



Western

Australia

RECORD OF INVESTIGATION INTO DEATH

Ref No: 31/2012

*I, Alastair Neil Hope, State Coroner, having investigated the death of **Wendy May ASLETT**, with an Inquest held at Perth Coroners Court on 21 September 2012 and 5-6 December 2012 find that the identity of the deceased person was **Wendy May ASLETT** and that death occurred on 29 October 2010 at Royal Perth Hospital as a result of Multiple Organ Failure Following Haemorrhage from Penetration of the Femoral Artery in the following circumstances -*

Counsel Appearing :

Sgt L Housiaux assisted the State Coroner

Mr Dominic Burke (Clayton Utz) appeared on behalf of Dr Alida Lancee

Mr P Tottle (Tottle Partners) appeared on behalf of Dr Naga Gunjikunta

Ms Nikita Barsby (Lynn & Brown Lawyers) appeared on behalf of the family.

Mr Quail appeared for Dr Deague

Mr Bourhill appeared for Professor Playford

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INTRODUCTION

Wendy May Aslett (the deceased) was a 60 year old female who died at about 12.40pm on 29 October 2010.

Following the death a post mortem examination was conducted on 3 November 2010. On 3 December 2010 after obtaining results of further investigations the forensic pathologist, Dr C T Cooke, concluded that the cause of death was multiple organ failure following haemorrhage from penetration of the external iliac artery in association with coronary arteriosclerosis.

It was clear as a result of investigations into the circumstances of the death that the deceased died as a result of bleeding resulting from a coronary angiography procedure which she underwent at St John of God Hospital, Subiaco, on 21 October 2010.

A coronary angiography is a procedure in which a special x-ray of the heart's arteries is taken to see if they are narrow or blocked. It is a test used when a doctor suspects or knows a patient has coronary heart disease.

During a coronary angiography the patient is given a local anaesthetic and then a catheter (a long thin tube) is put into an artery in the groin (as in this case), or at the inside of the elbow or near the wrist. The catheter is moved



up the inside of the artery until it reaches the heart. A special dye is then injected into the coronary arteries and x-rays are taken. The x-ray image (a coronary angiogram) gives detailed information about the state of the heart and coronary arteries.

Information provided to the deceased described the process of an angiography in essentially the above terms and also advised that, as with many medical tests “there are some risks, but serious problems are rare”.

In this case it is clear that after the procedure there was bleeding from the site of the puncture where the catheter was put into the artery. That bleeding caused the death.

On 27 October 2010 when the deceased was taken to Armadale Hospital the extent of the bleeding was such that Professor Playford, the consultant cardiologist who had conducted the angiogram on 21 October 2010, was “shocked and upset”¹. Professor Playford considered that the extent of the bleeding was such that she had a “massive haematoma”² and at one stage there was effectively no blood pressure. The deceased’s condition was not survivable and in spite of considerable efforts made to save her which included transferring her to Royal Perth Hospital,

which took place at around 12pm, she died on 29 October 2010.

The result of the angiogram was that no thrombus or other life-threatening cardiac condition was detected.

The family of the deceased raised a number of concerns in relation to her treatment and were particularly concerned that she had died as a result of what was an exploratory procedure.

A further concern expressed by the family was the fact that the deceased had presented to her general practitioner, Dr Alida Lancee, on 25 October 2010 at a time when she was complaining of considerable pain and there was already a very extensive haematoma, but Dr Lancee did not refer her to a cardiologist or take immediate steps to transfer her to hospital, but provided her with opioid pain relief and sent her home, to be seen again on 27 October 2010.

On 27 October 2010 the deceased's condition was non-survivable.



THE CORONARY ANGIOGRAPHY OF 21 OCTOBER 2010

On 20 October 2010 Dr Lancee referred the deceased back to Dr Deague. The deceased had described symptoms believed to be consistent with unstable angina. As Dr Deague was unavailable on 21 October 2010 she referred the deceased to her colleague, Associate Professor David Playford, at St John of God Hospital, Subiaco.

A coronary angiography was performed on 21 October 2010 by Professor Playford through a left femoral approach.

A summary of the procedure contained on the medical file reported that:

"...at the end of the procedure, femoral arteriography was performed to determine suitability for device closure of the femoral puncture. The anatomy was favourable and the femoral artery was closed using a 6F Angio-Seal device."

