Coroners Act, 1996
[Section 26(1)]

Western *Australia*

RECORD OF INVESTIGATION INTO DEATH

Ref No: 35/13

I, Evelyn Felicia VICKER, Acting State Coroner, having investigated the death of ***Wilma Ray Jones,*** *with an Inquest held at Geraldton Coroners Court, Geraldton, on the 19-21 August 2013 and Court 58, Perth Coroner's Court, 501 Hay Street, Perth, on 23 August 2013 find the identity of the deceased was* ***Wilma Ray Jones*** *and that death occurred on 21 March 2009 at Geraldton Regional Hospital as the result of Abdominal Infection Following Surgery (repair of recurrent umbilical hernia) in the following circumstances:-*

Counsel Appearing :

Ms K Ellson assisted the Acting State Coroner
Mr S Denman (instructed by Denman Popperwell Lawyers) appeared on behalf of Doctors M Hudson and G Greenhill
Ms W Meggison (instructed by Panetta McGrath) appeared on behalf of Dr L Nash
Ms R Young (instructed by State Solicitor's Office and with her Ms Philpot) appeared on behalf of WA Country Health Service.

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INTRODUCTION

Wilma May Jones (the deceased) attended Geraldton Regional Hospital (GRH) on 19 March 2009 for a procedure to repair a recurrent umbilical hernia. The operation was completed by Mr Hudson without any apparent complications and her recovery from surgery was satisfactory. She was returned to the general ward as a surgical patient.

That evening she reported pain and later vomited. This appeared to stabilise to a level of pain consistent with her operation.

The deceased was reviewed on 21 March 2009 and various investigations undertaken. Her kidney function was impaired. The deceased did not recover and she developed acute renal failure.

The deceased died on the morning of 22 March 2009.

The deceased was 75 years of age.

BACKGROUND

The deceased was born on the 26 July 1933 in Kalgoorlie as the middle child of five. She had two older sisters and two younger brothers. She was raised in Kalgoorlie and there met her husband where they married and had four sons and two

daughters. The deceased was an active member of her community and also made her own porcelain dolls, which she then dressed. This incorporated many of her hobbies.

In the early 1990s the deceased and her husband retired to Jurien Bay where the deceased maintained an active lifestyle as part of that community as well as continued input with her, by now, extended family.[1]

MEDICAL

According to the statement of the deceased's husband, Mr Reginald George Jones (now deceased) the deceased had always been in good health. He recalled the first indication of a problem for his wife with respect to a hernia, was some time in 2008.

According to Mr Martin Hudson, he first saw the deceased on 11 July 2008, after referral from a doctor in Jurien Bay, with the recurrence of an umbilical hernia. The hernia had been repaired previously, although not by him. He repaired the hernia at Moora District Hospital on 22 October 2008. The hernia contained colon and omentum and the defect in the umbilical cicatrix was closed with a Ventralex mesh patch. On Mr Hudson's reviews the deceased's recovery was uneventful.

[1] Obtained via email from Mr Jones Jnr

The facilities at Moora District Hospital were not as comprehensive as those at GRH but there were no concerns about the deceased's ability to recover successfully from that type of operation in a relatively remote setting.

In February 2009 the deceased again complained of a bulge in her abdomen, near the original hernia scar. She was referred back to Mr Hudson, due to her complaints. The hernia appeared painful and the overlying skin was slightly reddened.

Mr Hudson examined the deceased on 20 February 2009 and could feel the Ventralex mesh he had inserted in the earlier repair had migrated and was palpable in the subcutaneous tissues deep to the previous scar. The umbilical hernia reduced when the deceased lay down and Mr Hudson considered she required another hernia repair.

In evidence Mr Hudson stated that on this occasion the deceased was getting episodes of pain in the hernia and gave a history of reddening of the skin. He believed the repair should be done at a more sophisticated hospital than Moora, which would be able to provide more comprehensive care and review.

Mr Hudson placed the deceased on the urgent waiting list for repair of the recurrent umbilical hernia and completed

the relevant paperwork for her admission to GRH. The deceased signed a consent form to the surgery after the consultation and Mr Hudson's discussion with her about the risks associated with any surgery.

Mr Hudson did not consider the deceased to be at high risk of venous thromboembolus (VTE) despite the fact she was classified as obese (117 kgs BMI > 35) and she was over 60 (75) years of age. She was a non-smoker, did not have hypertension nor any history of any concern. He considered an umbilical hernia repair to be a simple, short procedure and non-intrusive of the abdominal cavity. In Mr Hudson's view one weighted prospective surgical patient's overall clinical presentation for the risk of VTE against an increased risk of intra-operative or post-operative bleeding if prophylactic Heparin was to be used. Mr Hudson said it was unlikely he would have suggested the use of prophylactic Heparin.

Surgery was planned for 19 March 2009 at GRH.

On 20 February 2009 the deceased was also reviewed by Dr Beckett (anaesthetist) for a pre-anaesthetic check. Dr Beckett noted the deceased appeared to have a cardiac murmur which he required be investigated by use of an echo. There is a note on the deceased's Pre-Operative Anaesthetic Check that was undertaken on 9 March 2009.

Dr Beckett reviewed the preliminary report of her echo as 'normal'.[2] The only medication Dr Beckett was aware of the deceased taking was paracetamol.

On Thursday 19 March 2009 the deceased and her husband left Jurien Bay early to make sure they arrived at GRH by 10.30am. The deceased did the driving and once they had arrived and checked into the hospital, Mr Jones returned to Jurien Bay awaiting his wife's operation and recovery.

