reached only as high as the right kidney[49]. Clearly, the bleeding continued after Mr Stock's collapse and this accounts for the differences between the size of the collection as revealed on 13 December 2009 and the size of the collection as revealed at autopsy.

4.5. Dr Sandhu commented that he himself has frequently performed angiography in pelvic fractures that are actively bleeding to embolise them. He remarked that a small artery like the one that was noted to be actively bleeding in Mr Stock's case could be repaired but added:

> 'But the proviso with that is that you need good blood that clots.' [50]

He described the artery as having a diameter of between 2mm and 5mm[51].

4.6. Dr Sandhu was asked if he could offer an opinion as to the time over which the collection that he observed in the second set of CT scans might have been accumulating. His response was that he could not do so, that it would be pure conjecture and that all he could say was that at the time of the CT scan there was active bleeding[52]. Dr Sandhu added that it could have happened within the previous 20 minutes, or on the other hand that it could have happened intermittently over quite some time[53].

5. <u>The evidence of Dr Allcock</u>

5.1. Dr Allcock provided an expert report for the Court[54]. He is a consultant orthopaedic surgeon working at the Lyell McEwin Hospital and the Women's and Children's Hospital. Dr Allcock's opinion was that the observations at 1600 hours on 12 December 2009, to which I have earlier made reference, were significant. He

[47] Exhibit C2a
[48] Transcript, page 243

[49] Transcript, page 242
[50] Transcript, page 245
[51] Transcript, page 245
[52] Transcript, page 252
[53] Transcript, page 253
[54] Exhibit C29

attached significance to the fact that the oxygen saturations had dropped to 92% which he believed was an indication that Mr Stock was bleeding[55]. He commented that the blood pressure at that time was not abnormal but that the peak blood pressure does not drop until a very significant amount of blood has been lost because the body is very good at adopting compensatory mechanisms for making the best of the blood that is still left[56]. Dr Allcock was also influenced by the clinical signs reported by Mrs Stock of clamminess at 4pm. He said he thought it was clear that something was happening that needed closer observation[57].

5.2. Dr Allcock did not agree with the suggestion of Dr Schmitt that the bleed may have happened very suddenly, immediately prior to the collapse. He said that he did not agree with that proposition because arteries, if cut, will often go into spasm and limit the blood loss. He remarked that venus bleeding is in many ways more troublesome[58]. It was Dr Allcock's opinion that at approximately 4pm a doctor should have been called to Mr Stock. His view was that the doctor should then have examined Mr Stock and would have noted that he was cold and clammy in the peripheries as reported by Mrs Stock. That would be a sign of shock that would then lead to investigations for both pulmonary embolus and, if that were excluded, bleeding[59]. Dr Allcock said that had he been called at 4pm that day he would have sought to exclude pulmonary embolus and if that was negative he would have then looked for another cause. He said:

> 'I would have stayed trying to find out what was going on until I found something.' [60]

6. The evidence of Professor Cade

6.1. Professor Cade provided an overall expert report for the Court[61]. Professor Cade is the Principal Specialist in Intensive Care at the Royal Melbourne Hospital and a Professorial Fellow in the University of Melbourne. He was the Director of Intensive Care at the Royal Melbourne Hospital for over 30 years. His first doctoral thesis was in the discipline of anticoagulation and he has done much subsequent research work

[55] Transcript, page 270
[56] Transcript, page 271
[57] Transcript, page 273
[58] Transcript, page 281
[59] Transcript, page 286
[60] Transcript, page 287
[61] Exhibit C30

and publication in the field of thrombosis in general. He was Head of the Anticoagulant Clinic at the Royal Melbourne Hospital for many years[62].

6.2. Professor Cade was critical of the anticoagulation protocol at Flinders Medical Centre for orthopaedic patients, at least as it applied to pelvic fractures. He explained the differences between prophylactic and therapeutic anticoagulation and said that a patient such as Mr Stock would be placed on a prophylactic dose of an anticoagulant such as enoxaparin[63] after the first day or two when he was stable from the point of view of bleeding. The typical prophylactic dose would then be enoxaparin 40mg per day and in addition the patient would have mechanical assistance such as a foot pump or stockings[64]. He said that with enoxaparin it is not necessary to carry out APTT tests but that clinical monitoring was very important. He said that it was important to look for signs of thrombosis and that is best done by examining the legs to see if there is swelling or discomfort[65]. On the other side of the coin it is necessary to consider the risk of bleeding. For that purpose he said it was important to track the haemoglobin levels and pulse, blood pressure, pallor and ensure the clinical findings were in order and stable[66]. Professor Cade said that the benefit of regular tracking of haemoglobin is to give an idea of whether or not there is ongoing bleeding and to give an idea of the extent of the initial bleeding and provide a baseline against which to assess whether there is any further bleeding over the course of the patient's admission[67]. Professor Cade noted that other than the admission bloods for Mr Stock which were analysed for haemoglobin, there were no further haemoglobin results. Professor Cade regarded that as an oversight[68].