The procedure was uncomplicated and the angiogram showed no significant stent, thrombosis or restenosis.

Because the deceased had diagnosed acute coronary syndrome previously despite taking aspirin and clopidogrel, her medication was changed from clopidogrel to prasugrel 10mg (a different blood thinning agent which inhibits platelet aggregation). She was continued on aspirin and was to be given enoxaparin subcutaneously for 10 days. Enoxaparin (also known as clexane) is a blood thinning agent that inactivates clotting factors.



The deceased stayed at the hospital overnight for review and was seen by Dr Deague and the resident, Dr Irani, at about 11am on 22 October 2010.

The multidisciplinary notes contained in the St John of God Hospital file record that at that time the patient was well with left groin bruising and tenderness.

The notes further describe the left groin bruising as “small haematoma 2cm x 1cm”. In addition it was noted that there was “slight tenderness”.

The recorded plan was that she would be discharged home and would take ‘Clexane’ for two weeks and panadeine forte for pain relief.

An entry in the notes timed at 11.30am records that the clinical pharmacist provided the deceased with a medication list and that she was discharged with an adequate supply of medications.

The deceased was discharged with documentation in standard form provided by St John of God Health Care Cardiac and Vascular Intervention Laboratory which provided discharge advice and advice about her Angio-Seal. This documentation did advise that should her haematoma worsen she should contact her consultant and that Duty



Nurse Managers were also available and emergency numbers were provided.

THE ANGIO-SEAL

Professor Playford used an Angio-Seal device to close the puncture in the artery where the catheter was inserted for the angiography procedure.

The Angio-Seal is a vascular closure device which is intended to quickly seal femoral artery punctures, minimising bleeding and enabling a patient to be discharged from hospital early. The device is intended to create a mechanical seal. The Angio-Seal device is intended to place an anchor over the puncture hole inside the artery and later deploy collagen outside the artery creating a complete seal.

The description of the device given by the manufacturer, St Jude Medical, is as follows:

DEVICE DESCRIPTION

The ANGIO-SEAL™ Vascular Closure Device VIP Platform consists of the ANGIO-SEAL VIP device, an insertion sheath, an arteriotomy locator (modified dilator) and a guidewire. The ANGIO-SEAL VIP device is composed of an absorbable collagen sponge and a specially designed absorbable polymer anchor that are connected by an absorbable self-tightening suture (STS). The device seals and sandwiches the arteriotomy between its two primary members, the anchor and collagen sponge. Hemostasis is achieved primarily by the mechanical means of the anchor-arteriotomy-collagen sandwich, which is supplemented by the coagulation-inducing properties of the collagen. The device is contained in a delivery system that stores and then delivers the absorbable components to the arterial puncture. The delivery system features a Secure Cap that facilitates proper technique



for delivery and deployment of the absorbable unit. The Angio-Seal Vascular Closure Device components are not made from latex rubber.

It is clear that in this case the Angio-Seal did not create a complete seal from at least 22 October 2007, the day after it was inserted. This could have been because of a failure of the device itself (although this suggestion was not supported by the manufacturer, St Jude Medical) or it could have been because of a failure to achieve vascular closure with the device. One suggested reason for a failure to achieve vascular closure was that the puncture site was possibly too high, proximal to the inguinal ligament. This is a matter which was explored at the inquest and is referred to later in these reasons.

THE CONDITION OF THE DECEASED FOLLOWING HER DISCHARGE FROM HOSPITAL

The deceased was discharged from St John of God Hospital in Subiaco on Friday 22 October 2010. She was picked up by her daughter, Kerry Cornish. When she left the hospital she had some minor pain but was able to walk without using a wheelchair.

Later that day or the next day, Saturday 23 October, Mrs Cornish saw the area of bruising and she estimated its size as 10cm x 2cm.



On that day, the deceased was showing a number of people the extent of the bruising, which was already fairly extensive.

On Monday 25 October the deceased called Mrs Cornish and asked her to take her to the doctor. Mrs Cornish went with her to see her general practitioner, Dr Alida Lancee, who worked at the Byford Family Practice. Dr Lancee had been the deceased's general practitioner for a number of years and she clearly trusted her. According to Mrs Cornish, the deceased 'loved' Dr Lancee.