The GRH records indicate that on admission the deceased's medications were recorded as Panadol and Naprosyn and, due to her age, obesity, and type of surgery, from a nursing perspective she was considered to be at high risk of VTE and therefore TEDS stockings were measured and fitted as part of the nursing protocol. Other decisions with respect to prophylactic thrombo-embolics are surgical decisions and had already been assessed by Dr Beckett and Mr Hudson as not required pending the surgery.

SURGERY

Mr Hudson from his notes and perusal of the GRH file informed the court the procedure commenced shortly after 2.30pm on Thursday 19 March 2009. There was a 10x5cm defect in the linea alba and the Ventralex mesh patch he had inserted in October 2008 had become dislodged and

[2] t 19.8.13 p70

moved. Adhered to it was a small 5cm segment of the wall of the small bowel which required separating from the mesh. After the separation Mr Hudson checked to ensure there was no perforation of the small bowel by both visual inspection and by attempting to observe air bubbles following gentle pressure. He checked the vascularity of the bowel was normal by inspecting its colour and viability and then performed a Mayo overlapping repair of the linea alba, using two layers of interrupted and continuous Prolene sutures. The overlying skin was then stapled.

Due to the adherence of the small bowel to the mesh the procedure to repair the hernia took longer than was usual for this type of operation. Mr Hudson confirmed the procedure took approximately 75 minutes, rather than the expected 30 minutes. The complications arose due to the need to take care in freeing the adhesions from the mesh and ensuring no damage to the bowel. Mr Hudson indicated it wasn't a problem with the deceased's obesity, rather the mesh had risen a considerable distance from the original repair and brought a loop of the bowel with it closer to the surface. Originally, due to the fact the deceased's hernia reduced when she lay down, he had suspected no bowel to be caught in the mesh, but this was not the case when he actually performed the surgery.

Mr Hudson was relatively confident he had not damaged other areas of the bowel or intestine by movement during his repair of the hernia.

From the anaesthetist, Dr Beckett's, perspective the operation was unremarkable other than the time it involved. On two occasions the deceased's blood pressure had fallen below 100 systolic and her pulse rate had slowed to 50 beats per minute. As a result Dr Beckett had used ephedrine and atropine to improve her blood pressure. On both occasions, after the administering of the appropriate medication, the deceased's blood pressure and pulse rate improved and Dr Beckett was satisfied the deceased's drop in blood pressure and pulse rate were not of concern. Dr Beckett advised it was a matter of using medication to adjust for the fact the deceased had been given other medications such as morphine and anaesthetic drugs. These naturally drop pulse and blood pressure, which is why it is necessary to monitor those signs to ensure the situation doesn't become critical. Dr Beckett advised the inquest this was perfectly natural, especially in the circumstances of the deceased, and wasn't considered by him to be anything out of the ordinary.

Dr Beckett made the comment he did not consider the deceased to be particularly high risk with respect to surgery as, although her weight was outside the healthy range, she

had no systemic disease that the clinicians were aware of prior to the surgery, she was on very few medications and was living quite well independently. From a cardiovascular perspective there was nothing about the deceased's presentation pre-operatively to alert the clinicians to any cardio-vascular problems. As far as the clinicians were concerned all the deceased's clinical signs were supportive of the fact she was managing her life well.

The operation and the deceased's immediate recovery period accorded with the clinicians expectation the deceased would recover successfully from her surgery.[3]

The deceased arrived in the recovery room from theatre at around 4pm and her recovery appeared to be uneventful. Dr Beckett and Mr Hudson completed the postoperative directions for medical and nursing staff to ensure adequate hydration of the deceased post-operatively, and appropriate analgesia in the event she suffered pain. Dr Beckett said the failure to prescribe thromboembolic prophylaxis (Heparin or Clexane) would be due to Mr Hudson's discussion with him. They considered it to be unnecessary in the deceased's case.[4]

Mr Hudson also had standing orders for the ward staff for post operative surgical patients.

[3] t 19.8.13 p71
[4] t 19.8.13 p77

Nurse Glass collected the deceased from recovery and took her to the general ward where she was a surgical patient at approximately 6pm.[5]

The deceased's observations following her return to the ward postoperatively are recorded as "satisfactory" and the drug chart shows the anaesthetist had written the deceased up for Tramadol, Oxycodone, Maxolon, Ondansetron, Fentanyl and Morphine.

A nursing note written at 3am on 20 March 2009 (Friday) indicated the deceased had earlier complained of pain and given it a score of 8/10. The indication is this was at the commencement of the shift which was presumably around about 9 o'clock the previous evening. The deceased was noted as distressed and diaphoretic (sweaty). Later it was recorded she had vomited approximately 200mls.[6] By the time the note was written at 3am the nurse observed the analgesia had good effect and the deceased was now sleeping. She was receiving IV fluids as prescribed by Mr Hudson in his postoperative plan. The deceased was recorded as sleeping for long periods, without complaint despite the vomiting.

[5] t 19.8.13 p90
[6] Exhibit 1 Tab 14 Integrated Progress Notes

20 MARCH 2009

Mr Hudson gave evidence he reviewed the deceased on the morning of the Friday (20th) and at the time he reviewed her, the deceased's IV fluids were progressing as he had charted postoperatively. He noted in his personal notes[7] he had conducted a ward round and in evidence stated he usually conducted a Friday ward round between 8 and 9am due to a requirement he begin consulting at 9 o'clock in his rooms. On the evidence of Nurse Little this would have been before the deceased's shower.

