Coroners Act, 1996

[Section 26(1)]

Western Australia

RECORD OF INVESTIGATION OF DEATH

Ref No: 07/14

I, Evelyn Felicia VICKER, Deputy State Coroner, having investigated the death of ***Julissa Teresa GILBERT*** *with an inquest, held at* ***Geraldton Coroner's Court, Geraldton Court House, Geraldton,*** *on* ***4-5 March 2014*** *find the identity of the deceased person was* ***Julissa Teresa GILBERT*** *and that death occurred on* ***18 September 2008 at Geraldton Regional Hospital*** *as a result of* ***Intra-abdominal Haemorrhage following Appendectomy*** *in the following circumstances:-*

Counsel Appearing :

Ms K Ellson assisted the Deputy State Coroner
Mr S Denman (instructed by Denman Popperwell) appeared on behalf of Mr Hudson
Mr G Bourhill (instructed by Tottle Partners) appeared on behalf of Dr John Stace
Mr D Bourke (instructed by Clayton Utz) appeared on behalf of Dr Ruth Highman
Ms B Burke (instructed by Australian Nursing Federation) appeared on behalf of Nurses Monaghan and Howie
Ms R Young (instructed by State Solicitors Office) and assisted by Ms A Salapak appeared on behalf of WACHS

Table of Contents

INTRODUCTION

Julissa Teresa Gilbert (the deceased) presented to Geraldton Regional Hospital (GRH), Emergency Department (ED), at 7:50pm on 17 September 2008 complaining of abdominal pain.

She was taken to theatre at 11:30am on 18 September 2008 for an appendectomy, with hypoxia and significant hypotension and a history of chronic obstructive airways disease (COAD). The base of her appendix was gangrenous but the appendix had not ruptured and there was no evidence of peritonitis. The operation was essentially uneventful other than the deceased's fluctuating blood pressure (BP). The deceased's recovery was largely uneventful and she was transferred at 4pm to the high dependency unit (HDU) in view of her pre-existing hypoxia and hypotension.

Dr John Stace was called from ED to HDU at approximately 5:30pm to review the deceased who was increasingly hypotensive and requiring increasing metaraminol (aramine) infusion. He formed the opinion her hypotension was due to septic shock and ordered more IV fluids with increased aramine infusions. The deceased's BP did not improve and she died at 7:45pm.

The deceased was 57 years of age.

BACKGROUND

The deceased was born on 17 July 1951 and, at the time of her death, resided in Rangeway with her de facto partner of many years.

The deceased had a history of hypertension, arthritis, asthma and COAD. She had smoked for many years. She was centrally obese and her general practitioner was recorded as the Aboriginal Medical Service (AMS).

On 17 September 2008 the deceased had eaten a hot dog at approximately midday. From about 1pm she complained of an abdominal pain and had vomited at 6pm & 7:30pm. She complained of a "little bit of pain" and so presented to the ED at GRH.

ED/GRH

The deceased presented in the ED at 7:50pm. She was recorded as afebrile with a temperature of 36.9°C. She had a normal pulse at 75 beats per minute and her blood pressure was 136/67. On examination her abdomen was soft and tender in the epigastrium and below her umbilicus. The doctor noted her onset of pain had occurred following eating a hot dog and that she was distressed. Examination of the deceased's abdomen indicated "soft lower quadrant,

tenderness, no guarding or rigidity, bowel sounds +".[1]

The doctor gave a provisional diagnosis of "gastroenteritis" with management by way of IV access and IV fluids and analgesia. This was commenced.

Dr Knausenberger reviewed the deceased at 10:15pm and noted she was still in pain. On examination he recorded she had a generalised lower abdominal tenderness with no mass or rebound. He checked the results of her blood tests and noted she had a raised white cell count (WCC) and that her X-ray showed a dilated large bowel with no fluid levels. At 11:40pm he planned the deceased be kept in overnight with a continuation of her IV fluids and analgesia. The radiologist's report indicated the abdominal and chest film was normal other than stones in the gall bladder.

The deceased was admitted to a ward at 2:45am on 18 September 2008. She had been provided with morphine and her pain appeared to have settled.

The deceased's integrated progress notes (IPN) noted her oxygen saturation had dropped to 84% on room air and as a result she was provided with 3L of nasal prong oxygen which improved her oxygen saturations to 93-94%.[2] There was no doctor review at that time.

[1] Ex 1, Tab 13, Tab A
[2] Ex 1, Tab 13, Tab R

At 6:05am her temperature had risen to 38.5°C and her pulse increased to 108 beats per minute. She was using her abdominal muscles for breathing and Dr Knausenberger was asked to review her, which he did at 6:15am. He recorded she was uncomfortable with pain in her lower abdomen and coughing up yellow sputum. Her oxygen saturation was 91% on 3L of oxygen and, on examination, he noted crepitations in both her lungs, and that her abdomen was distended and tender on the right side. There was no rebound and no guarding and he gave a provisional diagnosis of "pneumonia" with a differential diagnosis of "ileus with secondary sepsis". He added IV antibiotics to her management and asked she be surgically reviewed with an abdominal CT scan in the meantime.

At 8am on doctor review the deceased's temperature remained the same with an oxygen saturation of 88% on room air. The deceased's blood pressure had dropped to 99/66 and her pulse was 106 beats per minute. There was tenderness over the deceased's lower right abdomen with guarding and Dr Stafford considered the deceased was suffering "acute abdomen" with an exacerbation of a COAD which was causing the low oxygen saturation. The deceased's x-ray was normal and the abdominal CT scan ordered earlier showed appearances "consistent with acute appendicitis". As a result Dr Stafford ordered repeat blood tests and planned she be sent to theatre after discussions

with the surgeon.

Dr Stafford noted that in his view the deceased was significantly dehydrated.

Dr Stafford reviewed the deceased at 9:30am and noted her improved oxygen saturation and normal hemoglobin levels (128g per litre) with a continuing elevated WCC in keeping with an infection. She had almost normal liver function and normal renal function.