By that stage the bruising was very extensive and, according to Mrs Cornish, it had gone across from one leg to the other and up to her navel, across the whole of her stomach³. According to the deceased's daughter, Clare Fifield, who was a registered nurse, there was then a "black band" over her "belly" which had been there since the Saturday and the bruising went down the leg.

By Monday 25 October the deceased was in much more pain than on the Saturday and, according to Mrs Fifield, by the Monday she was wanting to be in bed all the time.



THE APPOINTMENT WITH DR LANCEE OF 25 OCTOBER 2010

According to the deceased's daughter, Mrs Cornish, when the deceased went to see Dr Lancee she was in extreme pain with a huge bruise. She was barely able to get up on the examination table and at one stage said to Dr Lancee, "I'm in fucking agony"⁴.

According to Mrs Cornish Dr Lancee said, "Wendy, you have a least 500 mls to a litre of blood there", indicating her belly.⁵

According to Mrs Cornish Dr Lancee told the deceased that her body needed to be knocked out so that it could recover and she was given both slow release morphine in tablet form and fast acting morphine to be taken immediately (these were in fact Oxycontin and Oxynorm, synthesised opiates with similar properties to morphine).

There is no doubt that Dr Lancee did not contact Professor Playford or Dr Deague to obtain more information in relation to her patient's condition, did not refer her to either of them and did not send her to hospital.

This failure was extremely important in the context of the events which followed as at that time, based on the expert evidence provided to the inquest, I am satisfied that

⁴ ts 21

⁵ Exhibit 1, tab 6; ts 22



the deceased's condition was survivable. Had immediate action been taken to send her to hospital or to refer her to Professor Playford, the problems relating to the bleeding could have been corrected.

At the time of the appointment Dr Lancee made brief progress notes in which she recorded the fact that the deceased had the angiogram five days earlier and had developed a "very sore haematoma around arterial puncture"⁶.

Under the heading "examination" she recorded, "afebrile warm tender haematoma extending lower abdomen and inner thigh peripheral pulse is normal". Her plan was, "increased analgesics" and her diagnosis given under the heading "reason for visit" was, "infective haematoma angiogram".

There were additional progress notes recorded on the practice's computer system later relating to this visit⁷.

The circumstances in which these notes came to be written is significant in assessing their reliability.

It appears that after the deceased's collapse on 27 October Professor Playford sought to ascertain how it was that she presented with such massive bleeding and the

⁶ These notes are attached to the report of Dr Lancee at Exhibit 1, tab 8

⁷ Found at Exhibit 1, tab 8



problem had not been addressed sooner. He had been told by the family that the deceased had visited Dr Lancee but had not been sent to hospital. He then spoke to Dr Lancee on the telephone about the case. It is obvious that in that conversation Professor Playford was critical of Dr Lancee's lack of action.

Dr Lancee stated in evidence that in the context of that telephone call, she considered that "it was important for me to write down everything I remembered should there be any consequences afterwards from what he told me".⁸

According to Dr Lancee she made handwritten notes on 28 October which she copied onto the practice's computer system as progress notes for the deceased on 3 November 2010.

These transposed notes contained a number of obvious errors, particularly relating to the dates. According to these notes, for example, the deceased had presented on 22 October, whereas in fact she had presented on 25 October.

These notes include the following:

Said the large bruise was present on the day of discharge which was reviewed by cardiologist Dr Deague who checked it and advised [sic] continue anticoagulants and use panadeine forte.

The notes of the deceased's history continued:

Denied any increase size of the bruise since discharge or review
Dr Deague just needed better pain relief. As panadeine forte not
enough [sic].

The notes went on, 'specifically asked if the bruise was getting any bigger and she answered "no, the same as Friday".'

In evidence Dr Lancee claimed that these notes accurately recorded her interactions with the deceased.

Mrs Cornish in her evidence, however, disputed this account and said that her mother had never said that the bruise had not grown in size. She said if the deceased had been asked the question, which she doubted, '... the answer wouldn't have been "no", of course it had increased in size'⁹.

