Coroners Act, 1996
[Section 26(1)]

Western *Australia*

RECORD OF INVESTIGATION INTO DEATH

Ref No: 34/13

I, Evelyn Felicia Vicker, Acting State Coroner, having investigated the death of ***Valma May Ruth FORD*** *with an Inquest held at Geraldton Coroners Court, Geraldton Court House, Geraldton, on the 26-30 August 2013 find the identity of the deceased person was* ***Valma May Ruth FORD*** *and that death occurred on 22 November 2009 at Geraldton Regional Hospital as a result of Complications Following a Recent Fracture and Surgical Repair of the Right Hip in an Elderly Lady with Complex Chronic Heart Disease and Chronic Renal Impairment in the following circumstances -*

Counsel Appearing :

Ms K Ellson assisted the Acting State Coroner

Ms R Young (instructed by State Solicitors Office and with her Ms Philpott) appeared on behalf of WACHS, Geraldton Regional Hospital
Mr G Bourhill (instructed by MDA National) appeared on behalf of Dr R McLaren
Ms B Burke (instructed by ANF Legal Services) appeared on behalf of Nurses Kohler & Mpikamezo
Mr D Bourke, with him Ms K McNally (instructed by MDA National) appeared on behalf of Dr Figueiredo

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INTRODUCTION

On 16 November 2009 Valma May Ruth Ford (the deceased) fell at home. She was taken to the Emergency Department (ED) of Geraldton Regional Hospital (GRH) where an x-ray showed a comminuted pertrochanteric fracture of the right upper femur.

On 19 November 2009 the deceased underwent repair of her fracture by open reduction and internal fixation (ORIF) with a Richard's pin and plate. She appeared to recover appropriately with some fluctuations in her observations.

On 21 November 2009 the deceased's condition began to deteriorate and she was transferred to the High Dependency

Unit (HDU) in the early hours of 22 November 2009. Her condition was discussed with her family and a Royal Perth Hospital (RPH) doctor in the Intensive Care Unit (ICU). Arrangements were made to transfer the deceased to RPH ICU by Royal Flying Doctor Service (RFDS) but her condition deteriorated before she could be safely transported. She was returned to HDU GRH.

The deceased died in the early evening of 22 November 2009. She was 77 years of age.

BACKGROUND

The deceased was born on 18 March 1932 as the youngest of three children. They were fifth generation West Australians.

The deceased was raised in South Perth and attended Hollywood Primary School before becoming employed as a shop assistant and photographic colourist. She was married at 20 years of age and had four children. The family moved around the state to follow the deceased's husband's employment, until he retired in 1987, and the deceased and her husband made their home in Geraldton.

The deceased had many hobbies including genealogy and at the time of her death had seven grandchildren, seven great grandchildren, with another four born in the year of her death.

Mr Ford died in January 1995 and the deceased maintained their home in Rangeway until her death.[1]

On 16 November 2009 the deceased fell at her home and was transported to GRH by ambulance following a call for assistance from the deceased's granddaughter.[2]

MEDICAL

The deceased's medical history included atrial fibrillation and aortic stenosis, managed by a cardiologist, Dr K Woollard, hypertension and hyperlipidaemia. She was being treated with Warfarin to prevent thrombus formation, Avapro HCT for hypertension, amiodarone, an antiarrhythmic, and atorvastatin to lower her cholesterol. She had a history of urinary tract infections (UTIs) for which she was treated with antibiotics from time to time.

The deceased's GRH records indicate she had presented to GRH in June, July and September 2009 with falls which had caused lacerations/ulcerations of both legs, and with a urinary tract infection. She is recorded as having a history of aortic stenosis and being on Warfarin for paroxysmal atrial fibrillation, although she remained in sinus rhythm throughout her final admission. Aside from the Avapro HCT she was also

[1] t 26.8.13 p3,4
[2] Exhibit 1 Vol 2 Tab 23 SJA Patient Care Card

prescribed Felodipine, Lipitor and Vagifem. She was recorded as a long time smoker.[3]

GRH 16 NOVEMBER 2009

The deceased was recorded as arriving at the ED at 1120 hours having had a fall at home. She recorded no loss of consciousness but was complaining of pain in her right hip and leg, and her leg was noted to be externally rotated and shortened. She was afebrile but mildly hypotensive (BP 104/37).

X-rays revealed a comminuted pertrochanteric fracture of the right upper femur. A chest x-ray showed a normal sized heart and clear lung fields. Her INR was checked and found to be well controlled by Warfarin therapy at 2.5. She was provided with a femoral nerve block to reduce her pain and admitted to the ward as a surgical patient. She was given morphine, an indwelling catheter was inserted and she was fasted in preparation for theatre.

There is an admission notation, appearing to be for the 16th , stating the deceased had cloudy urine. Later evidence indicated this did not relate to the 16th , but postoperatively in the presence of an indwelling catheter (IDC) on the 19th.[4] There is no reliable evidence of a urinalysis at admission.

[3] Exhibit 1 Vol 1 Tab 11
[4] T 28.8.13 p228

In 2009 Mr Ross McLaren was a visiting orthopaedic surgeon to the GRH. His normal weekly schedule was to fly to Geraldton on the Monday evening and spend his time whilst in Geraldton working between GRH and St John of God's Hospital. The 16 November 2009 was a Monday. Mr McLaren received a call from the nursing staff on the 16th informing him of the deceased's presence on the ward awaiting surgery.[5] He ordered that her Warfarin be withheld pending his review.

17 NOVEMBER 2009

On 17 November 2009 at about 9.30am the deceased was reviewed by Dr Fiebelkorn, Senior Medical Officer (SMO) and he ordered blood tests. When the results of the blood tests were received it was noted the deceased had a haemoglobin of 69, her platelet and white cell counts were normal, both sodium and potassium were low and her C-reactive protein (CRP) was 49.