SURGERY

The theatre list on 18 September 2008 was performed by Mr Martin Hudson, general surgeon, with the assistance of Dr Ruth Highman as anaesthetist. Mr Hudson had received a call from Mr Perry asking him to include the deceased on his theatre list for that day as an urgent appendectomy.

Preoperatively the deceased was reviewed by Dr Highman. Dr Highman was aware the deceased needed an urgent appendectomy and it was her function to accommodate the risk factors in the deceased's preoperative presentation in an attempt to ensure safe anesthesia. Dr Highman noted the deceased was a centrally obese female who was shocked, likely by septic shock secondary to peritonitis in the view of her diagnosis of appendicitis.

Dr Highman noted the deceased's blood pressure was now 96/47, her pulse was 101 beats per minute, and her temperature had dropped to 37.6°C. She assessed the deceased as moderately dehydrated although she had received 2L of normal saline over the past 12 hours. She considered the deceased to be tachycardic and hypotensive (as opposed to her normal hypertensive state) and that she had a urine output of greater than 50ml per hour. From a cardiac perspective the deceased denied any cardiac history and her ECG recorded a normal sinus rhythm with apparently nil ischemic changes. The deceased advised Dr Highman she took medication for hypertension but was unaware of which medication that was.

The deceased's respiratory status was recorded as hypoxic with an oxygen saturation of 86% on room air increasing to 97% on 6L of oxygen through a Hudson mask. Her arterial blood gases (ABG) at that stage were a pH of 7.34, pC02 at 54, p02 at 94, BE at 3.3, and HC03 at 29.1. This indicated mild respiratory acidosis which Dr Highman queried as being caused by the deceased's smoking. She was known to have COAD, was producing a productive cough of yellow sputum and that there were "creps" in the lower zones of both lungs. Her chest x-ray indicated her lung fields were clear. Dr Highman recorded an impression of infective bronchitis and atelectasis.

The deceased's renal function appeared good with her urine

output being satisfactory and her other readings for urea and electrolytes (U&E) normal. She had an acute abdomen. She had good movement of her neck and mouth opening, which would assist intubation. Overall Dr Highman assessed the deceased as having Australian Society of Anaesthetists (ASA) rating of IV.[3] In evidence Dr Highman indicated she believed the deceased was an ASA III/IV, which reflected a patient with a severe to incapacitating systemic disease which limits activity and is potentially life threatening.[4]

Due to the pre-operative presentation of the deceased Dr Highman planned an anesthetic technique which included the insertion of an arterial line to ensure her arterial blood pressure was monitored due to her pre-existing hypotension. The deceased was provided with deep vein thrombosis prophylaxis with a calf compression device and she was administered IV antibiotics because of her airways disease. Dr Highman also commenced the deceased on an infusion of aramine to assist with her low BP, with the rate to be titrated to maintain her systolic blood pressure >100mg of mercury (Hg). In evidence Dr Highman indicated she considered a central venous line, however, determined the arterial line was more appropriate.[5]

Mr Hudson reviewed the deceased briefly at 11:20am in the

[3] Ex 1 Tab 8
[4] t 5.3.14 p54
[5] t 5.3.14 p56-7

reception area of theatre and confirmed the deceased was apparently suffering from appendicitis. Mr Hudson noted she had COAD, hypoxia and was suffering significant hypotension.

The operative anaesthetic chart[6] starts recording the anaesthesia at 12:15pm with the arterial line being inserted at approximately 12:50pm following a fluctuation in the deceased's BP from 100/60 to 185/105. Following the arterial line insertion the reading at 1pm was 125/55.

In evidence Mr Hudson confirmed the fact there had been no signs of bleeding during the procedure nor would he expect bleeding during an appendectomy. The blood supply to the appendix is with a singular artery which is small and often thrombosed in the presence of severe appendicitis. The risks of bleeding are negligible.[7]

Mr Hudson advised Dr Highman, which she does not recall,[8] the deceased's appendix was gangrenous at its base. It had not perforated and he could see no evidence of peritonitis. Mr Hudson remarked on the fact he did not think the deceased's appendix was sufficiently infected to account for the degree of septic shock that was apparently exhibited by the deceased.[9] Other than those observations Mr Hudson

[6] Ex 1 Tab 13 reverse Tab V
[7] t 4.3.14 p76
[8] t 4.3.14 p58,68
[9] t 4.3.14 p80

described the operation in his personal notes as uneventful and taking approximately thirty minutes. He performed the procedure by suturing off the cut stem of the appendix and enclosing it in the mesentery.

In evidence Mr Hudson stated the deceased's tissues were very friable or fragile[10]and as a result small tears in the mesentery were not uncommon or concerning unless there was evidence of bleeding which there was not.

For the purposes of the operation Dr Highman recorded intubation of the deceased was easy and, while her airway pressures were in the higher range of normal, she did not have unexpected difficulty in maintaining satisfactory pressure. Any difficulties she considered to be as a result of the deceased's obesity and infective lung process.[11]

Dr Highman was still concerned the deceased was dehydrated and charted 2L of fluids during surgery and provided for 3L post surgery to provide for 7L over the 24hr period.[12] There was an anomaly in the fluid chart signing which meant Dr Highman was not certain the deceased had received all of the charted fluids.[13]

It was Mr Hudson's belief, at the conclusion of the

[10] t 4.3.14 p79,77
[11] t 4.3.14 p59
[12] t 4.3.14 p57
[13] t 4.3.14 p58

procedure, they had effectively cured the deceased. There was no evidence of peritonitis, no evidence the appendectomy had caused a problem and he expected her to make a full recovery to her pre-operative state.[14]

Similarly Dr Highman said in evidence she expected the need to use aramine post-operatively would taper off as the deceased recovered and this was supported by the recovery room observations.[15]

RECOVERY ROOM

The recovery room record on the reverse of the pre-operative anaesthetic record in the deceased's medical notes commences at 1:50pm with a blood pressure of 160/90 and continues until 3:45pm with some overlap with HDU observations.

In recovery the deceased was extubated on her right hand side and Dr Highman noted a fall in her oxygen saturation to 90% which required suctioning, a plug of mucus was removed.[16] Dr Highman initially had some difficulty in maintaining the arterial line due to the deceased's agitation but once the aramine infusion was recommenced and titrated to the appropriate level this decreased.