As the bruise had obviously grown dramatically larger at the time of Dr Lancee's review compared with its size at the time of the deceased's discharge from the hospital, it is difficult to see why the deceased would have said to Dr Lancee that it was not getting any bigger. In that context I prefer the evidence of Mrs Cornish and do not accept that the deceased in any clear or specific way provided information to the effect that the bruise was unchanged after her release from hospital.

In addition I do not accept that the section of these notes, purporting to record the deceased as saying that Dr Deague had checked the “large bruise” and advised her to use panadeine forte, was accurate. When Dr Deague saw the bruise it was very small and there was only mild tenderness not then requiring panadeine forte (although this was prescribed for use if needed by the registrar). In that context I do not accept that the deceased ever provided this history.

Dr Lancee did accept that the deceased was suffering from pain which had been inadequately controlled by taking eight panadeine forte per day. She said for that reason she had prescribed Oxycontin 15mg twice a day, with Oxynorm (a rapid acting opioid) in between as required.

Dr Lancee made a decision not to investigate the bruise which she saw on the deceased for a number of reasons, the most significant of which were a claim that the deceased had told her there was no increase in the size of the “haematoma” and that she understood that Dr Deague had reviewed the deceased after the angiogram and had not felt it necessary at that stage to investigate for ongoing bleeding.

Dr Lancee’s diagnosis was that the deceased had developed an infection in the haematoma and for that reason commenced her on Cephalexin, an antibiotic.



In respect of the size of the bruising and, or, haematoma Dr Lancee provided the court with a diagram on which she had marked separately “bruising” and “haematoma”. This diagram¹⁰ depicted extensive areas of “bruising” and “haematoma”, though not as large as the areas described by the deceased’s daughters. Her understanding of the difference between these terms was that “haematoma” referred to a raised area, while “bruising” related to areas where the skin was flat.

Clearly both terms related to bleeding under the surface.

Dr Lancee accepted readily that the extent of bruising she saw was vastly bigger than the 2cm x 1cm bruise recorded at the hospital prior to the deceased’s discharge, but said that she was unaware of that information.

She stated that she assumed that the bleeding had come from the femoral puncture site at the time of the procedure. She said, however, that even if there had been no bleeding after that time, she would have expected that the area of visible bruising would have appeared bigger over time and would have become more evident.

¹⁰ Exhibit 8



Dr Lancee was unclear in her evidence as to whether she had used the word “bruise” or the word “haematoma” when she spoke with the deceased.

In the above context, even if the deceased had said something to Dr Lancee to the effect that the bruise or haematoma had not got bigger, it is difficult to see why such a statement would have provided Dr Lancee with any comfort, particularly as on her own account such a claim would have been unreliable.

In my view, in the context where the deceased presented with very extensive bleeding, that should have been cause for significant concern and Dr Lancee should have at the least discussed the extent of the bleeding with Dr Deague.

According to Dr Lancee, she specifically asked the deceased whether Dr Deague was aware that she had a painful haematoma, to which the deceased had replied “Yes”.¹¹ I do not accept that this claim was accurate. Dr Deague definitely did not know that the deceased had a painful haematoma as when the deceased left the hospital she was only suffering what was described as “slight tenderness”. It was very clear from evidence of her daughters that the extent of pain had increased dramatically after the deceased left hospital and I do not

¹¹ ts 112



accept that the deceased would have ever told Dr Lancee that Dr Deague was aware of her suffering from significant pain.

In my view pain which was inadequately controlled by taking 8 panadeine forte per day was such that immediate positive action needed to be taken. Even if the pain levels could be attributed to infection and possible sepsis in the haematoma, in a context where there was extensive bleeding in the area of an arterial puncture, further immediate investigation was required. Even if the diagnosis of possible sepsis been correct, prescription of an antibiotic and provision of pain relief was not an adequate response.

EVENTS OF TUESDAY 26 OCTOBER 2010

On 26 October 2010, according to her daughter Mrs Cornish, the medication which the deceased had received from Dr Lancee was making her “groggy and drowsy”.

The deceased began to vomit multiple times and the Byford Family Practice was again contacted. Dr Lancee was not available and Dr Naga Ganjikutna spoke to the deceased on the telephone. Dr Ganjikutna’s progress notes record that the deceased reported continuous vomiting since she had been on “morphine” (this referred to the Oxycontin and



OxyNorm). She said that she had severe pain, but the bruise was the same and there was “no change in size”.