On his ward round Mr Hudson's personal notes record the deceased as appearing satisfactory with normal observations and that she should remain on sips of water until she had passed flatus. She had recorded a normal oral intake of fluids. He palpated her abdomen which was soft with no tenderness and ordered bloods be collected including a full blood count and urea and electrolytes (U & E). Mr Hudson stated his intention was to check the deceased's haemoglobin to reassure himself there had been no internal bleeding and also to check her white cell count and or creatinine for possible infection. His expectation was that the nurse and medical practitioner accompanying him would write up the ward round and arrange for the bloods. He had no expectation he would be contacted with the results unless there was a problem. The fact of Mr Hudson's

[7] Exhibit 1 Tab 9 page 4

morning review on the Friday does not appear in the Integrated Progress Notes (IPN) and it is unclear as to the results of any of the tests he ordered.

A nursing note at 1.15pm on 20 March 2009 recorded the deceased had been medicated as per her charts and the MR140. She was tolerating small amounts of clear fluid and self caring with daily living. She required supervision when ambulating due to being unsteady on her feet and the note records her IV cannula fell out while she was showering herself in the morning.

The evidence from Nurse Little[8] estimates this to have been at approximately 9.30am. Nurse Little then escorted the deceased back to bed and recorded the fact the deceased had vomited twice during that shift with an antiemetic being given with good effect. The deceased had rested in bed for most of Nurse Little's shift but not voiced any further complaints. It appears there was no IV competent nurse on the ward at the time and the deceased's cannula was not reinserted.[9]

There is an entry in the IPN by Dr Nash to indicate he was asked to assess the deceased at an unspecified time. Dr Nash was the medical, rather than surgical, registrar and his note indicates he found the deceased to be vomiting

[8] t 19.8.13 p104
[9] t 21.8.13 p271

and dry and not tolerating oral fluids with severe nausea. He reinserted the deceased's cannula and her intravenous fluids were commenced again at 3pm, according to the fluid charts, at a rate of 1 litre every 12 hours.[10]

In evidence Dr Nash clarified he was only assessing the deceased for the purposes for which he had been called; that is her presentation at that time, and he did not consider she may have been dehydrated as a result of any form of sepsis at that stage.

Dr Nash said he gave a moderate rate of hydration due to the fact he was trying to strike a balance in an older woman. He stated:

> Giving intravenous hydration too quickly can precipitate pulmonary oedema. However there is a balance, and it wasn't clear to me just how much oral fluid she was tolerating, so I really struck a middle path. And I think, with the benefit of hindsight, I probably would have introduced them faster.

Dr Nash was attempting to ensure the deceased had hydration but not too quickly so as to cause her any difficulties.

It became apparent later in the evidence Dr Nash had been called to assess the deceased due to there being no medical cover for surgical patients on the general ward at

[10] t 23.8.13 p302

that time. The roster for the Friday indicates a gap in surgical coverage for the general ward.[11]

From the evidence it would appear Dr McMillan, who at that stage was a junior surgeon, assisted with the deceased's surgery on the 19th, and during the morning of the 20th was in theatre assisting the urologist. For that reason the surgical patients had to rely on the input from the medical doctors in the absence of a surgical registrar on the ward. [12]

The evening note was written at 8pm and noted the deceased continued to vomit and was given Maxalon and continued IV fluids. The deceased complained of a sharp pain in the left side of her abdomen on movement and Dr McMillan (surgical registrar) was made aware of her difficulties. Dr McMillan reviewed the deceased after she had been given IV paracetamol. This was stated to have had minimal effect. She had passed flatus during the evening. She had rested in bed most of the shift and was not given Naprosyn because she was not tolerating food and it was required to be taken with food.

In evidence Dr McMillan confirmed from the IPN nursing note he believed he first saw the deceased post-operatively on the evening of Friday 20 March 2009 but could not recall the

[11] Exhibit 9
[12] t 23.8.13 p316

specifics.[13] He checked her vital charts and that her IV fluid was adequate for overnight hydration. Dr McMillan did not record his visit anywhere and as far as he was concerned it was a routine check to ensure there was nothing alarming with respect to the deceased which needed immediate attention. He did not recall the deceased complaining of any pain, but was confident had there been any problems or concerning features he would have escalated her care or sought additional input.

Dr McMillan commented in spite of the deceased having stated she had passed flatus he does not believe he would have reviewed her fluids with a view to decreasing her intake because it was clear she was not tolerating fluids well and she needed supportive therapy by continued IV fluids.

21 MARCH 2009

The nursing note of the overnight shift appears again at 3am on 21 March 2009 (Saturday) and states the deceased appeared to be sleeping for long periods that shift with stable observations and medications given as charted. Other than the severe pain recorded in the early hours of the Friday (20th) for the previous evening, the deceased's medications tended to be of the milder analgesics and not the stronger Tramadol. The deceased had been assisted to the bathroom twice during that shift and her IV fluids

[13] t 23.8.13 p314

continued at the rate of 1 litre every 12 hours. No complaints were recorded for the deceased following the review by Dr McMillan the previous evening.

Mr Hudson again reviewed the deceased on the morning of Saturday 21 March 2009 following the observation by the nurses the deceased had slept well, or for long periods overnight.

Mr Hudson stated his review of the deceased on the Saturday was at approximately 10 am and again he did not write up the ward round in the IPN because he expected it to be written up by the Surgical Registrar, Dr McMillan. There is an extensive note by Dr McMillan in the IPN timed at 10.30am. Mr Hudson agreed his personal notes of the Saturday ward round were consistent with those of Dr McMillan in the IPN for 21 March 2009.

