Coroners Act, 1996

[Section 26(1)]

Western Australia

RECORD OF INVESTIGATION OF DEATH

Ref No: 08/14

I, Evelyn Felicia VICKER, Deputy State Coroner, having investigated the death of ***Brian John DAVIES*** *with an inquest held at the* ***Geraldton Coroner's Court, Geraldton Court House, Geraldton*** *on* ***7 March 2014*** *find the identity of the deceased was* ***Brian John DAVIES*** *and that death occurred on* ***14 May 2011*** *at* ***Sir Charles Gairdner Hospital*** *as a result of* ***Multiple Organ Failure and Intra-Abdominal Haemorrhage following Cholecystectomy*** *in the following circumstances:*

Counsel Appearing:

Ms K Ellson assisted the Deputy State Coroner
Ms R Young (instructed by State Solicitors Office) and with her Ms A Salapak appeared on behalf of WA Country Health Service (WACHS)

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INTRODUCTION

On 12 May 2011 Brian John Davies (the deceased) underwent a cholecystectomy at Geraldton Regional Hospital (GRH). Following the procedure the deceased's condition steadily deteriorated and he was transferred to the Intensive Care Unit (ICU) at Sir Charles Gairdner Hospital (SCGH) on 13 May 2011. He did not recover and died on the morning of 14 May 2011.

He was 57 years of age.

BACKGROUND

The deceased was born in Perth on 17 October 1953. He worked in various occupations and a number of locations throughout Western Australia and his wife and family travelled with him.

In 2005 the deceased and his wife retired to Kalbarri following the deceased suffering a number of medical problems.

MEDICAL HISTORY

The deceased's medical history was extensive and included hypertension, type 2 diabetes mellitus, cerebrovascular disease and a stroke eleven years prior to his death, hypercholesterolaemia, gastroesophageal reflux disease

(GORD), obstructive sleep apnea and chronic obstructive airways disease (COAD) and depression. The deceased was medicated for his illnesses.

On 14 March 2011 the deceased presented to GRH by ambulance with right upper quadrant abdominal pain. He was examined and noted to be tender in the upper right abdominal quadrant. Blood tests were ordered. These showed an elevated inflammatory marker (CRP) and white cell count (WCC). An abdominal ultrasound showed thickening of the gallbladder wall and the presence of a gallstone with biliary "sludge". He was diagnosed with cholecystitis and admitted to GRH under the care of Mr Martin Hudson, Consultant Surgeon. He was treated with clear oral fluids, antibiotics and, Clexane and TED stockings to prevent DVT formation in view of his history.

The deceased remained unwell for some days despite medication and he developed urinary retention requiring an indwelling catheter. By 19 March 2011 he had improved and his chest and abdominal x-rays were normal and the blood cultures grew no organisms.

The deceased was discharged on 22 March 2011, back to Kalbarri, with a plan to have his gallbladder removed once he had recovered from his episode of cholecystitis. The intention was to remove his gallbladder before there was another period of serious inflammation. It is generally

preferable to allow a patient to recover from cholecystitis before removing the gallbladder, but before it becomes infected again.

The deceased signed a consent form for a laparoscopic cholecystectomy when he was reviewed by Mr Hudson on 26 March 2011. The deceased was provided with information about the proposed operation. Mr Hudson advised the deceased there was a risk the procedure would have to be converted to an open procedure because he was a very high risk patient due to his extensive comorbidities and the fact he was categorised as obese. Obesity affects the ability of a surgeon to both visualise and deal with the body tissues. Mrs Davies in her statement to the court confirmed the deceased understood he was in a high risk category and the operation would be risky.[1] Mrs Davies does not recall appreciating there was a risk her husband could die.

12 MAY 2011

The deceased was admitted to GRH for his elective laparoscopic cholecystectomy on 12 May 2011. On admission and preoperatively the deceased's oxygen saturations were 98% and his other observations in the normal range.