[14] t 4.3.14 p81
[15] t 4.3.14 p63
[16] Ex 1, Tab 8 p3

Dr Highman recorded the deceased as initially distressed and restless when in recovery which made it difficult to reconnect the arterial line and monitoring devices. She noted a difficulty in maintaining the arterial line blood pressure and, while in recovery, the deceased's blood pressure varied as did her pulse rate. The oxygen saturations remained normal and the deceased's final set of observations indicate readings all within the normal range, including her blood pressure being 126/62.

Mr Hudson did not see the deceased once she had returned to recovery but was satisfied her condition was apparently satisfactory because he was not called to assist her.

Dr Highman had already indicated the deceased should be transferred to the High Dependency Unit (HDU) following her recovery due to the existence of her pre-operative co-morbities. Dr Highman believed the deceased would require close monitoring.

Dr Highman believed she did not leave recovery until 3:40pm and was confident she would have given Dr Stace a substantial hand-over.[17] Dr Stace does not recall reviewing the deceased in the recovery suites or receiving a hand-over from Dr Highman who he knew from an earlier practice in the Northern Territory.

[17] t 4.3.14 p63

HDU

Clinical Nurse (CN) Regan Howie was on duty in the HDU on the afternoon of the 18 September 2008 when the deceased was transferred from the recovery room to the HDU.

CN Howie was contacted by the recovery room at approximately 3:30pm and informed the deceased was ready for transfer to the HDU. CN Howie went to collect the deceased with a student nurse. The information CN Howie says she was given was that the deceased was ready to go to the HDU following removal of a ruptured appendix. At hand-over CN Howie was informed the deceased had a removal of a "ruptured gangrene appendix".[18] While Mr Hudson had noted the base of the deceased's appendix was gangrenous he had observed there was no rupture and no sign of peritonitis or excessive inflammation.

CN Howie reviewed the deceased's notes and post-operative orders on collection of the deceased. In evidence CN Howie recalled that she had observed Dr Stace, a doctor from Accident and Emergency (A&E), leaving the recovery suites as she arrived at approximately 3:20 – 3.30pm.[19] CN Howie did not recall seeing Dr Highman with Dr Stace and believed on the information she received Dr Highman had already left recovery with an instruction Dr Stace was to be called to review the deceased because she would not be available for

[18] t 5.3.14 p133
[19] t 5.3.14 p134,142/3

consultation.

Dr Stace qualified as a doctor in New South Wales in 1967 and was registered as a practitioner in Western Australia in 1983. He had been a medical officer at the Port Hedland Regional Hospital where he practiced mainly in the Emergency Department and as a GP Anaesthetist from 1995-2007 when he retired. He did an anesthesia up skilling workshop in June 2008 and following that time had practiced in a number of locum positions in regional WA hospitals. At the time of the deceased's death he was a locum at GRH between August and September 2008. Dr Stace recalled he was on the afternoon shift in the Emergency Department on the 18 September 2008.

It was the practice in GRH in 2008 the after hours care of patients in the HDU would be provided by doctors from A&E. Dr Stace did not remember this being the situation[20] and while he believed he may have seen the deceased in the recovery suites while he was visiting another patient does not recall a hand-over, nor signing for medication for the deceased before being called to see her later in the evening.

CN Howie stated that at hand-over the deceased's blood pressure was 110/49 with an aramine infusion running at 5-10mls/hr, oxygen saturation of 99% on 6L on Hudson mask, with a heart rate of 75. Dr Highman had charted a

[20] t 5.3.14 p101

morphine pack to deliver 1mg of morphine every five minutes.

CN Howie recorded in the IPN she had been informed Dr Stace had reviewed the deceased in recovery and he was her doctor to call for review. CN Howie had not been advised the deceased had an arterial line or was receiving infusions and as such had not bought the appropriate monitoring equipment with her from the HDU. She noted the deceased had two IV cannulae in situ and that she needed aramine, titrated to maintain her blood pressure > systole 100. CN Howie noted that apart from the IV cannulae and the arterial line the deceased had an in-dwelling catheter.

CN Howie assessed the deceased as drowsy but easily roused and capable of obeying commands. She had a Glasgow Coma Scale (GCS) of 14 out of 15 and was slightly agitated and restless. The deceased kept removing her oxygen mask despite being asked not to and needing the oxygen. The deceased told the nurse she was pain free and CN Howie found her abdomen was soft due to her central obesity but not distended, as one would expect with intra-abdominal bleeding. The post-operative dressing was dry and intact. The deceased was peripherally hot to touch.

CN Howie returned to HDU to obtain a cardiac monitor and syringe drive for the purposes of the arterial monitor and on return connected the appropriate infusions to connect the

deceased to the HDU monitors. The observations at the end of the recovery room chart correspond to the first observations in the HDU. CN Howie stated they had returned to HDU by 3:45pm and she re-examined the deceased to ensure there had been no deterioration in transit. The deceased was alert on return to HDU and was able to be instructed in the appropriate use of the morphine pump. CN Howie converted some of the lines to enable her to assess their efficiency more appropriately.

Once CN Howie had settled the deceased and checked her management she reviewed the deceased's notes to familiarise herself with the deceased's history to date. As part of that review CN Howie noted the deceased had a low blood pressure pre-operatively with a history of smoking. Due to the deceased's obvious agitation with the Hudson mask she was changed to nasal prongs delivering 2L of oxygen to maintain an oxygen saturation at 97%. The deceased was awake, orientated and able to drink water. All the post-operative orders were followed including CN Howie requesting Dr Stace sign off for additional aramine infusions.

The deceased's observations on leaving recovery can be gleaned from three separate charts, although the majority of the observations following one at 3:47pm are found on the Patient Control Analgesia (PCA) Observation Chart[21] which

[21] Ex 1, Tab 13, Tab EE

starts at 4:30pm with an oxygen saturation of 100%, pulse 68, BP 113/55, is blank for temperature, respirations 12, morphine 1mg, zero pain score, a sedation score of 5, asleep, zero vomiting score and GCS of 14 over 15 and PERLA + 2. Taking that set of observations as the base line it can be seen that at approximately 5pm the deceased's blood pressure dropped to 107/58 despite titrated aramine to support her blood pressure. Her aramine infusion was increased, however, no other action was taken as all other observations remained relatively stable.