The deceased was afebrile, although her pulse was elevated at 100/per minute. Her abdomen was tender with a small distension and further investigations were ordered, including blood tests and an ECG and anti VTE measures implemented. [14]

At the time of the review there was a record of two vomits with a lack of clarity as to whether they were "coffee

[14] Exhibit Vol 1 Tab 14 IPN 21.3.09

ground" which would indicate internal bleeding from the gastrointestinal (GI) tract. The deceased's oxygen saturation was 95% and she was still notably dehydrated which, in the context of her presentation, Mr Hudson did not feel was of great concern. Mr Hudson asked that the deceased be given Nexium for her epigastric discomfort and heartburn, and Clexane, continuation with her TEDS stockings and for her to be mobilised two to three times a day.

The original plan was for the deceased to remain for three days in hospital, with two days postoperatively. The fact she was not mobilising well indicated to Mr Hudson it was not likely she would be discharged on the Saturday. Mr Hudson stated it was because of her reduced mobility he had decided her risk for VTE had elevated and therefore instituted the Clexane. He did not consider there to be any clinical basis for concern other than her prior risk factors to do with weight and age. The elevated risk at this time was due to her decreased mobility postoperatively.

Mr Hudson also ordered the deceased have a bladder scan and an ECG to investigate her continued low urine output and the small abdominal distension which could have resulted from a full bladder retaining urine, and to ensure her epigastric discomfort and complaint of heartburn did not

have a cardiac cause. The charts indicate she was commenced on Ceftriaxone at 10am.[15]

Due to Mr Hudson's note she was dry he ordered the rate of infusion of her IV fluids to be increased.

The fact Mr Hudson believed she was still dehydrated on the morning of Saturday 21st March, and ordered an increase in her rate of fluid flow, seems to support Dr Greenhill's comment in evidence. He considered the deceased was always clinically dehydrated[16] regardless of the five and a half hours omission in IV fluids during the mid part of the 20th. The fact of the deceased being dehydrated postoperatively was explicable in all the circumstances without necessarily indicating a concern with developing sepsis.

As far as Mr Hudson was concerned, following his review of the deceased on the morning of Saturday 21 March 2009, the deceased was progressing satisfactorily, albeit more slowly than he would have envisaged.

The next recorded event with respect to the deceased followed approximately two hours after review from Mr Hudson when Dr McMillan, who had been present at the review, was asked to review the deceased by the nurses due to a change in her presentation.

[15] t 20.8.13 p139
[16] t 20.8.13 p140

At 12:30pm Dr McMillan described the deceased as "cold and clammy" and noted she had continuous epigastric and subcostal pain with sips of fluid. He noted there was no chest pain nor past history of DVT or cardiac pathology. He noted an elevated pulse at 106 bpm with a blood pressure of 140/83, respirations at 18 per minute and an oxygen saturation of 90% on room air after mobilising. This presentation caused Dr McMillan to query whether or not the deceased might be suffering from a pulmonary embolus.

Dr McMillan considered the deceased's pain to be consistent with the operation she had undergone and thought her ongoing pain might be a small amount of gastric irritation possibly from the continued vomiting. [17]

Dr McMillan confirmed the only clinical sign he believed may have been an indicator of sepsis with the deceased was her elevated pulse, but that in isolation did not indicate any of her later problems. He thought it was more likely she had been mobilising and that her pulse and oxygen saturations were outside normal range as the result of exertion.

Dr McMillan wrote out a plan to increase the deceased's fluid intake by giving her two x250mls fluid bolus IV and to monitor her urine output if the fluid bolus produced a

[17] t 23.8.13 p324

response. There is a request to the nursing staff to notify him if there is a decrease in oxygen saturation or a further increase in her respiratory rate which would be indicative of a pulmonary embolus. He asked to be notified of any further concerns.

The nursing note at 2.15pm continued to describe the deceased as cold and clammy with an elevated pulse of 112 bpm. The deceased had complained of developing pain under her right breast and by noon had complained of pain all across the top of her diaphragm.

The deceased was reviewed again by Dr McMillan at 3pm who noted the blood test results from the 11am collection. Dr McMillan did not believe the results of the blood tests taken at 11am indicated the deceased was suffering from either peritonitis or infection. He considered her white cell count to be within the normal range at 11 o'clock that morning. Her baseline creatinine was elevated to 261 and Dr McMillan considered the diagnosis for the deceased was acute renal failure. He ordered more IV fluids, monitoring of her urine output, and a repeat of her bloods.

The deceased was reviewed at 9pm and the nurses noted she had only voided 50mls in the past two hours despite the increased fluids. The nurses described the deceased as

remaining cold and clammy, short of breath and to be now complaining of feeling very unwell.

Dr Greenhill was asked to review the deceased and he attended her at 9.30pm. He noted she was three days postoperative and, in his view, peripherally cyanosed, cold, clammy and afebrile. Dr Greenhill noted her abdomen was distended and that she had no bowel sounds. Her blood pressure was 120/80 with a pulse of 110 bpm. He noted she had a diagnosis of acute renal failure and wrote "? Septic".[18] He indicated she required close monitoring and an indwelling catheter (IDC). He ordered blood cultures and repeat U & E testing.

The drug chart indicates the antibiotic Ceftriaxone was continued and the deceased's temperature and general observations charts throughout the day of the 21st show a continuous rise in the deceased's pulse rate accompanied by a fall in her temperature. Her blood pressure readings were variable although the systolic rate generally remained above 100. Dr Greenhill asked for the deceased to be transferred to the high dependency unit (HDU).

On Dr Greenhill's review at 11.30pm he noted she was now in rapid atrial fibrillation at 158 bpm with a sinus tachycardia

[18] t 20.8.13 p138

two hours earlier. He treated the arrhythmia with IV Amiodarone which served to drop her pulse rate.

Dr Greenhill's shift ended at midnight on 21 March 2009 and he handed over to the night doctor in the emergency department to ensure he was aware of a problem with the deceased which Dr Greenhill had noted as being possibly septicaemia.