Due to the deceased's low haemoglobin she was transfused with two units of blood and surgery was postponed to enable her Warfarin level to decline and promote a suitable INR for surgery.

Dr Said, Consultant Physician, when asked to review the deceased with respect to the withdrawal of Warfarin noted

[5] Exhibit 8

the deceased was an ex-smoker, suffered from paroxysmal atrial fibrillation and had aortic valve disease. He could see no past history of strokes, DVT or pulmonary embolism and for the forthcoming surgery he recommended administration of Vitamin K to reverse the action of Warfarin. There were intended to be repeat blood tests to check her INR, followed by more Vitamin K if warranted. This did not occur.

Generally withdrawal of Warfarin with the administration of Vitamin K is preferred to stabilise an INR where emergency surgery is not time critical.[6] Dr Said noted the deceased was "febrile and has a moist cough" with "decreased air entry over both lung bases". Her CRP was elevated to 49, but there was no evidence of pneumonia on the previous x-rays, although there was evidence of emphysema. Due to the fact the deceased was not mobilising freely Dr Said ordered physiotherapy and nebulised saline.

Dr Said instituted DVT prophylaxis in the form of LMW Heparin once it had been established there was no evidence of ongoing bleeding from the fracture site. He indicated it was his view the deceased may need to be nursed in HDU postoperatively due to her co-morbidities.

Mr McLaren reviewed the deceased on the evening of the 17th in preparation for his GRH theatre days which were

[6] t 28.8.13 p 221-222

generally on a Wednesday or Thursday, depending on his commitments for that week whilst in Geraldton. His untimed entry in the deceased's Integrated Progress Notes (IPN) indicate a query as to whether the fracture has aggravated the deceased's anaemia and that she was for fracture fixation (Richard's) when stable, meaning the withdrawal of her Warfarin and stabilisation of electrolytes.

18 NOVEMBER 2009

On 18 November 2009 the deceased was again reviewed by Dr Fiebelkorn and repeat blood tests showed a rising CRP (80) and still low sodium and potassium. The deceased had been stable overnight and was afebrile with a satisfactory urine output. At 10am all her observations were normal. Due to the fact the deceased's INR had still not reduced satisfactorily for surgery, it was decided she would not be operated on on the Wednesday, and her operation was delayed until the Thursday.

In evidence Dr Said said his intention in prescribing the Vitamin K and repeat test for INR was an attempt to reduce the deceased's INR gradually as the most appropriate way to stabilise her whilst other factors such as her electrolytes were also an issue. His aim for her operative INR was between 1.1 to 1.5 and on the 17th when he initiated the Vitamin K treatment it had been 2.4. Dr Said said to stabilise the deceased's INR without the addition of Vitamin K and

only withdrawal of Warfarin would have taken about five days.[7]

The deceased's CRP which had been 49 on 17th had risen to 80 on the 18th and was 122 on the 19th, peri-operatively. The deceased was afebrile and her white cell count remained normal. [8]

19 NOVEMBER 2009

The 19th, Thursday, was the last day upon which Mr McLaren was prepared to conduct surgery on the deceased. Consequently it was decided she should receive fresh frozen plasma (FFP) to lower her INR because the administration of Vitamin K had not succeeded in taking her IRN below two.

The deceased received a blood transfusion prior to her operation to stabilise her electrolytes and assist with her haemoglobin levels. A spike in the deceased's temperature at approximately 3pm on the 19th was related to the blood transfusion.[9] Otherwise the deceased's temperature remained normal and there had been no comment there was any issue with blood cultures.

Mr McLaren believes he reviewed the deceased in the morning prior to her operation with the intention he take her

[7] t 28.8.13 p211
[8] t 28.8.13 p186
[9] t 28.8.13 p186

to theatre that evening to enable her recovery period to be while he was still in Geraldton, before flying out on the Friday evening.

An entry in the IPN on 19th indicates Mr McLaren noted significant pressure sores on her left heel, the need for careful attention and the nurses were directed to watch for pressure sores on her buttocks. [10]

During the course of the evidence it became apparent a notation about urine analysis (UA) for 16 November actually related to 19 November, preoperatively, when the deceased had an indwelling catheter in place.[11]. This caused some confusion for the admitting nurse, in evidence, who, it appears, had omitted to take a midstream UA on 16th, but assumed from the note he had.[12]

The observation chart from 19th records a cloudy urine for the deceased. In reality this is the first time there was such a record. [13] The results of the UA were not of concern for 19 November 2009.

Mr McLaren considered the deceased's increasing CPR related to the inflammatory process due to the fracture of

[10] t 28.8.13 p188
[11] t 28.8.13 p229 Exhibit 1 Tab 23Y
[12] t 28.8.13 p262
[13] t 28.8.13 p229

the femur. He was not of the view on his review of the notes the deceased had a significant infection pre-operatively.

Mr McLaren's note of the theatre times were the procedure commenced at 7.53pm and finished at 8.24pm. The FFP had reduced her INR to 1.4, suitable for surgery. Her fracture was treated by open reduction and internal fixation with a Richard's pin and plate.

During the procedure the deceased was administered 1g of Keflin at the introduction of anaesthesia as a standard antibiotic prophylaxis for orthopaedic operations.

At the conclusion of the operation Mr McLaren wrote post-operative notes for her ward care and included *"care – she already has black heels"* and an indication her haemoglobin should be checked again in the morning. In evidence Mr McLaren stated he did not recall whether he observed a pressure sore in the deceased's sacral area but believed it would not have been an area he observed during the course of the operation.[14]

The sacral pressure area seems to have been noted by the nurses and notes were made for the deceased's mattress to be replaced with one suitable to care for her pressure sores.