At the 5:30pm observations CN Howie became concerned due to a deterioration in the deceased's observations overall despite the increased aramine. CN Howie again increased the aramine and sent a request to Dr Stace in A&E to come and review the deceased immediately. Pending his arrival she commenced gelofusine via rapid infuser in an attempt to increase the deceased's blood pressure. There was still no sign the deceased had an intra-abdominal bleed although she did complain of increased abdominal pain for which she was advised to increase her morphine intake.

Dr Stace stated his first conscious knowledge of the deceased was when he was called to HDU from A&E at approximately 5:30pm. He arrived at 5:40pm and examined the deceased who at that stage was being infused with gelofusine and increased aramine.

Dr Stace had been told she was deteriorating post removal of a ruptured appendix at approximately 5:30pm. All nurses dealing with the deceased believed she had a ruptured appendix. She was being treated with antibiotics. This all contributed to a perception the deceased's deterioration may be related to sepsis (infection).

Dr Stace recalled the deceased in HDU as hypotensive and being supported with aramine infusions. He noted HDU was at capacity and all beds were filled. The nurses were fully occupied with patients. He understood the nurses were concerned the deceased's blood pressure was decreasing despite the aramine infusions and she was complaining of pain. He understood she was being infused with gelofusin. It is clear CN Howie was actively managing the deceased pending his arrival.

Dr Stace noted the deceased had two IV lines attached to the aramine syringe driver and the morphine pump. She had an arterial catheter for arterial pressure and a continuous reading, but no central venous line. He noted the urinary catheter for volume and that she was actively taking morphine.

The deceased was alert and orientated and was breathing freely although her BP was 80/40 and her temperature now 37.3^{0}C. Having asked for a fluid warmer for additional fluid because he believed she required further fluid intake

Dr Stace assessed her medical history to enable him to continue her management. He read her medical history including, the fluid balance charts, the operation charts and the anaesthetic record. The anaesthetic record made him believe the deceased's low blood pressure had been an issue during the operation and the order for aramine post operation indicated the anaesthetist thought low blood pressure may continue. Realistically the deceased was also hypotensive pre-operation.

There was no record of any blood loss during the course of the operation so Dr Stace was satisfied an intra-abdominal bleed was unlikely, especially in view of the type of surgery.

Dr Stace formed the opinion the deceased had hypotension due to septic shock because[22] –

- He believed she had a perforated appendix;
- Her hypotension preceded the operation;
- The anaesthetist had administered aramine throughout the operation;
- The anaesthetist had inserted an arterial line during the operation;
- There was no mention of blood loss during the operation;
- There was a post-operative order for aramine infusion if her systolic blood pressure fell below 100mmHg.

Dr Stace believed her pain was usual post appendectomy pain and the morphine had been ordered to manage that

[22] Ex 1, Tab 9, para 30

issue. Similarly Dr Stace did not believe the deceased was suffering an internal haemorrhage because –

- The hypotension preceded the operation;
- Preoperatively her platelets were within the normal range so she was unlikely to have an increased risk of bleeding;
- There was no mention of intraoperative bleeding;
- The anaesthetist had made provision for a drop in systolic blood pressure post operation;
- Internal bleeding after an appendectomy is extremely unlikely;
- Dr Stace did not believe she had pneumonia.

Due to Dr Stace's value judgment the deceased was suffering from septic shock rather than internal haemorrhage he considered the deceased was suffering a medical problem (sepsis) rather than a surgical or anaesthetic problem. As a result he did not consider consulting with either the surgeon or an anaesthetist, and understood there was no general physician with whom he could consult over her management.

Dr Stace's management of the deceased from that time was on the assumption she was suffering from septic shock and required additional fluids to control her hypotension, whilst balancing that with her need for aramine infusion to assist with her perfusion. Dr Stace ordered additional fluids, checked the urine output and ordered arterial blood gas tests.

The deceased's urine flow ceased at 6:30pm. Dr Stace had a bladder scan conducted which indicated there was no urine. He ordered a frusemide infusion and at about 6:50pm the deceased's urine output restarted. Dr Stace attempted to insert a central venous catheter into the deceased's right femoral vein but the procedure was unsuccessful. This would have given a better indication of the degree of the deceased's hypovolemia (low blood volume).

CN Howie recalled the deceased's aterial blood gas results returning at approximately 7:30pm after being collected at 7:15pm.

When the arterial blood gas print out arrived Dr Stace had difficulty interpreting it. He selected what he believed would be key indicators for the deceased's welfare. The results are recorded in the Integrated Progress Notes and appear on the reverse of Tab T.[23]

Dr Stace found interpretation of the blood gas analysis (BGA) difficult and concentrated on the readings with which he was most concerned. He did not register the extent of the deceased's hemoglobin dilution to 53gms per litre, and the very low haematocrit at 17%. In the stress of attempting to find acceptable ways of increasing the deceased's blood pressure without overloading her with fluids and so cause

[23] Ex 1, Tab 13

cardiac failure, he did not notice those two indicators were critically abnormal. Had he been suspicious of a surgical problem, such as an intra-abdominal haemorrhage, Dr Stace may have been more concerned with a consideration of her hemoglobin levels. They were an indicator there may be an internal haemorrhage because they had been normal pre-operatively.

The deceased's family had arrived during the time things were critical for the deceased and there were a number of things happening which Dr Stace believed he needed to deal with alone. While he considered ringing a consultant at a tertiary hospital he believed the delays in obtaining additional advice prohibited him from managing the deceased competently. She required constant input from Dr Stace, CN Howie and the after hour hospital co-ordinator. Her deterioration was so rapid it is doubtful management for an intra-abdominal bleed would have had time to be instituted.