In evidence Dr Greenhill stated "I was obviously concerned but I must admit that in retrospect I didn't recognise she was so unwell as she was - as she obviously became very rapidly after that. I thought I had more time – well, we had more time but I was, I must admit, unaware that she was as badly down the track as she was".[19]

22 MARCH 2009

The emergency night doctor in HDU began monitoring the deceased at approximately twenty past midnight in the early hours of the Sunday morning. His HDU entries for the deceased indicate he considered she had been showing signs of sepsis since the previous evening and had been seen by the surgical team and commenced on Ceftriaxone and fluid resuscitation. He also commented she was cold and clammy and that the Frusemide dose had not produced any effect. Five minutes later he discussed her management with the microbiology consultant at RPH who recommended

[19] t 20.8.13 p139

adding Meropenem and Metronidazole to her antibiotic regime. He reported the CT result at 1.25am and discussed that with Dr McMillan who later had a discussion with Mr Hudson and transfer to RPH was recommended.

There are no entries in the deceased's IPN between 11.30pm on 21 March until that of Dr McMillan at 3.30am on 22 March 2009.

The note appearing in the deceased's IPN for the 22 March 2009 given as 1530 must be incorrect. It would appear to be a note in Dr McMillan's handwriting and Mr Hudson's personal notes record a telephone call from Dr McMillan at 3.17am on Saturday 22 March 2009. He informed Mr Hudson the deceased's condition had deteriorated. This surprised Mr Hudson as he had not considered her to be a problem when he saw her in the morning round on Saturday 21 March 2009 and had not been contacted with any concerns since that time.

Mr Hudson was told the deceased's condition had suddenly deteriorated in the early hours of the Sunday morning and that an abdominal CT scan showed gas in the wall of the small bowel and gas in the portal vein indicating infarction of the small bowel. Mr Hudson directed Dr McMillan there should be urgent arrangements made to transfer the deceased to ICU at Royal Perth Hospital by the Royal Flying

Doctor Service. He did not attend as there was nothing for him to do to assist with that transfer.

Dr Qazi then contacted the relevant organisations to organise the transfer as soon as possible. The deceased's blood analysis showed "severe respiratory acidosis".

The next entry is at 4.15am on 22 March 2009 and indicated the deceased arrested after vomiting large coffee grounds. Her resuscitation included CPR, drugs, defibrillation, intubation and ventilation.

At 4.55am the deceased had a second arrest and repeat blood gases again showed severe acidosis. The deceased received continued aggressive resuscitation, however, remained with fixed and dilated pupils and a very low oxygen saturation.

The ventilator was turned off at 7am and the deceased passed away on 22 March 2009.

POST MORTEM EXAMINATION[20]

A post mortem examination was undertaken by Dr Clive Cooke on 24 March 2009 and found a transverse 13cm abdominal incision with 14 staples in the surgical wound. He found infection in the deceased's abdomen with peritonitis,

[20] Exhibit 1 Vol 1 Tab 13

increased fluid and necrosis and infarction of parts of the intestine, including a specific 15cm length of small intestine. There was softening of parts of the body as seen with septicaemia and her lungs were congested.

Dr Cooke also noted obesity, a nodular thyroid gland, arteriosclerotic hardening of the arteries and scarring of her kidneys. The arteriosclerotic hardening of the deceased's aorta indicated moderate arteriosclerosis along its length while her coronary arteries revealed a moderate degree of fibrous and partly calcified arteriosclerosis, throughout the coronary artery system with areas of narrowing up to 50%. Dr Cooke did not locate any thrombus in the arteries. In addition, there was some enlargement of the deceased's heart reflecting the cardiac disease. Dr Cooke identified arteriosclerotic nephrosclerosis and later microbiological investigations identified *Enterococcus spp* as well as mixed bacteria.

Dr Jamieson explained in evidence *Enterococcus spp* is a bacteria found in the bowel and would indicate the infection supported ischemia of the bowel resulting in sepsis rather than an infection from an external source or arising out of the operation.[21]

[21] t 21.8.13 p238

Dr Cooke concluded the deceased had died as the result of abdominal infection following surgery (repair of recurrent umbilical hernia).

Following receipt of the post mortem examination results a concern was raised as to the aetiology of the abdominal infection leading to the deceased's sepsis and death. An overview of the deceased's management was sought from Mr Philip Childs, consultant surgeon.

REVIEW BY MR CHILDS

The issues Mr Childs was asked to comment upon related to the cause of the deceased's abdominal sepsis following repair of her recurrent umbilical hernia; whether the sepsis was identified early enough, as in were there early warning signs postoperatively which should have alerted the surgical team to the deceased's illness, and whether her management was appropriate once her infection had been suspected.

Mr Childs noted the deceased was an elderly and obese lady with no significant medical history. In life, the deceased appears to have been relatively healthy for her weight and age, and as Dr Beckett commented when confronted with the deceased's cardiovascular post mortem results, the fact she had cardiovascular disease to that extent was not surprising given her age and weight, although she was

showing no clinical symptomology which would support that level of disease.

Mr Childs noted the previous hernia repair in October 2008 and the fact surgery on the afternoon of the 19 March 2009 had lasted an hour 20 minutes. In evidence Mr Childs said this was longer than one may have anticipated but not surprising in view of the fact it was a repeat repair involving mesh which had to be separated from the bowel. He had not noted the episodes of low blood pressure and pulse rate discussed by Dr Beckett in evidence but did not consider there were any significant anaesthetic or surgical complications.

Mr Childs noted the commencement of serious pain for the deceased some hours after she was returned to ward described as 8/10. She was noted to be distressed and sweaty and later vomited. She was reviewed by Dr Nash later who considered she was dehydrated and in Dr Child's words "increased her IV fluids".