[14] t 28.8.13 p188-9

Mr McLaren is certain he would have reviewed the deceased on the Friday morning to ensure her operation and recovery were appropriate. While he has not made a note in the IPN there is one by Dr Fiebelkorn stating she had been seen. Mr McLaren said in evidence there would not have been any concerns with her progress or he would have made an entry. Mr McLaren was clear there was nothing in the test results available that he was able to review to suggest the deceased had a significant infection and needed additional antibiotic treatment to that received during the course of the procedure. He was not contacted about the deceased again.

The deceased had been returned to the ward at 9.30pm and the record of observations overnight did not reveal any concerns.

20 NOVEMBER 2009

The deceased was reviewed at 7.45am on 20 November and seemed to be recovering well with no complaints. She was seen by the physiotherapist and Dr Fiebelkorn. She was afebrile with a normal pulse and blood pressure.

The nursing notes indicate ongoing pressure area care and that the deceased was appreciative of the benefits of the

new mattress and the ability to tilt her weight from the sore area.[15]

At 2pm on 20 November the deceased's blood pressure fell to 94/33 and she was treated with IV saline because it was believed she was dehydrated. This appeared to improve her blood pressure to 108/36 by 3.30pm. The deceased's blood pressure fell again that evening and recovered at about 10pm to 107/82. The deceased's oxygen saturations dropped to 90% on room air and she was provided with oxygen. TEDs stockings were in place.

21 NOVEMBER 2009

On the 21 November 2009 at 6am it was noticed the deceased's urine output had dropped to only 120mls in the last five hours. Dr Milne was informed and more IV saline was administered. The deceased had a large loose bowel action. Her blood pressure was recorded as 95/42 and the deceased complained of being very thirsty. She was provided with oral fluids and when the physiotherapist attended for her review she noticed the deceased's blood pressure had dropped to 88/33 on exertion.

The deceased was reviewed by Dr Ray Varghese later that day. He diagnosed "hypotension secondary to anti-hypertensive/diuretics" as well as UTI. He ordered the anti-

[15] t 28.8.13 p234

hypertensives be ceased and the administration of more fluids, this time with IV antibiotics (ceftriaxone)[16]. The deceased was not appearing unwell but her blood and urine tests indicated an E coli infection.

By 3pm that afternoon the deceased had vomited and her blood pressure had been very variable. At 4.30pm the nurses again recorded a drop in blood pressure and the deceased complained of her neck and head being heavy. The 21st was a Saturday and the cover for the general wards was generally provided by the A & E doctors out of normal business hours. The deceased was reviewed by an A & E doctor who indicated the deceased's fluids were to be continued. Her temperature at that time was low at 35.5°C.

On that Saturday evening at 10pm there is a note from the Locum Senior Medical Officer from the Emergency Department stating he had reviewed the deceased due to her blood pressure being "less than 100 motd (most of the day), now less than 80". He noted her IV intake of fluids and that there was inadequate urine output before her indwelling catheter had been removed earlier in the day. He noted she was afebrile with dry mucous membranes, had a low blood pressure and her pulse indicated she was in atrial fibrillation. He believed she was hypotensive secondary to inadequate fluid intake contributed to by an E coli urinary

[16] Exhibit 1 Tab 5 Tab 23J

tract infection. He ordered a bolus of saline fluid and more IV fluids with potassium supplements. He ordered her catheter be replaced to monitor her urine output and noted she was already on antibiotics for a UTI.

The deceased later passed another loose bowel action followed by three more and the notes describe her bowel action as "uncontrollable".[17]

22 NOVEMBER 2009

In the early hours of the Sunday at 4am (22nd) the doctor noted the deceased still felt dry and her blood pressure was 72/30, her pulse was 74 bpm and she was afebrile. Her urine output remained poor although he noted her chest was clear. He ordered a Gelofusine as a volume expander and requested an ECG. By 5am there was no improvement in her blood pressure and her condition was discussed with the ICU registrar at RPH who recommended a dopamine (an inotrope designed to raise the BP) infusion.

The plan was to move the deceased to the High Dependancy Unit (HDU) and then consider transferring her to RPH if she did not improve.

[17] Exhibit 1 Tab 23J

By 5.35am the deceased was described as "conscious and alert, marked pallor, with cool peripheries". By 6am her blood pressure had improved to 107/34.

The deceased had been transferred to HDU at 5.30am. At 8.30am she was reviewed and arterial blood was collected which showed a low Ph but marked hypoxia and the beginnings of acidosis. There was by now significant hypoatremia (Na 127) and there was an instruction to increase inspired oxygen.

A medical note in the IPN indicates it was believed the deceased had developed sepsis secondary to her urinary tract infection and an anaesthetist had been called to insert a central line and arterial line.[18] A repeat chest x-ray showed shadowing in the left midzone, and blood tests hypoatremia, renal impairment and a marked rise in her CRP to 253. This is indicative of inflammation, most likely due to infection. The tests also showed marked anaemia and following the discussion with the senior registrar at ICU at RPH it was decided to continue to manage the deceased in the HDU rather than an ICU due to her co-morbid conditions. She was given the antibiotics (ceftrixone then ciprofloxacin) and her blood pressure improved to 111/45. Her Glasgow Coma score was 15 out of 15 and her oxygen saturation was 95% on 6 litres of inhaled oxygen.

[18] Exhibit 1 Tab 5

The plan was to further discuss the deceased with Royal Perth Hospital ICU if any further problems arose and to consider a CT pulmonary angiogram to rule out a pulmonary embolus if she remained hypoxic.