CN Howie's reading of the arterial blood gases indicated the deceased's oxygen and carbon dioxide levels were only slightly out of the normal range and consistent with her pre-operative results in the A&E. However, it was clear the deceased was acidotic and had high lactate levels. Her blood pressure continued to drop and there was no sustained effect on maintaining a blood pressure above systolic 100mgs Hg from the aramine infusion. The

deceased was becoming agitated and distressed. Dr Stace administered midazolam which calmed the deceased, although she remained confused in her conversation.

By 7:35pm the deceased's conscious level decreased and her systolic blood pressure dropped further to between 40 and 30mgs Hg. Dr Stace considered the deceased to be irretrievable.

The deceased's family had been speaking with the deceased but were ushered out during her decline. Dr Stace went to talk to the family to advise them of the negative prognosis for the deceased and the family was brought in to spend their last few minutes with her.

Dr Stace declared the deceased dead at 7:45pm.

POST MORTEM EXAMINATION

A post mortem examination was undertaken by Dr Clive Cooke, Chief Forensic Pathologist, PathWest, on the 24 September 2008.[24]

Dr Cooke's examination showed a recent appendectomy with haemorrhage into the abdominal cavity and haematoma formation. He also found hardening and narrowing of the arteries and arteriosclerotic

[24] Ex 1, Tab 5

nephrosclerosis. He confirmed gallstones to be present.

On microscopic examination Dr Cooke confirmed the presence of arteriosclerosis with some microscopic scarring of the heart muscle. With respect to the arteriosclerosis of the coronary arteries and aorta he noted a marked degree of calcified arteriosclerosis throughout the left anterior descending and circumflex branches of the left coronary artery, with heavy calcification resulting in narrowing along much of the length to a pinpoint luminal diameter. There were lesser changes present in the right coronary artery and no evidence of thrombosis. Her aorta was also moderately arteriosclerotic along its length. The reduction of the lumen of the left anterior descending coronary artery to a pinpoint diameter is severe coronary artery disease and may have contributed to the deceased's level of pre-operative hypoxia.[25] It also would have put her at risk of serious cardiac complications such an infarction, arrhythmias or acute heart failure.

Microbiological analysis showed a mixed bacterial growth typical to most post mortem findings and there was no evidence of serious sepsis although the deceased had been provided adequate antibiotic cover.

Dr Cooke located two tears of the mesentery extending into the region of the haematoma in the deceased's abdominal

[25] t 7.3.2014 p12 (Davis Inquest)

cavity around the mesentery of the terminal ileum.

It would appear the tears in the mesentery were associated with the significant blood loss into the deceased's abdominal cavity and would have accounted for her lowering blood pressure, hypoxia, and low haemoglobin.

There had been no evidence of intra-abdominal bleed at the time of the operation and it is a very rare event for tears in the mesentery to bleed to the extent apparent in the deceased. Neither Mr Hudson nor Dr Stace could recall another instance, in their experience, of an intra-abdominal haemorrhage resulting from a mesenteric tear in an appendectomy.

Dr Cooke found the deceased's cause of death to be Intra-abdominal Haemorrhage following Appendectomy and Coronary Arteriosclerosis.

The coronary arteriosclerosis was a serious pre-existing condition even though the deceased had not had obvious cardiac problems. The narrowing of her coronary arteries would have adversely affected her cardiac function and consequently oxygenation of her organs regardless of the added insult of an operation complicated by serious haemorrhage.

In evidence Mr Hudson stated he would generally expect to

be called when one of his patients deteriorated post-operatively. He had not understood the deceased had died until he attended the hospital the following morning to make a ward round of his post-operative patients.

Mr Hudson's evidence was that in the event there was a significant tear in the mesentery observable at the time of the operation he would have repaired it. However, it was very unusual for tears in the mesentery to bleed in the way the tears found at post mortem had bled unless they were in very close proximity to a blood vessel.

He agreed the deceased was unwell and because of her general hypoxia, hypotension and central obesity (in the abdominal cavity) visibility was difficult. He had made an incision which was considerably larger than the normal incision to assist him in being able to visualise the appendix in the abdominal cavity.

The deceased tissues were very fragile (friable) and he had been very careful in an endeavor to ensure there was no excessive damage when attempting to visualise and remove the appendix. The fact of there being tears in the mesentery in such friable tissue was not surprising. However, from his perspective what was surprising was the fact he had not seen them and certainly seen no bleeding which would indicate there was a problem he might need to address.

Mr Hudson believed the tears may have occurred while releasing tissues and closing the abdominal cavity as a result of the release of tension on the tissues needed to achieve visibility and work space.[26]

He did recall being surprised at the level of the deceased's apparent shock but believed, in view of the fact she did not appear to be suffering infection, it was probably as a result of her known lung disease. So, while the state of the deceased's appendix did not account for a shocked state, there were other comorbidities which may have explained her pre-operative hypoxia and hypotension and supported the anaesthetist's view they were dealing with dehydration and sepsis.

Mr Hudson did not believe the operation had been difficult other than the need for an extended opening to assist with visibility and work within the abdominal cavity which is why he had not made any notes either in the operative record or in his personal notes, as to any difficulty with the procedure. He confirmed he had never personally had an experience with mesenteric tears bleeding to the extent that apparently occurred in this case but hoped, had he been called at about 5:45pm, he would have decided a return to theatre by 6:15pm was an appropriate course of action.[27]

[26] t 4.3.14 p71
[27] t 4.3.14 p86

While Dr Stace's review of the circumstances led him to believe it was not a surgical issue, rather a medical issue arising out of the deceased's presumed septic shock, I speculate a surgeon would approach the available indicators from the perspective of a surgeon. This may have presented an alternative view for the deceased's deterioration, but may still have been too late to have altered the outcome.

MR CHILDS' EVIDENCE

The input of Mr Philip Childs, General Surgeon, was sought with respect to the post-operative management of the deceased, in view of the fact it appeared from the post mortem findings there had been an intra-abdominal haemorrhage arising out of tears in the mesentery during the course of the appendectomy to remove the deceased's gangrenous appendix.[28] The significant matter was how quickly the deceased had deteriorated post-operatively when there had been no indication at the time of the operation there may be an intra-abnormal bleed.