Whether the external area of the bruise had in fact changed significantly since the day before was unclear. It was clearly very large on both 25 and 26 October.

Dr Ganjikunta prescribed Maxolon for the vomiting without seeing the patient.

The deceased’s daughter picked up the prescription for her.

THE EVENTS OF 27 OCTOBER 2010 UNTIL THE TIME OF DEATH

On 27 October 2010 the deceased’s condition deteriorated significantly. The deceased felt “cold and clammy”.

Mrs Cornish telephoned Dr Lancee and explained that the deceased did not have the energy to get out of bed.

On this occasion Dr Lancee responded very positively and told her to hang up and call an ambulance.

Mrs Fifield then called for an ambulance to attend and the deceased was taken by ambulance to Armadale Hospital.



The deceased presented to the Emergency Department in full cardiac arrest with resuscitation in progress by the St John Ambulance staff.

On examination in the Emergency Department the deceased was pale, peripherally shut down, with no spontaneous respirations and no peripheral pulse. A large left groin haematoma was noted. Cardiopulmonary resuscitation was continued.

An electrocardiogram showed sinus rhythm although there was no cardiac output. The deceased was given adrenalin intravenously but at 10.32am she went into ventricular fibrillation and was given a 200 joule shock, which caused her to revert to a sinus bradycardia.

Emergency resuscitation efforts continued at Armadale Hospital until around 12pm when she was transferred to Royal Perth Hospital Emergency Department.

On arrival at Royal Perth Hospital the deceased went to the operating theatre where her left groin was explored and the bleeding site in the artery was secured.

The Royal Perth Hospital notes record that the puncture wound was seen in the left external iliac artery. This was potentially significant as the puncture should have been in the femoral artery.



The external iliac artery and the femoral artery are one vessel, differently named where the inguinal ligament crosses over in front of the artery.

The puncture should have been below the inguinal ligament not above it. If the puncture was in the external iliac artery it would have been above the inguinal ligament.

Precisely where the puncture site was situated is discussed later in these reasons.

At the time of the surgery the deceased was already showing signs of multiple organ failure. Following return to the Intensive Care Unit the deceased was commenced on continuous venovenous haemodiafiltration to counteract acute renal failure.

On 28 October 2010 the deceased's condition continued to deteriorate and an exploratory laparotomy was conducted. At this time a section of small bowel showing signs of ischaemia was resected. The deceased was subsequently returned to the intensive care unit.

By this stage aggressive multiple organ failure was evident. There was evidence of progressive respiratory failure requiring high inspired oxygen concentrations to maintain adequate oxygen levels.



On 29 October 2010 the deceased was returned to theatre for a further laparotomy. There was then extensive bowel necrosis which was beyond resection and incompatible with survival.

The deceased's family were informed of these findings and further resuscitation efforts were ceased. The deceased was certified life extinct at 12.45pm on 29 October 2010.

ANTICOAGULANT THERAPY

At the time of her death the deceased was receiving three different blood thinning agents and this would have certainly increased the extent of bleeding at the puncture site.

It was believed by Dr Deague and Professor Playford that the deceased had previously suffered from acute coronary artery syndrome despite the use of aspirin and clopidogrel therapy. She was changed from clopidogrel to prasugrel 10mg along with her aspirin therapy. As she had previously had several courses of enoxaparin (clexane) subcutaneously with good result, she was continued on that therapy as well.

According to Dr Deague, the deceased was aware of how to inject herself with the enoxaparin and was also



aware that it was an anti-coagulant, increasing the effect of aspirin and prasugrel.

Both Dr Deague and Professor Playford were aware of the fact that the deceased would be continued on anti-coagulant therapy following the procedure and when she saw Dr Lancee on 25 October 2010, Dr Lancee continued the deceased on the anti-coagulants on the basis that it was understood that she had previously suffered from a clot in her coronary stent and Dr Lancee did not consider there was any active bleeding at the time of her presentation.

Dr Donald Latchem gave expert evidence at the inquest to the effect that the deceased was at risk of retroperitoneal bleeding because of this regime (which was agreed by all the experts). Dr Latchem, however, went on to express the opinion that he would normally expect that clexane would be stopped 12 hours before an angiogram and that he would not have continued with clexane unless there was an identified thrombus (clot).