The next note in the IPN is that of Dr Varghese in which he had a discussion with the deceased's family and explained to them the deceased had developed septicaemia secondary to urinary and chest infection. Dr Varghese believed the deceased was in septic shock with multi organ failure including kidney, lung and heart. He indicated her prognosis was poor even with active treatment and the family were in discussions.

Dr Varghese further discussed the deceased with the doctors at RPH ICU and they agreed it was time to accept the deceased into RPH ICU. The Royal Flying Doctor Service was contacted and arrived.

Evidence from the deceased's daughter indicated the deceased was by now extremely frightened and distressed at the changing of plans for her care and they felt her level of agitation could have been reduced with appropriate counselling and family input. [19]

[19] t 29.8.13 p323

The deceased was transferred by ambulance to the airport but her condition rapidly deteriorated on the way to the airport. She was returned to GRH about 4.20pm because RFDS were not happy to transport the deceased in her condition. The deceased was hypoxic, bradycardic, hypotensive and acidotic. She was intubated and ventilated and given adrenaline, fluids and atropine.

Despite this treatment the deceased failed to respond or recover and died at 6.23pm on 22 November 2009.

POST MORTEM EXAMINATION[20]

The post mortem examination of the deceased was carried out on 25 November 2009 by Dr J White and Dr C Unwin of the PathWest Laboratory, Forensic Pathology.

The initial post mortem examination revealed fluid within the chest and abdominal cavities and congestion and oedema of the lungs. There was widespread atherosclerosis (narrowing and hardening of the arteries). The examination also showed enlargement of the heart, scarring of the kidneys, congestion of the liver and small gallstones.

With respect to the atherosclerosis there was mild calicific sclerosis of the aortic valve of the heart, while the coronary arteries revealed 50% luminal occlusion over 3 cms of the left

[20] Exhibit 1 Vol 1 Tab 13

anterior descending artery, 40% luminal occlusion over a length of 2 cm of the circumflex artery, and the right coronary artery 80% luminal occlusion over a length of 1cm. This is a significant restriction in the deceased's efficient blood flow. The aorta itself showed numerous ulcerated atherosclerotic plaques.

In evidence Dr Jamieson commented on the enlargement of the heart with respect to the clinical observation, prior to the deceased's death, the deceased's lungs and heart appeared normal. He indicated the hyperinflation of the deceased's lungs due to her condition would have made the heart appear comparatively normal and explained the fact post mortem, it was in fact enlarged.[21] The post mortem also indicated the recent surgical repair of the deceased's right hip without apparent problems.

Further investigations were conducted in an attempt to elucidate a comprehensive cause of death for the deceased and on 22 November 2010 Dr White wrote to the Geraldton Coroner with a synopsis outlining her view the deceased died as a result "complications following a recent fracture and surgical repair of the right hip in an elderly lady with complex chronic heart disease and chronic renal impairment".

[21] t 29.8.2013 p368

Dr White indicated the complications would include underlying bladder infection and possible chest infection, acute on chronic renal failure, and heart failure leading to generalised sepsis, shock and multi-organ failure.

Microscopy of the sampled tissues showed some scarring and hypertrophy of the heart muscle, mild inflammatory changes in the lungs with features of congestion, hyaline membrane disease and some mild scarring in the liver. Dr White observed the kidneys showed severe nephrosclerosis and commented the fact the deceased's kidneys appeared to be functioning adequately clinically did not remove the fact her post mortem examination revealed chronic renal impairment which is why she had used the word impairment rather than failure.[22]

Dr Klimaitis, Consultant Physician, who had reviewed the management of the deceased on behalf of the Coroner's Court had commented the deceased's "Creatinine was normal on admission and only rose on the last day. Even then it was only 126." Dr Klimaitis was of the view renal failure did not play any part in her death, however, it is clear from the post mortem results any additional stress on the deceased's physiology and biochemistry would not have been managed well by her system.[23]

[22] t 26.8.13 p57
[23] Exhibit 1 Vol 1 Tab 11 page 2

In evidence of Dr White outlined the fact of the deceased's fluid at post mortem in her lungs, chest cavity, liver and abdominal cavity indicated to her the deceased was in heart failure and was overloaded with fluid at the time of her death. Dr White explained the effect of a fluid overload on the deceased's system relevant to her death. Dr White stated the deceased:

> "Had underlying vulnerability because of her existing heart disease, and it would appear that she was probably in mild chronic heart failure '....' by what I found at post mortem, both grossly and under the microscope, and one of the complications which refer to as contributing to her death would be the acute on chronic heart failure".[24]

The atherosclerotic arterial system would have placed her heart under great stress with reduced blood flow to the heart muscle. Under the microscope the deceased had features of both hypertrophy of the muscle and also old areas of scarring of the heart muscle which would be consistent with pre-existing areas of ischaemia in the heart muscle.

Dr White stated the state of the deceased's kidneys also indicated features of chronic heart disease. The features of both the heart and kidney indicated that impairment had been existent for several years.

[24] t 26.8.13 p47

The post mortem examination also revealed purple bruising over the backs of both of the deceased's heels with a measurement of up to 8cm. Over the back of the deceased in the sacral area there was an ulcer which measured approximately 8 x 5cm. Dr White was confident these areas were pressure areas. Dr White was not able to age the pressure areas but did indicate it is her experience pressure areas can develop quite fast depending on various features to do with the individual. The perfusion of an individual can affect their vulnerability to pressure areas as can body fat. In the case of the deceased Dr White was of the view that the deceased was of medium build. Whilst it was a guesstimate Dr White stated it could be a number of days for the pressure areas to develop.[25]

Dr White indicated the condition of the deceased's skin in her lower limbs was consistent with people suffering from poor peripheral circulation however, she was not prepared to say the deceased did have poor peripheral circulation, rather "she had features which would certainly support that"[26].