Mr Childs was advised during the course of his evidence the deceased's cannula had fallen out and her prescribed fluid therapy ceased for 5½ hours before Dr Nash reinstated her fluids in the afternoon of the 20th. Mr Childs did not believe dehydration for 5½ hours in the overall scheme of management was significant in itself.

Mr Childs noted in his report he considered it to be *"Likely that her severe illness of ischaemia of the small bowel commenced at about the time of the first complaint to the nurse in the early hours of 20th March 2009"*.

The evidence is this was probably later in the evening of the 19th March 2009 but nevertheless corresponds to a period of hours after the operation.

Mr Childs also considered:

> It is likely she developed a thrombosis in one of several branches of the superior mesentery artery causing ischaemia to the subtended area of small bowel. Her clinical and biochemical assessments from that time are in keeping with ischaemic gut with resultant sepsis, acidotic picture, increasing distress, lowering blood pressure, rising heart rate and increasing abdominal distension and pain. These are all in keeping with the diagnosis of ischaemic gut.[22]

In evidence Mr Childs indicated it was his view the deceased's sepsis arose from her ischaemic gut and he was of the view her ischaemic gut resulted from an arterial occlusion of some description in one or several branches of the superior mesenteric artery which then caused ischaemia to the areas of small bowel being served by those arteries.[23] The arterial blood supply to the gut wall was compromised but it was impossible to be adamant about what exactly had occurred.

[22] Exhibit 1, Tab 13
[23] t 19.8.13 p16

Mr Childs reviewed three possibilities which, postoperatively, he considered to be worth considering on the overall evidence.

Directly referrable to the process of the operation rather than the conduct of the operation, Mr Childs stated that small vessel disease due to the trauma of the surgery was a possibility, in that when the gut was handled for the purposes of the hernia repair that could have produced trauma to the blood vessels to the bowel which would not necessarily be visible at the time Mr Hudson examined the gut for perforations. However, as Mr Childs stated the fact there was no frank perforation and the gut appeared to be viable with a good blood supply and healthy at the time Mr Hudson returned it to the abdomen would tend to indicate this was a lesser possibility.[24]

The other two possibilities were that there was a vascular event postoperatively whereby a clot formed in the artery or there was a low blood flow situation which resulted in clotting. The low blood flow possibility could relate to a narrowing of some branches of the artery and maybe her blood pressure being low, the flow rate being low to the point that her blood clotted and she developed ischaemia of the subtended bowel.

[24] t 19.8.13 p16

The other possibility for a low flow situation was that of an embolism whereby a piece of blood clot or a piece of plaque from an ulcerated area in the arterial wall had "flown off" and gone down the artery to the point of obstruction and blocked the artery. Mr Childs added sometimes these can be multiple so there are little bits of clot showering downstream into little blood vessels and produce an effect on the bowel without necessarily making the mesentery itself look ischaemic.[25]

Mr Childs went on to explain that any one of those possibilities would result in a lack of appropriately oxygenated arterial blood to the part of the gut it was serving, and as a result those parts of the small bowel became ischaemic. Once there were patches of ischaemia then the bacteria in the bowel migrated through the wall into the peritoneal cavity and multiplied, releasing an endotoxin which is absorbed by the abdominal lining, the peritoneum. From there the infection gets into the circulation and then has direct affect on various organs. This results in the clinical picture of multi organ failure which occurs with sepsis. This is particularly the case with some of the gram negative organisms from the gut.

[25] t 19.8.13 p16

Mr Childs went to some length to explain how difficult it can be to diagnose sepsis arising from an ischaemic gut. In a situation where the post mortem results indicated a relatively small stretch of the gut was affected, but with patchy areas of ischaemic and healthy gut surrounding that area, it was consistent with a showering of emboli in the arterial system causing patchy ischaemia.

Mr Childs was clear the only management for an ischaemic gut is operative. The ischaemic parts of the bowel need to be removed and the healthy parts reconnected. This would be impossible in the circumstances of GRH where there is no ICU facility.

Mr Childs agreed the deceased's general preoperative presentation did not indicate she was likely to have a problem with her arterial system, her clinical signs were all within the normal range, and the risk factors inherent in her age and weight made the choice to carry out surgery at GRH sensible. They did not necessarily pre-empt surgery in the metropolitan region rather than GRH. Mr Childs noted it was not usual to preoperatively prepare for an arterial event in the circumstances of the deceased. He did not believe there was anything that one could really do other than thrombo-prophylaxis once the vascular event had occurred.

> If the artery had blocked, if she had had Heparin beforehand that would reduce the extent of clotting downstream of that event but whether it would make any difference or not, probably not. So this lady didn't get thromboembolic disease: she got

> arterial ischaemia of the small bowel. The role of Heparin in that is really to manage it post event, not as a preventative of it.
>
> So using Heparin before her operation may not have made a difference for her?---Correct: it may not have made any difference to her small bowel ischaemic event. That's open to debate but there's really very little evidence for that.
>
> And what about her management after her operation: would Heparin have made a difference for her?---Only in helping to prevent thromboembolic disease, in other words clots and pulmonary emboli, not to manager her ischaemia.[26]

Further Mr Childs indicated the event to which he was referring as the initiating vascular event may have been totally unrelated to the hernia, but be an isolated vascular occurrence to do with the fact the deceased had had an operation, her blood pressure had fluctuated and an event had happened. The event could be totally unrelated to the surgery and arisen because the deceased had had a shower of small arterial plaques or emboli into the small vessels all over the small bowel and produced the diffuse pattern of ischaemia. That ischaemia of the gut then caused her sepsis.[27]

The outcome from the initiating vascular event for the ischaemia not being detectable, meant the diagnosis of the resulting sepsis would have been particularly difficult.