Mr Hudson performed the operation assisted by surgical

[1] Ex 2, Tab 4

registrar, Dr Sundenarajan, and Dr Murray James-Wallace, as anaesthetist. The operation record indicates there was difficulty with visualisation of Calot's triangle (area bounded by the cystic artery, cystic duct and hepatic duct) due to the presence of dense adhesions and the fact of the deceased's obesity. Mr Hudson changed to an open procedure making quite an extensive opening in the deceased's upper right abdominal quadrant. Mr Hudson still had difficulty identifying Calot's triangle and decided it was safer to leave part of the gallbladder in situ while removing the rest.

Mr Hudson does not recall any other difficulties and there was no mention of haemorrhage despite the need for conversion to an open procedure. Mr Hudson recorded his view the operation had been relatively uncomplicated.

The deceased was treated with antibiotics and, from an anaesthetic perspective, the procedure was uneventful with the deceased emerging from anaesthesia after having had his residual muscle relaxants reversed. He was able to communicate and speak. Due to the need to convert the operation from the laparoscopic procedure to an open procedure the deceased was provided with a morphine administration device (PCA) in anticipation of expected increased post-surgical pain and he was directed in its use once he was in recovery.

In recovery, the deceased became agitated and pulled the

nasogastric tube from his nose and dislodged his IV access. It took the anaesthetist, with the assistance of the recovery room nurses, some time to reinsert another IV cannula.

It was directed the deceased's blood sugar be tested four hourly, that his CPAP oxygen equipment be available and he was not to recommence his diabetic medication until he was eating proper meals. The anaesthetist was not called again to the recovery room for the deceased.

The deceased was kept on the day surgery ward.

The deceased complained of pain and the surgical registrar reviewed him. There was some minor oozing from the incision wound and the surgical registrar injected local anesthetic with adrenalin around the incision to stop any further ooz and reduce the pain. Dr Sundenarajan reported minimal drainage from the intra-abdominal suction drain and Mr Hudson was satisfied with the treatment. He was not contacted again that day with any concerns about the deceased.

When treating the deceased's wound at approximately 6:30pm Dr Sundenarajan noted the deceased's Clexane was to be withheld until the following day.

The deceased's oxygen saturations dropped to between 89-91% on 6L of oxygen via a Hudson mask in contrast to his

preoperative good oxygen saturations.

The deceased continued to experience pain and as a result the anaesthetist, Dr James-Wallace, was contacted by the nurses on the day surgery ward at 8pm. The nurses asked if the deceased could have anything stronger than morphine. This caused Dr James-Wallace some concern. Dr James-Wallace was told the deceased was drowsy and in pain and his oxygen saturations were low. He was advised the deceased had been seen by the surgical registrar who had reviewed him and injected his wounds with anesthetic, but the deceased was still complaining of pain. Dr James-Wallace asked the staff to ensure the deceased was using his CPAP machine and to use his PAC. He did not prescribe any further pain control. He asked to be re-contacted should the deceased continue to experience problems.

Dr James-Wallace followed that phone call with a phone call to the night nurse manager. He expressed his concern the deceased was in the day surgery ward. Dr James-Wallace asked the night nurse manager if the deceased could be transferred to the high dependency unit (HDU) and, if that was impossible due to it being over capacity, he wished the deceased to be "specialled" with one on one nursing care. He was assured the deceased would be managed.[2]

The deceased's observations at 8pm record a Glasgow coma

[2] Ex 1, Tab 6

score (GCS) of 15/15 and also indicate Clexane had been administered despite the recent with holding order by the surgical registrar.

It would seem Dr James-Wallace's request was complied with. The next doctor to see the deceased was Dr Carter, the Medical Registrar, who recalled he was working as the Medical Resident Officer at GRH in the emergency department (ED) on the evening 12 May 2011 and was called to HDU at approximately 8:45pm to review the deceased.[3]

Dr Carter arrived at HDU shortly after 9pm and found the deceased's oxygen saturations were dropping. He required 15L of oxygen via a non-rebreather mask to keep his saturations at 92%. He was also tachycardic with a heart rate of 123bpm. Dr Carter noted the deceased had a history of obstructive sleep apnoea, was obese and usually used a CPAP machine to help with his sleep apnoea. The deceased's blood sugar level was raised and he had an elevated blood pressure at 164/104. This was higher than it had been at admission. His respiratory rate was normal at 18 bpm. Dr Carter confirmed his GCS as 15/15.