Poor peripheral perfusion in conjunction with a low blood pressure might mean the flow to the skin surface would be less and so pre-empt the formation of pressure sores.

[25] t 26.8.13 p 49
[26] t 26.8.13 p50

If the deceased had in reality been underweight rather than of normal weight, as appeared at post mortem, that could also exacerbate the incident of pressure areas. [27] It would also emphasise the enlargement of her heart.

Dr White saw no evidence of the deceased being long term anaemic, but agreed it could predispose a person to heart failure due to the lack of availability of haemoglobin to carry oxygen around the body.

Dr White confirmed tissue and blood samples had been taken from the deceased to examine the growth of any organisms by culture however, indicated the fact the deceased had been on intensive antibiotic therapy would probably negate positive results despite the organisms being present.

Overall Dr White believed the deceased's heart was acutely decompensating on the background of features of longstanding chronic disease. Dr White also considered the deceased's lungs showed features of "shocked lung" often seen postoperatively with infection, low blood pressure, and for various other physiological reasons. Dr White believed the deceased was septic despite the finding of no pathogens nor micro abscesses indicating long term infection. Plus the acute ischaemic changes observable in the kidneys on the

[27] t 26.8.13 p 56

background of the features of chronic disease in the kidneys with a lot of scarring,[28] supported the fact of a serious infection although she could find no focus for that on post mortem.

The lack of abscess formation in the kidneys tended to indicate any infection in the kidneys was not longstanding. From the post mortem results Dr White was of the view the deceased's heart failure was the most relevant factor to her death, although she could not comment on any underlying sepsis because there was no extreme evidence other than the fluid overload which could not be confirmed at post mortem, but was consistent with the clinical picture prior to death.

Clinically the deceased did not show indications of renal impairment until the day of her death and then only mildly. Dr White commented that grossly, under the microscope, the deceased had evidence of chronic renal disease and as part of her age and her generalised athersclerosis, Dr White felt it would have made her much more vulnerable to stresses and her kidneys had very little reserve. If the deceased's kidneys were just functioning in a borderline fashion then any stresses such as infection, or surgery itself, was going to tip her kidneys into the poorly functioning zone. Dr White's view was they were just starting to see evidence of

[28] t 26.8.13 p52

that at the time she died. Dr White had observed under the microscope features of the deceased's kidneys which indicated they were starting to struggle and that there were acute ischaemic changes.[29]

The whole scenario with respect to the deceased led Dr White to the view her overall underlying co-morbidities would make her much more vulnerable to any problems postoperatively. She had borderline functioning. This would certainly make it more difficult for the deceased to cope with any additional insult to her system.

The signs at post mortem were not consistent with the deceased having long-term sepsis and, the clinical picture of no sign of sepsis until possibly the last day or two before death, would appear to be the extent of an infection in the deceased.[30]

OVERVIEW OF THE CARE OF THE DECEASED

Following the death of the deceased her family expressed concern to the Geraldton Coroner as to the deceased's management, in view of their perception the deceased was suffering a urinary tract infection (UTI) at the time of her operation. They were also very distressed by the pressure sores they observed on the deceased prior to her death and

[29] t 26.8.13 p 57
[30] t 26.8.13 p 60

the pain those were causing her. They believed these were caused by inappropriate 'splinting' of the deceased's limbs. By the time the deceased died she was very frightened because she did not understand what was happening to her. This certainly caused her family great concern because it had appeared the deceased was coping very well with her life up until the time of the operation.

A request was made to Dr A Klimaitis, Consultant Physician, to overview the management and postoperative care of the deceased for his input with respect to the family concerns and any general concerns he may have raised. He was specifically asked to consider the relationship between a possible infection and the timeliness of the administration of any antibiotics, the deceased's fluid management, and general postoperative care with respect to comments and clinical notes about dehydration, and the post mortem result of fluid overload. Dr Klimaitis also raised the issue of the delay between the deceased's admission to hospital and the actual operation. [31]

INFECTION

Initially, there had been some concern with the appropriate recording of admission urinalysis in the deceased's medical file. While there was some documentation which implied there had been an admission urinalysis taken there were no

[31] Exhibit 1 Vol 1 Tab 11

results, and later results, which may have reflected a problem were revealed, by the evidence, to have been taken at a much later time in the deceased's admission. These were transposed into the notes in an effort to provide some results. This was not done maliciously but was an attempt to make the records comprehensive. Unfortunately this tended to confuse fairly clear facts because witnesses relied on the notes for their actions in the absence of recall.

Firstly, an admission midstream urinalysis should have been used as a diagnostic tool on a patient with the deceased's profile to establish whether she was suffering from a UTI at the time of admission.[32] It was not.

Secondly, on the whole of the evidence it is very unlikely the deceased was suffering a UTI at the time of her admission. The post mortem results do not support this and the clinical signs of infection by way of hypotension and elevating CRP are not significant until 21 November 2009, two days postoperatively.

The fact the admission nurse believed he had taken a urinalysis on admission was due to the difficulty mentioned above with respect to the notes and his inability to recall the issue independently.[33] Due to the partial filling out of the

[32] t 29.8.13 p349
[33] t 28.8.13 p 262/3

request for urinalysis he believed it had been done, when it appears it had not.

Admission staff were not able to comment on the splints the family were concerned had caused the deceased pain on arrival, and pre-empted pressure areas and another site for insfection in the event there was an infection on admission. However, the evidence was splints of the type described by the family would have been used on transfer by ambulance to prevent movement and additional trauma to the fracture itself.[34] Stabilisation of the fracture site was a means to reduce further trauma to the area and may well have continued pre-operatively. Nursing staff agreed the deceased's daughter had assisted with the provision of a supplementary cushion to help alleviate pressure pain for the deceased. [35]

The deceased's pressure sores were well noted on 19 November, at the time of her operation, and there are entries in the nursing notes which indicate the deceased did receive pressure care, however, the fact of the pressure sores would also indicate an elevated suspicion for infection may have been warranted.