In his original report Mr Childs had commented the deceased, on her risk factors, probably should have

[26] t 19.8.13 p18
[27] t 19.8.13 p26

received thrombo-embolic prophylaxis in the form of Heparin, as well as either flowtrons or TEDS stockings, but agreed in evidence the effectiveness of thrombo-embolic prophylaxis for arterial blockages is only debatable.

The other comment Mr Childs made in his report was of the need to treat the sepsis aggressively, at an earlier time once her postoperative progress was not normal. He agreed he doubted that would have made any difference in this case as cases of ischaemic gut resulting in sepsis are frequently a fatal condition even when the diagnosis is made early and appropriate operative intervention possible. He was clear there would have been little to prevent the deceased's death once the small bowel ischaemia had commenced. He doubted earlier transfer to a major facility would have changed her ultimate outcome. He did not believe it likely that an earlier laparotomy or diagnosis of the condition would have been beneficial.

Overall the issue of what caused the arterial blockage to result in the ischaemic event cannot be determined with any certainty. Once the ischemic event had occurred it is likely it would have progressed to the state where it was irretrievable without diagnosis in most situations. Mr Childs believed operative intervention on the afternoon of Saturday 21 March may have provided a diagnosis for the deceased

but there is no certainty the prognosis would have been any different.

In evidence Dr Jamieson commented on the use of ephedrine during the course of the deceased's operative procedure. Although unremarkable, its use may have contributed to a spasm of the deceased's vessels which may have predisposed an event by the occlusion of the arterial system by a thrombus or plaque embolus.

Ephedrine is an alpha-stimulant and works to stimulate the smooth muscle to contract. It's a derivative of adrenaline and an adrenaline surge can cause the heart to beat faster to increase blood flow to the essential organs.[28]

It was possible a vasospasm caused by the ephedrine could have dislodged a piece of plaque or a clot which had been formed from low blood flow.

Additionally many patients are dehydrated prior to operative procedures due to the fast requirements. The deceased's dehydration prior to the procedure may have aggravated the effect of the ephedrine in terms of her venous response.

There was also the issue of a five and a half hour interruption of the deceased's post operative hydration although this

[28] t 21.8.13 p242

was after her severe pain in the hours post operatively. Dr Jamieson commented:

> "It's apparent from the various attempts to untangle the fluid balance chart that she was – there was a period where she was behind the curve in terms of her hydration. People, various clinicians have commented that they felt that she was dry. If one adds to that the fact that she was vomiting, I think there's no doubt that she wasn't as well hydrated as she could have been, and as Mr Hudson said you know that's – I believe certainly a contributing factor in terms of increased viscosity. And we don't really have any evidence for lowered flow in her blood pressures but we have evidence for low blood flow in the fact that she was cold and clammy on a number of occasions and that is the thread that comes through, and cold and clammy is a better indication of poor flow than blood pressure, because a lot of people maintain their blood pressure until the body just can't do it by shifting fluids around, and then it drops dramatically because the body is no longer able to compensate."[29]

Mr Hudson pointed out that if the blood is more viscous and there is a narrow superior mesentery artery, it is possible that could add to the reduction in blood flow through the superior mesenteric artery. [30]

The result of all those observations is a possible mechanism whereby there was an ischaemic event. The deceased had a not insignificant amount of atherosclerosis commensurate with her age, was quite calm in her everyday movements, experienced a period of lowered blood flow during the operative procedure, with the use of ephedrine, which may have irritated a dislodgment of plaque and post operatively her blood becomes more viscous due to ongoing

[29] t 21.8.13 p244
[30] t 20.8.13 p193

dehydration and so there is an increased build up of platelets on plaque.

The improved hydration and reduced viscosity of the deceased's blood allowed the plaque and/or platelets to break off and then shower through the deceased's arterial system and initiate ischaemia of the gut which then led to the septicaemia. That chain of events becoming critical around midday on Saturday 21 March 2009 which was the beginning of the signs for a clinical change in the deceased's presentation which could not be explained by post operative recovery alone.

If the operative procedure was related to the arterial blockage in any way, it would seem that all those factors may have played a part in the development of the deceased's ischaemic gut. It is a rare event and extremely difficult to diagnose with enough time to intervene operatively for a successful prognosis. Certainly not in the situation available in GRH at that time.

The evidence is also that this type of arterial event leading to ischaemic gut may have been completely unrelated to the fact of the operation. Or, it was related merely because the circumstances of an operation, any operation, are such there is always the possibility of an unexpected event which

can lead to death. It is for that reason patients are required to fill out consent forms prior to surgery.

CONCLUSION AS TO THE DEATH OF THE DECEASED

I am satisfied the deceased was a 75 year old woman with a BMI consistent with a diagnosis of obesity. She was fit and apparently healthy for the lifestyle she led and there were no indications prior to post mortem examination the deceased had significant arterial atherosclerosis. There was no clinical symptomology during life which would have alerted clinicians to the fact the deceased may have fallen into the category of an arteriopath.

The deceased had a recurrent umbilical hernia which in October 2008 had been repaired using a Ventralex mesh patch. In February 2009 the deceased complained to Mr Hudson of a recurrence of her hernia and on review he found the Ventralex mesh patch had migrated from the surface of the abdominal wall to a position underneath the deceased's skin. Due to the fact recurrent hernias become progressively more difficult to repair, Mr Hudson suggested the deceased have the operation at GRH which had better facilities for the purposes of the operation.