Dr Latchem further stated that following the procedure, in the event he considered that clexane should be continued, he would have waited 24 hours to ensure that there was no ongoing bleeding post procedure.

In this case it was noted that on 22 October, the day after the procedure, there was only a small haematoma of



2cm x 1cm and no evidence of ongoing bleeding. There was, therefore, no evidence of significant bleeding almost 24 hours after the procedure.

It was the evidence of Professor Playford that it was not uncommon for patients undergoing angiography to require blood thinning medication and in this case he considered that there was good evidence of clotting which if it occurred could have been fatal. In his view while there might always be the risk of a bleed, this should be capable of being corrected, while a thrombus could result in fatal complications.

While the expert opinion evidence in relation to this issue was to an extent contradictory, I do accept that Dr Deague and Professor Playford had the opportunity of assessing the deceased's condition directly and were convinced that she had suffered from ongoing angina which had been effectively treated in the past by antithrombotic medication. It was also their view that intermittent stent thrombosis had been suspected by other medical experts and she had previously been put on clexane.

While it would not be appropriate for me to address the issue of anticoagulant therapy in the context of angiography in any detail herein, I do accept that the treating medical practitioners, Dr Deague and Professor Playford, had reason to suspect unstable angina and to consider that it was



important for there to be robust antithrombotic medication prescribed.

I further accept that it was a legitimate expectation that retroperitoneal bleeding, even if made worse by anti-coagulant therapy, should have been reversible, while, assuming that the deceased had previously suffered from acute coronary artery syndrome, there was a risk of development of a thrombus which could have proved fatal.

THE POSSIBILITY OF HIGH ARTERIAL PUNCTURE

It appeared to be accepted by all of the experts in this case that the arterial puncture was high, although there was dispute as to whether the puncture was in the external iliac artery or the femoral artery.

According to Professor Playford it was necessary for him to insert the catheter at a relatively high location because further down he was not able to feel the pulse from the artery.

He accepted there was a slightly higher risk of retroperitoneal bleed with a higher puncture, but referred to literature which suggested that the increased risk was in the order of 0.5%.

Professor Playford was positive in his evidence that the insertion was not above the inguinal ligament and provided



the court with film indicating the location of the insertion which was played using a thumb drive (exhibit 16).

It was noted that the Royal Perth Hospital notes relating to the procedure of 28 October contained reference to the puncture wound being in the external iliac artery, although it was unclear whether at the time of the procedure the inguinal ligament (which defines the separation between the external iliac artery and the femoral artery) was in place.

In the post mortem report of forensic pathologist, Dr C T Cooke, there is reference to sutures being identified on the external iliac artery. In evidence, however, Dr Cooke stated that at the time of his examination the inguinal ligament had already been moved. Based on his examinations in other cases where the inguinal ligament was in place, he expressed the view that the area of puncture which he identified would have been at about the location of the inguinal ligament.

As indicated earlier in these reasons, it was the view of the Managing Director of St Jude Medical, in a letter provided to the court, that a possible reason for failure of the Angio-Seal device could have been the fact that the puncture site was high and proximal to the inguinal ligament.



The Angio-Seal device was not located at the time of the procedure on 27 October 2010 when the defect was closed and it was not found at post mortem examination.

It is not now possible to determine precisely why the Angio-Seal device did not adequately close the puncture site until the puncture was healed. It is possible that a relatively high puncture increased the likelihood of a retroperitoneal bleed, but based on the available literature it is unlikely that there would have been a significantly greater risk of a bleed for that reason in this case.

On the basis of Professor Playford's extensive evidence and the evidence of the angiography (the film contained in Exhibit 16) I accept that the puncture was in fact in the femoral artery, though at a high location.

It is noted that there would always be some risk of a bleed following a puncture of the artery and in this context it was particularly important that when haemorrhage was noted, the deceased should have been immediately returned to the hospital for treatment.

CONCLUSION

The deceased was an otherwise relatively healthy woman who died on 29 October 2010 as a result of multiple organ failure following haemorrhage from penetration of the femoral artery.



The penetration of the artery occurred during a coronary angiography which was performed by Professor Playford on 21 October 2010, using a left femoral approach.