The deceased was provided, coincidentally, with antibiotics on the day of the operation as a prophylactic measure and

[34] t 29.8.13 p335/6 t 28.8.13 p273
[35] t 29.8.13 p292

these would have been effective for a period of time but were not continued due to there being no threshold of suspicion with respect to the deceased's overall profiles.

The deceased was always noted as being clinically dry. Dr Klimaitis believed this was probably a reflection of ongoing or developing sepsis, however, other than the antibiotics on the day of her operation there was not enough clinical suspicion raised by her presentation until midday of 21 November 2009 for antibiotic therapy. On that day the deceased was given ceftriaxone and gentamicin. Due to her continued deteriorating condition the deceased was discussed with a microbiologist from RPH who advised her antibiotics be changed to ciprofloxacin, timentin, and vancomycin in the early hours of 22 November 2009, while discussions were being held as to the deceased's transfer to ICU at RPH if there was no improvement.[36]

Dr Klimaitis was of the view the deceased's overall clinical picture pre-operatively was not supportive of any infection and, even postoperatively, the fact she appeared clinically well, did not have a particularly elevated temperature, had good oxygen saturation, and was able to participate in physiotherapy until the morning of 21 November 2009, all indicated it was not unreasonable for the deceased not to

[36] Exhibit 1 Vol 1, Tab 5, Tab 11

have been given antibiotics at an earlier stage, despite the reference to cloudy urine on 19 November 2009.

Evidence was received that the deceased's prior urinary tract infections related to the organism E. coli. There was some discussion during the inquest as to whether there was an E. coli infection at the time of the deceased's operation on the 19 November 2009.

The deceased had an indwelling catheter and there was a notation her urine appeared cloudy on the 19th, the day of her operation. The blood tests taken later which revealed E. coli were unable to determine whether E. coli was present in her system, or was associated with her indwelling catheter. Due to the fact no base line levels had been established with a midstream urine analysis on admission, and there was no midstream urine analysis without the indwelling catheter, the presence of E. coli was not able to be established by the time of her operation. The deceased was given keflin as a prophylactic antibiotic for the operation. That would have been effective on the E. coli for the duration for that dose. If preoperative E. coli was the source of the deceased's sepsis by the evening of the 21st, when other antibiotics were given, then she would have been unprotected from ongoing E. coli infection.

As Dr Klimaitis said

> "If there is an infection in the bladder and there's a catheter there, you know, it would have been nice-I mean, certainly it's better to have had that keflin than not have it, but it still would have been good to have antibiotics continued, assuming that there was infection in the urine earlier".[37]

In reality the deceased was not showing any clinical signs of infection until the 21st November. This may just have been reflective of the efficacy of the antibiotic with respect to any E. coli, which then multiplied due to the withdrawal of antibiotics.

In hindsight, it would have been preferable for antibiotics to have been continued following her operation but there is no significant evidence that would have changed the outcome for the deceased. It appears the main contributing factor to her death was the state of her cardiovascular system which, due to the developing sepsis postoperatively, became overloaded and failed. Therefore, a developing sepsis contributed to her death because it was an additional insult, arising from the fact of the fracture and need for an operation, that caused the deceased's system to fail. She was unable to compensate for any further insult.

Dr Klimaitis agreed, the presence of the pressure sores and the deceased's rising CRP may have been indicators however, on their own he could understand why earlier

[37] t 26.8.13 p45

antibiotic intervention had not occurred. It may be an indicator there should be a lower threshold for suspicion of infection in patients with the deceased's profile. [38]

DELAY TO OPERATION

The death of the deceased occurred following her admission to hospital for repair of a fractured neck of femur in 2009. There is a high morbidity rate for elderly patients with this type of injury and its frequency is increasing with increased longevity. Experience is indicating the more quickly repair of this type of injury can be successfully performed in the elderly the more improved the outcome for survival. This is largely due to a patient with a number of co-morbidities being very vulnerable to decline once their normal routine and mobility practises are severely disrupted. A decrease in mobility and pain arising out of a fractured neck of femur in the elderly inevitably pre-empts a period of immobility. Of its self the trauma of the injury and operation can destabilise patients with life threatening conditions.

This has resulted in the developing practise of operating upon patients with this type of injury as quickly as possible provided there are no contra indicators. In the case of the deceased she was on the medication Warfarin which thins the blood. This was to treat her serious cardiac disease. For

[38] t 26.8.13 p25-28

an operation the effects of Warfarin need to be reversed. This can be done gradually by withdrawing Warfarin, more rapidly by withdrawing Warfarin and providing prothrombinex or a very large bolus of Vitamin K or in emergency situations such as motor vehicle accidents, by the administration of fresh frozen plasma (FFP).

For the deceased her Warfarin therapy was not her only difficulty with respect to surgery. She was found to be anaemic, which is a risk factor and for which she was treated with blood transfusions, but she also had an electrolyte inbalance which needed to be stabilised. Due to the fact she was not assessed as time critical for surgery, and Mr McLaren was able to delay her surgery, the decision was made to withdraw her Warfarin and reduce her INR by the use of Vitamin K. The Vitamin K administered was according to the guidelines. Unfortunately the deceased did not respond to the Vitamin K as promptly as was expected. In addition a follow up Vitamin K administration was missed over night on 17-18 November 2009 (Tuesday-Wednesday) due to the fact GRH in 2009 had inadequate night medical cover.