In evidence Dr Jamieson, Director Clinical Governance for WACHS GRH, stated there were risks in leaving an umbilical hernia untreated, in that it may cause strangulation of parts

of the bowel or trapped intestine and therefore increase a likelihood of ischaemic or necrotic bowel. That condition leads to all the difficulties that were later seen with the deceased as a result of her ischaemic gut.[31]

Mr Hudson, supported by Dr Beckett, did not consider the deceased to be in a high risk category for VTE by way of her age and weight because she appeared to be clinically quite healthy. Both practitioners believed GRH to be an appropriate centre for her operative procedure and post-operative care, and to some extent this was supported by Mr Childs on the pre-operative clinical picture.

Mr Childs was also of the view the length of a repeat procedure, which would necessarily involve the separation of adhesions or tissues from the mesh, indicated it would be a long procedure and for someone in the deceased's age and weight category should have been treated with prophylactic thrombolytic medication beforehand. The pre-operative use of Heparin affects the clotting cascade and is used to correct venous system clotting. There are always tensions with using anti-clotting agents in surgical procedures.

Aspirin is the thromboembolitic medication used where there are concerns somebody has arterial system clotting

[31] t 21.8.13 p286

problems. Aspirin reduces the "stickiness" of platelets and is rarely used in situations where there is no evidence of clot formation. It is not used preventatively pre or post operatively without cause.[32]

The procedure did last for a long time and it was necessary for the anaesthetist to provide the deceased with two separate amounts of ephedrine due to her blood pressure dropping and pulse rate decreasing. This would imply there were periods during the operation when there was decreased blood flow. The deceased's post operative blood pressure results do not support continued reduced blood flow.

On the evidence it is likely the initial ischaemic event for the deceased occurred at the time she recorded severe pain some hours after her operation. The deceased responded to analgesia, although she remained with a low level of pain which appears to have been appropriately controlled without the use of strong analgesics. She vomited from time to time. There is evidence she was clinically dehydrated but this was addressed.

Following the undetected ischaemic event the deceased's recovery from the operative procedure did not progress as expected although there were no obvious clinical signs of an

[32] t 19.8.13 p15

infective process until approximately midday on the Saturday. All the indications are that although positively identified later that day, it was already too late to operate successfully on the deceased's condition.

Arrangements were made to transfer her to Royal Perth Hospital with appropriate antibiotic cover however, the deceased arrested before that could be finalised and died in the early hours of the 22 March 2009.

I find death arose by way of Natural Causes.

COMMENTS ON THE TREATMENT OF THE DECEASED

Ultimately it became apparent ischaemic gut is very difficult to diagnose with enough time to prevent death, it is often only diagnosed post mortem. Mr Childs said to successfully diagnose an ischaemic gut you need lots of experience and to have almost developed a "nose" for it, before it becomes symptomatic, to successfully operate. Where it is the result of an unknown arterial event it is not possible to treat it pre-operatively, as may be the case with a venous thromboembolus.

While it is likely the fact of the operation in the deceased's case triggered the event, it is not possible to say anything about the operation itself caused it. I note in this case the majority of the clinicians believe the severe pain

experienced by the deceased some hours after the operation later on 19 March 2009 was the initiating event. While the deceased may have been dehydrated due to pre-operative fasting on the 19th, that was before the events of the 20th of March where it is known the deceased was without extra hydration for a period of 5 and a half hours. This makes that occurrence (5½ hrs) irrelevant to the cause of the initiating arterial event. It may have been as simple as the need for ephedrine, unremarkable, on a pre-operatively fasted patient in the deceased's unknown cardiovascular circumstances.

The fact the deceased did not thereafter require strong analgesia, and maintained a relatively stable blood pressure, masked the progression of the ischaemia and resulting sepsis. By the time the deceased became apparently symptomatic and efforts were undertaken for her transfer it was, tragically, too late to save her life.

Inevitably close examination of events post-operatively in many hospital settings, especially out of the metropolitan area, reveal unwanted occurrences in a patient's care. This was true in the deceased's case but there is no evidence any of the noted deficiencies caused or even contributed to her death.

Evidence was heard from Drs Hudson, Beckett, Jamieson and Acting Regional Director of Nursing and Midwifery Services in the Midwest, Patricia Baldwin, that many of the problems noted in the deceased's post-operative care either have been, or are in the process of being rectified by additional resourcing in staffing, technology and funding.

Since 2011GRH has had an increase in its medical staff which ensures better medical coverage of rosters on the wards.[33] I note here Mr Hudson is adamant he would have been accompanied by a surgical registrar on his ward round on 20 March 2009, regardless of surgical ward coverage later in the day.

There is now more coverage of rosters by IV competent nursing staff who would have been in a position to recannulate the deceased in the absence of medical staff, and higher staffing levels should ensure improved documentation. In the deceased's case there was not always consistent documentation, and in some instances the need to track what there was through a combination of the charts and IPN made it difficult to form a comprehensive chronology.

There has also been some improvement in nursing co-ordinator levels, however, not to the extent necessary to

[33] t 23.8.13 p359

allow co-ordinators to adequately do real time audits of nursing care as opposed to assisting staff with patient care. Real time audits are a much more effective way of ensuring compliance with nursing plans, protocols and consultant standing orders.[34]

This has suggested reasonable recommendations can be made in this case which would have improved the management of the deceased post-operatively, while recognising these may not, ultimately, have altered the outcome.

Recommendation No.1

All nursing staff employed in GRH be reminded about the importance and requirement for effective documentation.

Recommendation No.2

GRH consider increasing clinical supervision to allow more real time audits of documentation, including provision of more clinical nurse co-ordinators.

E F VICKER
ACTING STATE CORONER
November 2013

[34] t 21.8.13 p265-283