Essentially, Mr Childs explained the circumstances as he believed they had occurred on the evidence he had reviewed. By the time of Mr Childs' review it was known the deceased had suffered an intra-abdominal haemorrhage.

In Mr Childs view, and supported by the medical history,

[28] Ex 1, Tab 15

the deceased was dehydrated on presentation to ED GRH. She had been unwell and she had been vomiting. This would have caused a certain level of dehydration and this was recognised and managed by the administration of appropriate fluids. However, Mr Childs indicated in retrospect she should probably have been rehydrated more vigorously than had been done pre the operation.

The deceased presented as hypotensive and hypoxic. The fact the deceased's dehydration had been attributed to a septic appendix and her COAD was not surprising. There is always a concern with fluid overload where potentially there may be a problem with kidney or liver functions. The perception the deceased was dehydrated but should not be aggressively hydrated, resulted in the deceased remaining essentially dehydrated as evidenced by her declining blood pressure.

Due to the belief her blood pressure was low because of her sepsis, steps were taken to elevate the deceased's blood pressure by the use of drugs.[29] The fact aramine was the drug used was an appropriate choice in the circumstances, but still resulted in the deceased remaining dehydrated and falsely elevating her urine output at the cost of volume and resulted in increasing hypovolemia. This caused a continuing decrease in blood pressure.

[29] t 4.3.14 p22

Mr Childs emphasised in retrospect, with knowledge of the fact there had been an intra-abdominal bleed, the use of aramine without sufficient fluids meant the deceased never overcame her dehydration problem, and the continuing aramine infusions exacerbated the dehydration problem due to falsely elevating the deceased's urine output.

Mr Childs was anxious to confirm the value judgments the clinicians had made at the time of the operation had been reasonable on the information they believed they had.[30] It masked the fact the drop in the deceased's blood pressure following the operation was due to the intra-abdominal bleeding which also elevated the need for fluids. He did not think it unusual there had been no evidence of bleeding at the time the deceased's abdomen was closed and agreed it was extremely unusual to have this level of bleeding from a patient with mesenteric tears alone.

Mr Childs indicated it was not "out of the ball park" to put the explanation for the decreasing blood pressure into the sepsis category rather than the haemorrhage category, despite Mr Hudson's note the appendix was not ruptured and there was no peritonitis.[31] The clinicians' belief in the extent of the deceased's lung disease, and her record of yellow sputum pre operatively would have supported this view.

[30] t 4.3.14 p23
[31] t 4.3.14 p30-34

From Dr Stace's perspective I note all the nurses were also of the view the deceased had a ruptured appendix removed. This would have further supported his belief he was dealing with a decreasing blood pressure due to septic shock. Mr Childs indicated this was not unreasonable, but with the benefit of the post mortem examination, wrong.

The post mortem report disclosed the mesenteric tears, not noted at operation and masked by the maintenance of the deceased's blood pressure and the production of urine, as a result of the aramine used.

Mr Childs indicated that from his perspective, as a general surgeon, the clue to the reason for the deceased's deteriorating condition was the decreasing blood pressure despite the aramine infusion. He believed the need to return the deceased to theatre could have been determined, but unfortunately she deteriorated extremely quickly, and it was not understood before her death the clues were for an intra-abdominal bleed.

Mr Hudson, as a surgeon, stated in evidence an intra-abdominal bleed was something all surgeons are aware of in the circumstances of the deceased and any abdominal surgery. He would have expected to receive a call at approximately 5:45pm, after Dr Stace had first reviewed the deceased. He believed he would have needed to see the

deceased, which he would have done had he been called. He hoped he would have been able to provide a plan to return her to theatre by about 6:15pm.

All the surgeons agreed there is some doubt as to whether there would have been enough time to supply the deceased with sufficient blood products and return her to surgery to stem the bleed. Realistically, that was the only treatment which would have prevented the deceased's death. It is unclear, due to her very rapid deterioration, that could have been achieved in time to change the outcome. On the information available, and on the precautions taken by the anaesthetist to support the deceased's blood pressure, it was understandable Dr Stace had not recognised the available clues.[32]

Mr Childs indicated one would need to have had a high degree of suspicion there was the potential for an abdominal haemorrhage.[33] I speculate that degree of suspicion was more likely in a surgeon, due to their particular expertise and clinical judgment weighting. It was also supported by the low haemoglobin levels post operatively.

Overall, Mr Childs believed the clues the deceased was suffering an intra-abdominal haemorrhage were there post operatively and prior to her demise, but were easier to

[32] t 4.3.14 p33
[33] t 4.3.14 p34

assess in hindsight, with the information available from the post mortem examination. Before death the deceased was urgently in need of blood to replace the 1600mls located in the intra-abdominal cavity at post mortem and to correct the severe haemodilution evidenced in the blood test results.[34] There was a need for haemoglobin to carry oxygen to reverse the deceased's serious hypoxia.

CHANGES AT GRH WHICH MAY HAVE IMPROVED MANAGEMENT OF THE DECEASED

The inquest heard evidence from Dr Andrew Jamieson, Regional Medical Director WA Country Health Service (WACHS) Mid West. Dr Jamieson was not regional director at the time of the death of the deceased and started his role in June 2012. The role involves clinical governance of the region for the professional medical staff. The position is based at GRH and is responsible for the other smaller hospitals in the area. Dr Jamieson maintains a clinical role in the area by providing clinical services to the Aboriginal Medical Service.

Dr Jamieson holds a specialisation in remote and rural health. He is experienced in the provision of health services in remote regions having practised for a considerable amount of time in Northern Territory.