There is no indication this would have reduced her INR because later doses failed to achieve the required reduction in her INR.

Originally Mr McLaren was to have operated on the deceased on the 18th, Wednesday, being his normal theatre day at GRH, however, due to the fact her INR remained above 2.0 theratre was delayed until the 19th. Even then the deceased's INR was not appropriate for surgery and in the morning of the 19th she was provided with FFP and prothrombinex. This had the desired effect of reducing her INR to levels suitable for surgery and Mr McLaren carried out the surgery on the afternoon/evening of the 19th. This was a delay of three days from the time of her injury, and may also have contributed to her lowered haemoglobin which was corrected by blood transfusion.

With respect to the delay Dr Klimaitis stated the earlier the operative process the better, provided the patient is stable.[39]

In evidence, it became obvious the deceased's INR could have been reversed earlier by the use of the fresh FFP, however, as Dr Said indicated with great clarity, the fact of the deceased's elevated INR was not the only problem with which the clinicians were confronted. If Dr Said had been given to understand the deceased's surgery was time critical then he would have ordered the administration of the FFP in time for the operation to occur on the Wednesday. However, he was not given that indication and his involvement was merely to advise as to the most appropriate

[39] t 26.8.13 p16

way of reducing the deceased's INR in conjunction with her overall medical state. Had the FFP not been used on the 19th then the delay for the deceased's operation would have been in the order of a week rather than three days. By the time of the operation on the 19th her other risk factors had been corrected to the point it was safe to use FFP and operate. [40]

Dr Said pointed out the deceased had numerous risk factors with respect to her ability to recover appropriately from surgery. She had multiple co-morbidities and to have put her through urgent surgery without proper stabilisation would have increased her risk factors. Dr Said stated the deceased had anemia and required multiple blood transfusions. She had hypokalemia (low potassium) which is a risk under anaesthesia which needs to be corrected by the use of intravenous fluids containing potassium, which was done, and the third concern was her general well being. It was "not just a matter of her INR not therapeutic, but there was anemia, there was hypokalemia and there was signs of ongoing inflammation or infection. So yes, in her case, I would say it was not such-wouldn't have been very safe to rush in the first 24 hours or even 48 hours to do a surgery and she needed some sort of stabilisation and problem sorting". [41]

[40] t 28.8.13 p212-213
[41] t 28.8.13 p216

Dr Said also noted the deceased's history of smoking and her emphysema which also carried the risk of morbidity by way of chest infections. He had asked for blood cultures to be obtained and the deceased was provided with a saline nebuliser due to his observation she had a moist cough. The other indication there may have been an infection was her elevating CRP. However, as both Dr Klimaitis and Mr McLaren stated that may have been indicative of other things, although in the overall circumstances for the deceased it was a marker which warranted some consideration by clinicians preparing her for surgery.

Dr Said also pointed out there were some risks with the use of FFP in a patient showing high comorbidities. He indicated there was a risk of infection, a risk of volume overload with a patient with heart disease and a risk of a allergic reaction with the use of the prothrombinex. In all those circumstances it was his view it was more appropriate to stabilise the deceased by the administration of Vitamin K until they were satisfied they had stabilised all her other problems, and there was an urgent need for reversal of the walfarin due to the time for surgery becoming more critical.[42]

Dr Klimaitis finished his overview with the summary he would have preferred to see the deceased's operation performed earlier (between 24-48hrs after admission) with more

[42] t 28.8.13 p223

aggressive reversal of her Warfarin. In hindsight, starting antibiotics on the 19th would have helped, although, if she looked well withholding antibiotics was a reasonable option. The deceased needed careful monitoring and CRP levels should have been continued to be performed postoperatively. The persistent hypotension was also a pointer that sepsis may have been present.[43]

Overall the management of the deceased by GRH covered the matters Dr Klimaitis believed warranted attention and the explanations from the clinical medical staff as to why there were delays were not unreasonable in 2009.

CONCLUSION AS TO THE DEATH OF THE DECEASED

I am satisfied the deceased was a 77 year old female who, despite her serious comorbidities, managed her daily health very well. Consequently, there were no obvious indicators, other than her cardiac disease, which revealed the extent of her borderline capacity to function appropriately. The deceased's cardiac disease was managed with Warfarin therapy. For the life she led she appeared relatively healthy.

Unfortunately, on Monday 16 November 2009 the deceased suffered a fall and fractured the neck of her femur, a known traumatic injury in the elderly with a high rate of morbidity.

[43] Exhibit 1 Vol 1 Tab 11

The orthopaedic surgeon, Mr McLaren, operated at GRH on Wednesday's. Attempts were made to stabilise the deceased's status to those optimal for surgery on the 18th, however, due to her prior Warfarin therapy her INR remained out of the optimal range for successful surgery without excessive bleeding.

There were two factors relevant to the failure to reduce her INR. One was the reduced medical cover in the general wards over night from the 17th -18th. This required a reliance on nursing staff to obtain medical input from the emergency department practitioners dependant on the situation in ED. The other factor was the deceased's atypical response to Warfarin reversal by use of Vitamin K in any event.

Any decreased mobility in a person with borderline functioning can exacerbate the potential for infection to occur postoperatively. There is no reliable evidence the deceased was suffering an infection at the time of admission. However, it was not unreasonable to expect a patient with her profile to experience an increased risk of infection postoperatively, especially in the presence of pressure sores which indicate some difficulty with appropriate perfusion.

Nevertheless, there were no obvious clinical signs of a rising infection until 21 November 2009 when her physiotherapist indicated some concerns. The deceased was started on antibiotics but in the context of her overall deteriorating state due to the trauma of the operation, an acute sepsis developed, with the associated insult on an already compromised cardiovascular system.