6.3. Professor Cade said that he thought that the Flinders Medical Centre orthopaedic protocol for pelvic fractures was flawed in that instead of prescribing 40mg a day[69], Mr Stock was prescribed 100mg twice per day which Professor Cade said was a very large dose. He said that it is a full anticoagulant dose which would be given to a person with an existing thrombosis[70]. It was his view that such a dose carries a very high risk of haemorrhage and would only be given to someone who was

[62] Transcript, pages 296-297
[63] Also known as Clexane
[64] Transcript, page 302
[65] Transcript, page 304
[66] Transcript, page 305
[67] Transcript, page 305
[68] Transcript, page 305
[69] The usual preventative dose
[70] Transcript, page 305

haemostatically intact and would never be used for prevention because the risks were too high[71]. Professor Cade noted that Dr Macrow followed the protocol correctly. However, his criticism was of the protocol itself. Professor Cade said that he has never seen any literature supporting such a dose in this setting[72]. It was Professor Cade's opinion that it was inconceivable that the dose prescribed to Mr Stock did not place him at risk of major bleeding[73]. Professor Cade acknowledged that daily haemoglobin testing would not have picked up an acute bleed on 12 December 2009 but it would have shown the extent of the original bleed and provided a baseline against which to measure what was happening thereafter. Professor Cade was asked to comment on Dr Schmitt's theory that the cause of the bleed was the penetration of an artery by a piece of bony fracture. He responded:

> 'I think it's quite likely that the point of a bone moving as Dr Schmitt has said is very likely to be correct. That's what pelvic fractures do.' [74]

6.4. Professor Cade's comments about the dose rates of enoxaparin were supported by Dr Allcock's evidence. Dr Allcock also commented that the most common prophylactic dose of enoxaparin would be 40mg daily[75]. Dr Allcock believed that it would have been more likely that Mr Stock's bleeding would have been able to stop if he had been on a dose of 40mg daily[76]. Dr Allcock thought that on the balance of probabilities the dose of enoxaparin which was prescribed to Mr Stock contributed to the fatal bleed[77].

6.5. Professor Cade's view about the nursing observations at 1600 hours on 12 December 2009 differed from that of Dr Allcock. Professor Cade did not think that they were particularly different from the earlier vital observations. Professor Cade's opinion was that the observations made by Mrs Stock that Mr Stock was pale and clammy was the significant factor[78]. He said that these symptoms can be caused by other things, but it may be an early sign of blood loss. He said that skin changes occur well before haemodynamic changes and that changes in the skin precede changes in blood

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[71] Transcript, page 306
[72] Transcript, page 308
[73] Transcript, page 308
[74] Transcript, page 310
[75] Transcript, page 276
[76] Transcript, page 280
[77] Transcript, page 294
[78] This difference between the opinions of Professor Cade and Dr Allcock does not create a dilemma for me. The fact is that both the observations of Mrs Stock and the nursing observations occurred at around 4pm. Staff should have been alerted to the problem either from their own observations of Mr Stock, or as a result of Mrs Stock's pleas for a doctor to be called.

pressure[79]. He said that the paleness and clamminess should have been a flag that should have resulted in the following steps:

1) An assessment of the patient;

2) The taking of blood tests;

3) More frequent observations;

4) The insertion of an intravenous line in place ready for resuscitation if needed;

5) If there was a strong suspicion of a bleed a repeat CT scan.

6.6. Professor Cade said that if active bleeding is identified the blood loss has to be replaced and the circulation supported and then there has to be an attempt to stop the bleeding. He noted that bleeding from a pelvic fracture is notoriously difficult to control. Professor Cade noted that the bleed that occurred on 12 December 2009 was a very big haemorrhage and it was the cause of Mr Stock's cardiac arrest. Professor Cade said that he thought it incontrovertible that Mr Stock arrested as a result of hypovolaemia[80]. It was Professor Cade's view that Mr Stock's death was preventable up until the time of his arrest or shortly before[81]. He noted that it is a reasonably straightforward thing to enhance or increase the circulating blood volume, thus preventing the hypovolaemia that precipitated the cardiac arrest[82]. In short, it was Professor Cade's view that Mr Stock's condition was reversible 'well into the evening until shortly before he arrested'[83].