[34] Ex 1, Tab 13, Reverse Tab T

Dr Jamieson was asked specific questions relating to the provision of medical cover in GRH at the time of the deceased's death and Dr Stace's perception he could not call for assistance. One of the difficulties at the time of the deceased's deterioration was that it occurred at approximately the time GRH, in 2008, would have experienced a shift change resulting in most of the doctors going off duty. Dr Stace was rostered in A&E and in 2008 HDU after hours cover was provided by the ED doctors. While there was an experienced general physician employed by GRH in 2008 he only worked core hours to maintain his functionality on an ongoing basis. This may have contributed to Dr Stace's perception he was not able to call on the advice of a general physician easily once GRH was officially "out of business hours". Dr Jamieson also pointed out part of remote medicine is the appreciation of the fact one does actually have to be self-sufficient and that, in conjunction with a perception one is not expected to seek help, may create a particular "mind set".[35]

On 18 September 2008 there were other doctors working at GRH after 4pm, however, none of those were consultant general physicians which was the assistance Dr Stace believed he needed. In hindsight it is apparent the assistance of Mr Hudson could have been sought and would have been relevant, but this was something Dr Stace was not minded to do in view of the fact he believed he was

[35] t 5.3.14 p175

dealing with a septic shock situation rather than intra-abdominal haemorrhage.

In 2014 there are three full time general physicians available for consultation and assistance.[36]

With respect to the actual mechanism of how the tears arose Dr Jamieson advised surgeons are now assisted by surgical registrars during operations. He believed that the assistance of a surgical registrar in the operation on the deceased would have lessened the potential for there to be excessive traction problems when operating intra-abdominally on a patient with friable tissues.

With knowledge of the post mortem report Mr Hudson indicated the assistance of a surgical registrar during abdominal surgical procedures could well have improved his ability to remove the infected part of the deceased's appendix with less disturbance of the friable tissues.[37] It was Mr Hudson's view, from the post mortem report, the fact the tears in the mesentery appeared to be horizontal reflected they were caused by the release of traction during the operation. There was no bleeding observable at closure which led him to believe the tears occurred as traction was released and tissues returned to the abdomen in their post-operative position. He believes the bleeding occurred post

[36] t 5.3.14 p177
[37] t 4.3.14 p89

operatively. In addition, had he been called at approximately 5:45pm when the deceased's blood pressure continued to decline despite the increased aramine infusion, he believes he would have suspected the possibility of an abdominal bleed due to his knowledge of the state of the deceased's tissues.[38]

In addition the ABG analysis indicated the deceased needed blood products which would have also provided volume. The earlier ordering of ABG analysis may have allowed provision of suitable volume expanders.

Mr Hudson would have understood the deceased required fluids/blood and to be returned to theatre as quickly as possible. Waiting for a CT scan to support a bleed would have only delayed a procedure which needed to be done in any event.

One of the matters which has improved greatly since 2008 is the availability of surgical registrars to assist surgeons during surgery, and the availability of afterhours consultants. Dr Jamieson was able to advise that generally hospital wise, there is now much improved medical cover which allows doctors to call for assistance and discuss deteriorating patients in a more supportive and collaborative environment.

[38] t 4.3.14 p93

Evidence was also heard from afterhours Hospital Coordinator Senior Nurse Angela Joy Monaghan, with respect to improvements which have been made which would assist nurses treating patients in a similar situation to that of the deceased. SRN Monaghan was on duty at the time of the deceased's death and became involved with her management while Dr Stace was attempting to stabilise the deceased as she deteriorated. SNR Monaghan became involved with the provision of additional aramine and a fluid warmer for fluid resuscitation.

One of the improvements in the management of deteriorating patients has been the implementation of the adult observation and response chart (the rainbow chart) which uses colour to represent the different levels of observations requiring more senior or professional input.[39]

In the case of the deceased the observations were adequately documented throughout the deceased's medical file by way of various charts. The pre-operative and operative records are clear, as are the recovery room and the HDU records. However, there is still the issue of the observations being spread over a number of different charts. Provided the rainbow chart is used appropriately, the trend in a patient's overall responses is very much easier to follow.

In addition, there are clear indicators at which points

[39] Ex 4

increased surveillance or elevated review is necessary. In cases where there may have been some reluctance for nurses to involve clinicians in a patient's isolated responses, it is now predetermined that in specified circumstances they are required to take specified action. This prevents a culture of "when do we need to invoke more senior review?" It is delineated.

In this case, despite the fact the deceased's observations were appropriately recorded, the trend in her overall deterioration is not distinguishable as easily as is the case when plotting the results on a rainbow chart. In evidence, SRN Monaghan was asked to fill out a representative chart with the deceased's observations once in the HDU. From that charting SRN Monaghan was able to say it would have been appropriate for medical review to have occurred approximately half an hour earlier than was the case with the deceased. This was because the difficulties with her blood pressure became obvious earlier in a graph representation of the observations.

There is no suggestion CN Howie did not institute appropriate surveillance or reviews to the deceased's deteriorating state. She did. She took action to improve the situation for the deceased by appropriate management pending medical review. She also called for assistance from Dr Stace at an appropriate time. It is just the contemporaneous completion of a chart such as the rainbow

chart would have made the deceased's overall condition very observable to Dr Stace when he attended.

The fact the deceased was also requiring more and more oxygen support would also have been a matter to have involved a clinician earlier. It is not suggested this would necessarily have helped in the case of the deceased, whose observations were comprehensive in this matter but the information being so readily accessible does emphasise a deterioration which may have instigated arterial blood gas analysis earlier. A base line post operatively from which the deceased's very rapid deterioration would have been easier to assess, in conjunction with the arterial blood gas evidence of hemodilution, would have supported a blood loss, rather than sepsis.

The availability of additional medical support for Dr Stace may well have facilitated the ability to interpret the arterial blood gases effectively and provide the deceased with blood supplements, pending a rapid return to theatre.

Discussion with Mr Hudson on the appropriate completion of a rainbow chart may well have brought a surgical perspective to the deceased's deterioration at a slightly earlier stage which potentially could have supported earlier intervention and provision of blood products.

Regardless of the potential for improvements to have

facilitated earlier appreciation of an accurate diagnosis for the cause of the deceased's hypotension, the judgments made at the time on the information which included an erroneous belief the deceased was suffering sepsis, a situation Mr Hudson may have understood to be unlikely, were not unreasonable.

CONCLUSION AS TO THE DEATH OF THE DECEASED

I am satisfied the deceased was a 57 year old woman suffering central obesity, hypertension, and, unbeknown to the physicians, significant arterial atherosclerosis.