While there was pressure care, it did not appear to alleviate the deceased's pain, and so added additional distress in conjunction with her deteriorating functioning, and her lack of understanding as to why she had felt well pre-operatively, and was now experiencing a frightening decline.

Despite the fluid overload at post mortem there is no evidence the deceased's fluid management prior to her death was erroneous in view of the difficulties she was experiencing. Her declining blood pressure at times responded to the fluid boluses she was given and it would seem sepsis was mostly responsible for the dehydration observed. The fluid load post mortem was due to her cardiac failure.

Unfortunately, GRH does not have an ICU for comprehensive management of acutely unwell patients postoperatively. HDU ensures competent monitoring and nursing.

Attempts to transfer the deceased to an ICU in the metropolitan area were not effective by the time the receiving clinicians were prepared to accept transfer.

The deceased died in GRH on 22 November 2009, three days postoperatively and six days following her fall.

I find death arose by way of Accident.

COMMENTS ON THE CARE OF THE DECEASED

Evidence was heard from Dr Andrew Jamieson, Regional Medical Director responsible for the mid-west region of W.A. Country Health Service, at the conclusion of the inquest. He is mainly located in the Geraldton region. He considered his most important function to be "ownership of clinical quality and process from the medical perspective." [44]

With respect to the antibiotic cover for the deceased in 2009, Dr Jamieson, who has only been in his position since 2012, stated he was of the view, by 2013, there were criteria for recognising that in elderly female patients there is a propensity towards the development of UTI's. Consequently there should be a high index of suspicion for their development and there should have been specific diagnostic processes in place to test whether the deceased was suffering a UTI on admission. She should have been

[44] t 29.8.13 p348

screened thoroughly and earlier to ensure there was not a risk of infection during the operative process.[45]

Taking that into consideration an appropriate pathway would have been for competent and appropriate admission urinalysis by way of midstream urine prior to the insertion of an indwelling catheter. Dr Jamieson believes this is now recognised by two protocols which have been introduced into GRH more recently. One of those is the introduction of the "fractured neck of femur pathway", and also the "older patient initiative". These involve multidisciplinary expert teams reviewing patients with a view to managing any issues pre-emptively and getting people home as soon as possible following this type of high risk surgeon in elderly patients.

Dr Jamieson outlined that in the metropolitan region there are 100 geriatric early management (GEM) units. The benefits of these are being adopted in country areas as pathways and initiatives without the benefit of in-house orthogeriatricians. That has only occurred in GRH in mid 2013. [46]

Arising out of the difficulty with an appropriate administration of Vitamin K to the deceased over night 17-18 November 2009 the inquest considered there was room for improvement on the pathology request forms to indicate

[45] t 29.8.13 p349-351
[46] MRI 123, Ex 3

whether blood tests results were required urgently to enable appropriate clinical intervention.

Dr Jamieson agreed there was a systems failure due to the GRH not having ward medical night staff nor there being provision, in 2009, for adequate hand-over from the ward clinicians to the ED clinicians. The situation in 2013 is considerably improved with the addition of a number of medical staff since November 2012.

Factually, had the deceased been given a second dose of Vitamin K that evening, it may have become apparent earlier she was not responding to Vitamin K therapy. The operation could then have proceeded as planned on the 18th , but with FFP instead, had Mr McLaren and the medical staff been satisfied her other areas of concern had been corrected by blood transfusion.

Overall the delay caused by the failure to reverse her INR effectively prior to the 19th does not seem to have been critical on the evidence of Dr Said, which put her in a more receptive condition on the 19th for the trauma of an operation.

Evidence was given by the Coordinator of Nursing and Midwifery at GRH, Derek Fraser. Mr Fraser has been at GRH since February 2012. Prior to that time he was a Nursing

Resource Coordinator at GRH. There was evidence in the integrated progress notes some pressure care had been provided to the deceased by the nurses in accordance with the deceased's nursing plan. Now there are additional protocols and procedures in place, post 2009, which Mr Fraser believes would have lessened the trauma of the pressure sores for the deceased. These include "the turn clock"[47] for the management of pressure sores and the bed heel elevator[48] which is used to alleviate pressure from the heels. There are now new nursing protocols used to assess appropriate care.[49]

The deceased's family had been very distressed by the fact the deceased remained in what the family referred to as "splints" for a long period preoperatively. They believed this was the cause of her pain associated with pressure sores. None of the staff could remember this however, it was noted ambulance officers do use splints to decrease movement in a fracture which can be extremely painful. Sometimes these remained on patients for longer than transfer and various techniques were used to try and reduce movement of fractures to reduce pain and provide some cushioning to decrease pressure areas.

[47] Exhibit 4
[48] Exhibit 5
[49] MR1 28 November 2011 utilised 2012

Mr Fraser gave evidence there are now different types of pressure relieving devices available. He agreed foam boots did provide some comfort, however, evidence suggests they are not effective in reducing pressure areas because of the lack of re-distribution of the pressure and there are now more effective alternatives.

Recommendation No. 1

WACHS consider increasing the education of staff and reinforcing the need for and requirements of documentation, including timing entries, documenting family concerns, and communications between doctors and nurses.

Recommendation No. 2

WACHS to consider increasing education of staff and reinforcing the need for staff to communicate with patients and their families in relation to the patient's treatment and plan for care.

Recommendation No. 3

The pathology request forms in use have a clear field for the urgency of results, and there be a protocol as to when and how results are delivered, and to whom.

Recommendation No. 4

WACHS continue with the audits already in place to ensure real time appropriate adherence to policies procedures, documentation and the relevant completion of all forms requiring action.

E F VICKER
ACTING STATE CORONER
12 November 2013