The deceased denied any central chest pain but reported pain in his abdomen. He had no cough and he was afebrile. Dr Carter noted a soft tender abdomen, with no elevation of

[3] Ex 2, Tab 7

his jugular-venous pressure. The deceased had decreased air entry but Dr Carter noted no crackles or wheezes. The deceased was in no obvious respiratory distress despite his oxygen saturations and his respiratory rate was normal. Use of the CPAP caused the deceased's oxygen saturation to drop to 88% from 92% on the non-rebreather mask with 15L oxygen.

Dr Carter concluded the deceased's decreased oxygen saturation was likely post-operative atelectasis which is common in surgical patients. He considered the deceased's elevated pulse and blood pressure were due to pain. Dr Carter formulated a plan for the deceased to have a chest x-ray and also an electrocardiogram (ECG). He was concerned the deceased may have a collapsed lung or fluid in his lungs.

The chest x-ray showed a faint air bronchogram on the left consistent with a likely left lower lobe collapse or consolidation, otherwise the lung fields were unremarkable. The ECG was taken to exclude any cardiac cause and it demonstrated a sinus tachycardia with no changes and did not demonstrate any changes typical of an infarction.

Dr Carter noted the deceased's pre-admission oxygen saturation as 95% indicating some degree of lung insufficiency. He asked the nursing staff to administer oxygen to ensure the deceased's oxygen saturations

remained above 90% to prevent hypoxia. He requested the deceased be trialed on CPAP again and that the oxygen levels be adjusted according to the saturations.

Dr Carter instructed the nursing staff to seek further review from ED doctors if they were concerned about the deceased. Dr Carter was not called to the deceased again.

Despite Dr Carter's plan, by 10:45pm the deceased remained unwell with oxygen saturations down to 84%, despite 15L of oxygen via his non re-breather mask. ED was contacted again and Dr Suresh attended. Dr Suresh noted the presence of crepitations at both lung bases and requested an arterial blood gas analysis. This was done at 11:27pm.

The results of the arterial blood gas analysis show marked acidosis (pH 7.16), hypoxemia (p0266) and hypercapnia (pC0263). The deceased's urine output was poor and his potassium level elevated (6.3mmol/L). These results indicate renal failure. The diuretic frusemide was administered and the deceased discussed with the surgical registrar, Dr Sundenarajan.

In evidence Dr Jamieson, Regional Medical Director for the Midwest region of the WA Country Health Service (WACHS), stated that in hindsight it may be thought frusemide was a poor choice however, in his view there was an explanation

for that management.[4] Dr Jamieson indicated that sometimes the beginnings of kidney failure can be reversed by the use of frusemide. The deceased was being adequately hydrated, urine wasn't being produced, and the deceased's blood pressure was high suggesting there may have been significant fluid retention. This could be as a result of the kidneys closing down and the "jolt" with the diuretic may cause them to start operating again. In the case of the deceased this did not occur however, Dr Jamieson did not think the use of frusemide in this particular situation was unreasonable.

The arterial blood gas results indicate the deceased's system was showing early signs of renal failure on the evening of 12 May 2011 and there were indicators the deceased was experiencing the beginnings of organ failure.

Dr Sundenarajan ordered CPAP, blood tests and nebulised salbutamol. It was noted the deceased was having trouble with the CPAP mask but the doctors wished him to be continued with its use if possible.

13 MAY 2011

The next notes in the integrated progress notes (IPN) indicate that at half past midnight the deceased again became increasingly agitated by use of the CPAP mask

[4] t 7.3.14 p42

which he removed. He was given the non-reversible breathing mask which appeared to settle him.

The deceased's observations show a gradually rising tachycardia to 138bpm at 4am, whilst his blood pressure remained elevated with readings as high as 183/112 at 2:20am. The deceased's temperature remained normal and his oxygen saturations remained low at 89-92% considering he was on high delivery oxygen.