6.7. Professor Cade noted that the original protocol, of which he was as I have noted critical, has now been altered. He regards the protocol now as being exemplary[84]. It was altered following Mr Stock's death.

6.8. Dr Tamblyn, who was a staff orthopaedic surgeon who saw members of Mr Stock's family after his death, stated:

> 'I advised the family that the enoxaparin dose used for pelvic fractures is higher than the dose normally used for prophylaxis. I explained that this dosage would have contributed

[79] Transcript, page 313
[80] Loss of blood volume, Transcript, page 314
[81] Transcript, page 314
[82] Transcript, page 315
[83] Transcript, page 317
[84] Transcript, page 316

to the death. I am basing this contributing factor to enoxaparin contributing to more severe bleeding than would have occurred if enoxaparin was not given.' [85]

6.9. Dr Wiersema was an interventionist who treated Mr Stock. He confirmed that the cardiac arrest was attributed to hypovolaemia from the bleeding[86]. He had a conversation with the Stock family which he recorded in the notes[87]. In his statement he said that he told the family words to the effect 'this shouldn't have happened'. He said that he was wanting to empathise with the family. He said that it was unusual for death to result from the type of injury Mr Stock had sustained and he wanted to reassure the family that the hospital wanted to have the matter properly investigated.

7. Conclusion

7.1. In conclusion, I consider that Mr Stock's bleeding had commenced sometime prior to 3:30pm to 4pm on 12 December 2009. The cause of the bleeding was the movement of a sharp bony fragment that had been displaced as part of the original injury. It was likely caused to move by the requirements of daily nursing such as pressure area care and hygiene requirements and the use of a bedpan. The movement of a bony piece from these activities was to be anticipated. Mr Stock was on a high dose of enoxaparin, it being a therapeutic rather than prophylactic dose. This made the bleeding more difficult to control by the body's natural responses than it would have been had a prophylactic dose been in place. Nevertheless, the signs and symptoms were apparent in Mr Stock's pallor and clamminess from sometime around, or shortly before, 3:30pm to 4pm. Both Mr Stock and Mrs Stock separately requested the attendance of a medical practitioner but these requests were ignored by nursing staff and not acted on. No medical practitioner attended Mr Stock prior to his collapse. Had Mr Stock been medically assessed at any time after approximately 3:30pm to 4pm that day it is my opinion that he would have been placed on a higher level of observations and that investigations would have commenced to find an explanation for the clinical signs of early shock. There was a significant amount of time between 4pm and shortly before 10pm when he was found to be in cardiac arrest during which, with IV therapy to support his blood volume, his condition could have been retrieved.

7.2. In my opinion the Minister for Health direct that Flinders Medical Centre carry out enquiries with a view to determining why the staff on duty on the afternoon of 12

[85] Exhibit C8a, page 3
[86] Exhibit C5a
[87] Exhibit C18

December 2009 failed to respond to Mr and Mrs Stock's requests that a medical practitioner be summoned.

7.3. The enquiries should attempt to establish the identity of the nursing staff who failed to respond to Mrs Stock's pleas for a doctor to be called. If that staff member is identified he or she should be required to explain why a doctor was not called. Unless staff are held accountable in such situations there is every likelihood that they will repeat poor practices and behaviours. This case can only be explained by incompetence or, worse still, lack of care.

7.4. In my opinion had a doctor attended upon Mr Stock that afternoon at around 4pm his death would most likely have been prevented.

8. Recommendations

8.1. 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.

8.2. I recommend that the Minister for Health direct that Flinders Medical Centre carry out enquiries with a view to determining why the staff on duty on the afternoon of 12 December 2009 failed to respond to Mr and Mrs Stock's requests that a medical practitioner be summoned.

8.3. I recommend the enquiries should attempt to establish the identity of the nursing staff who failed to respond to Mrs Stock's pleas for a doctor to be called. If that staff member is identified he or she should be required to explain why a doctor was not called.

Key Words: Anticoagulation Therapy; Inadequate Examination

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 15th day of August, 2013.

State Coroner