The angiography was performed in order to ascertain the cause of pain believed to be consistent with unstable angina.

At the end of the procedure an Angio-Seal device was used to close the puncture hole.

On 22 October 2010 the deceased was discharged from hospital having been reviewed by Dr Deague who was satisfied that the procedure had gone well. At that time there was a very small haematoma evident at the puncture site.

Following discharge from hospital there was increasing bleeding from the puncture site and by 25 October 2010, when the deceased presented to her general practitioner, Dr Lancee, she was suffering from an extremely painful groin haematoma which had dramatically increased in size.

The deceased was not referred to hospital and her consultant surgeon was not contacted.



On 26 October 2010 because she had been suffering from continuous vomiting, following a telephone call contact she was prescribed Maxolon.

On 27 October 2010 the deceased's daughter contacted Dr Lancee by telephone and at that stage the deceased was seriously ill. Dr Lancee advised that an ambulance should be called immediately and the deceased was taken to the Emergency Department of Armadale Hospital in full cardiac arrest secondary to hypovolemic shock.

At that stage the deceased's condition was not recoverable and although she was transferred to Royal Perth hospital for further treatment her condition continued to deteriorate.

The deceased died on 29 October 2010 after it was discovered that she was suffering from extensive bowel necrosis which was incompatible with survival.

Had the deceased contacted the expert medical practitioners who had been treating her, Dr Deague or Professor Playford, in the days immediately following the angiography her life could have been saved. Even if the deceased had been referred to those experts by Dr Lancee or had gone to hospital following her visit to Dr Lancee on 25 October 2010, she would have almost certainly survived.



By the time the deceased went to Armadale Hospital on 27 October 2010 it was, tragically, too late and she died as a result of the ongoing bleeding.

I find that the death arose by way of Accident.

**COMMENTS ON PUBLIC HEALTH & SAFETY ISSUES:
SECTION 25(2) OF THE CORONERS ACT 1996**

This death was unnecessary and could have been avoided had the deceased contacted her treating experts, Dr Deague and Professor Playford, or had she returned to St John of God Hospital.

It is likely that the deceased did not appreciate the extent of deterioration of her condition or the sinister indications of the internal bleeding which she was suffering.

When the deceased saw Dr Lancee on 25 October 2010, three days after having been discharged from hospital, it is clear that Dr Lancee did not fully appreciate the fact that she was at that time suffering ongoing bleeding and certainly was not alert to the fact that her life was at risk.

If the deceased had been given a discharge summary document which accurately described her condition at the time of her discharge on 22 October, and had she brought



such a document with her to see Dr Lancee, Dr Lancee should have been alert to the need for immediate hospitalisation and treatment and it is very likely that the outcome would have been different.

It is also very likely that the outcome would have been different if a copy of such a discharge summary had been forwarded directly to the deceased's general practitioner, Dr Lancee, preferably by email, to ensure that she would have been reliably informed about the deceased's condition at discharge.

RECOMMENDATION 1

I RECOMMEND that all private and public hospitals at which angiograms are performed provide patients with a discharge summary which would preferably contain a diagram of the body on which the extent of any haematoma could be marked and which would provide reliable information as to the extent of any bleeding, pain levels and medications at the time of discharge.

RECOMMENDATION 2

I RECOMMEND that any such discharge summary given to a patient should encourage the patient to retain that document and take it to any doctor seen in the event of complications, such as ongoing bleeding.



RECOMMENDATION 3

I RECOMMEND the discharge summary provided to the patient should also be provided electronically, or otherwise as quickly as practicable, to the patient's general practitioner.

OBSERVATIONS IN RELATION TO THE RECOMMENDATIONS

1. It is recognised that medical practitioners and particularly consultants working in private hospitals are extremely busy and are reluctant to complete more paperwork than is absolutely necessary, but this case has highlighted the fact that provision of discharge summaries to patients and their general practitioners is an important part of the provision of health treatment in hospitals. Discharge summaries need not be lengthy, and standard forms and templates can be used for their completion, while still enabling effective communication (which may save lives) to occur.
2. While this case has necessarily focused on events relating to a procedure at St John of God Hospital Subiaco, the safety issues referred to in these comments have general application and so the



recommendations have not been limited to that hospital.

A N HOPE
STATE CORONER
17 January 2013