On 17 September 2008 the deceased was unwell and vomited to the extent she presented to GRH. By the time she presented to hospital she had become hypoxic and hypotensive. In hindsight this could indicate potential for a more significant degree of dehydration than was recognised by the doctors dealing with her. In addition, she suffered COAD and the doctors noted a productive cough with yellow sputum. There was a basis for an infective process to be considered.

The deceased was diagnosed with appendicitis and prepared for urgent surgery the following day.

The anaesthetist GP for the procedure on

18 September 2008 was Dr Highman. Pre-operatively she was aware of the deceased's hypotension and hypoxia and made provision during the operation for fluid support, which had already been instigated, and the use of a gentle drug to elevate the deceased's blood pressure artificially by constriction of the vessels.

The operation was undertaken by Mr Hudson who, realising there would be a visibility problem made a large incision to assist him with the procedure. It was necessary he push aside various tissues in order to elevate the appendix and deal with the problem. Mr Hudson observed the base of the deceased's appendix was gangrenous and needed to be removed, however, the appendix did not appear to be ruptured, there was no peritonitis, and as such there should not have been a serious source of infection from the abdominal procedure. When Mr Hudson mentioned the lack of a source for the deceased's perceived infection to the anaesthetist it was considered there was also the issue of COAD which may account for the pre-operative level of hypoxia.

Mr Hudson was operating unassisted by a registrar and needed to manage the deceased's tissues and organs alone by way of clamps which would have put significant tension on the mesentery.

During the course of visualisation of the appendix and the

appropriate removal of the gangrenous parts Mr Hudson was mindful of the potential for tears in the mesenteric tissue but did not observe any tears of concern. On completion of the procedure and suturing off the exposed surfaces, the tissues were returned to position in the abdomen and the abdomen closed. It is likely it was at the point of closure the tears arose which accounts for there being no bleeding observable, and no ooze at the time.

The deceased was taken into recovery and appeared to be recovering well and any fluctuations in blood pressure were dealt with by the aramine infusion. There was no reason at this stage for the clinicians or nursing staff to expect the potential for an intra-abdominal haemorrhage.

The deceased was agitated in recovery but appeared to respond well to management. Dr Highman was comfortable her situation was appropriate at the time she handed over to Dr Stace. I suspect handover may not have been as fulsome as Dr Highman believes was her usual practice and as a consequence Dr Stace, who believes he was in recovery for another purpose, does not recall involvement with the deceased at that time.

CN Howie did not see Dr Highman in recovery whilst she was there from approximately 3:20-3:40pm, however, she did see Dr Stace leaving the area and was informed Dr Stace had reviewed the deceased.

Handover to CN Howie informed her the deceased had suffered a ruptured appendix. This set the scene for the perception of all those dealing with the deceased thereafter to believe there was the potential for peritonitis and sepsis, in addition to COAD.

The post mortem examination found two significant tears in the mesenteric tissues and 1600mls of blood fluid and clot in the abdominal cavity.[40] This would suggest that once the deceased commenced to bleed she did so rapidly. The fact her continued hypotension was as the result of blood loss rather than dehydration and sepsis was masked by the reasonable, at the time of operation, use of aramine.[41]

Dr Stace was called at approximately 5:30pm to review the deceased due to the inability of the HDU to maintain a reasonable blood pressure for the deceased. Dr Stace reviewed the available information and believed the deceased to be suffering septic shock. She was being administered antibiotics and he saw his role as attempting to stabilise her blood pressure and support her medical management.

Dr Stace's review of the medical notes tended to confirm his view he was dealing with a patient declining due to septic

[40] Ex 1, Tab 5
[41] t 4.3.14 p23-25

shock and he had a "mindset", as he termed it, he was not in a position to call for assistance from a general physician. Due to his view it was a medical problem it did not occur to him it may be a surgical or anesthetic problem and he did not call Mr Hudson or the on call anaesthetist for a different perspective.

The deceased continued to decline and Dr Stace continued to administer fluids and elevate the aramine infusion while checking her urine output to ensure she did not suffer fluid overload due to cardiac failure. Although her urine output did stop at one stage it then continued and it appeared the elevated aramine was working. It was not until Dr Stace believed the deceased to be suffering from oedema he used frusemide because he was still of the view he was dealing with a septic shock reaction. He did not note the severely low haemoglobin levels in the arterial blood gas analysis and had been unable to insert a central venous line which would have given him a better indicator of the deceased's fluid balance. Both these investigations would have directed a consideration of hypovolemia, that is a loss of blood volume, indicative of the loss of blood.

Dr Stace was unable to stabilise the deceased and she rapidly deteriorated between 5:30pm and the time of her death at 7:45pm. It had not occurred to anybody the deceased may be suffering from blood loss.

I accept intra-abdominal haemorrhage, especially to the extent seen in this case, is an extremely rare complication of an appendectomy. The deceased was already compromised by a decreased respiratory affectiveness due to lung and artery disease. None of the clinicians involved had experience of intra-abdominal haemorrhage following appendectomy. As a result there was no suspicion raised that the observations which indicated the deceased was deteriorating may be accounted for by blood loss rather than septic shock. Nor was attention given to the indicators which may have supported blood loss such as the significantly lowering haemoglobin levels against normal levels preoperatively.

The expectation the deceased was suffering from septic shock and the focus of Dr Stace on trying to stabilise the deceased prevented an appreciation of the "clues", and resulted in misdiagnosis of the reason for the deceased's deterioration until the results of the post mortem examination. The fact of an intra-abdominal haemorrhage was not suspected, despite knowledge of recent abdominal surgery.

In view of how rapidly the deceased deteriorated and died, it is impossible to predict with certainty whether appreciation of a correct diagnosis earlier would have provided a different outcome. The deceased needed blood or blood products in sufficient quantities to replace the loss and provide

appropriate perfusion, and surgery to correct the source of the blood loss, if it could be located.

In all the circumstances I find death arose by way of Misadventure.

E F VICKER
Deputy State Coroner
27 May 2014