HDU nurses again contacted ED doctors for medical input on receipt of the earlier ordered blood results, continued poor urine output and the deceased's deteriorating observations. The deceased was not physically reviewed but telephone prescription given for antibiotics and another dose of frusemide over the telephone with the instruction the deceased was to be reviewed by his usual doctor in the morning.

The deceased's blood pressure reduced to 124/84 at 5am and 113/76 at 6am. At 6:30am his urine output was still low with only 100mls over the prior seven hours, and medical review was awaited.

Dr Sundenarajan asked Dr Said, Head of Department and Consultant Physician to review the deceased as soon as he was able. Dr Said was the only Consultant General

Physician at GRH. He was not available out of hours.[5] Dr Said's review of the deceased's condition, as reflected in the integrated progress notes, was extensive and he took control of the deceased's management from 7:30am onwards.[6] He reviewed the deceased's history to date and that he had been reviewed for hypoxia, tachycardia and oxygen therapy. He noted the deceased's ongoing poor urine output, despite adequate IV fluids and the use of a diuretic, his worsening oxygen saturations and significantly deteriorating blood gas analysis.

In his statement Dr Said[7] pointed out he was concerned the deceased was suffering acute renal failure and made attempts to have him transferred as soon as possible to a tertiary facility in Perth. He discussed the deceased's poor condition with SCGH and with the Royal Flying Doctor Service (RFDS). Dr Said requested the help of Dr Beckett to help prepare the deceased for urgent transfer to Perth for intensive management. The deceased was described as anuric, acidotic and hypoxic.

There were difficulties successfully intubating and ventilating the deceased. A central line, arterial line and a nasogastric tube were inserted and additional antibiotics administered. These are management strategies GRH is not equipped to deal with on an ongoing basis and had to be

[5] t 7.3.14 p50
[6] Ex 1, Tab 13, Tab C
[7] Ex 2, Tab 6

performed in ED resuscitation unit.

The deceased still had a normal haemoglobin and platelet count, though his total WCC was elevated. This suggested a systemic inflammatory response rather than an intra-abdominal bleed resulting from surgery. The deceased required intensive care unit (ICU) resuscitation and monitoring which GRH provided by using Dr Said and Dr Beckett in the resuscitation bay pending transfer to facilities where these were available.

It was only at this stage Mr Hudson, as the original surgeon, was contacted by Dr Said to discuss anticoagulation management and whether Mr Hudson had any other views as to the deceased's management. Mr Hudson confirmed Dr Said's view as to the necessary management for the deceased, and that he should be transferred to a tertiary care hospital as soon as it could be facilitated.

RFDS arrived at 3pm on 13 May 2011 and the deceased was handed into their care. In his referral letter Dr Said advised SCGH of his diagnosis the deceased was suffering acute renal failure complicated by metabolic acidosis and a state of volume overload. He gave a differential diagnosis of pulmonary embolism.[8]

In evidence Dr Jamieson explained acute renal failure is a

[8] Ex 2, Tab 6

condition which can become severe over a very short period of time but can be quite variable. It usually occurs over a period of about 8-48 hours and is associated with an *"inability of the body to push blood through the kidneys, which then triggers a cycle of damage to the kidneys themselves through lack of oxygen that makes the kidneys work, and enables them to filter the blood, so you end up with a spiral, a downward spiral, where there's less blood going through the kidneys, the kidneys are working more poorly, and therefore they tend to constrict, and you get less blood going through the kidneys again. The blood vessels tend to constrict. So, as a result, once you start with acute kidney failure, can get quite serious quite quickly."*[9]

Dr Jamieson could see no evidence the deceased suffered septic shock and pointed out the deceased had always been adequately covered with antibiotics. He believed the elevated neutrophils were associated with a reaction to surgery and a systemic inflammatory response. The evidence as a whole supports the deceased's system being physiologically shocked with a resulting multiorgan failure, ultimately leading to coagulopathy and the intra-abdominal haemorrhage seen at post mortem examination. In Dr Jamieson's view the intra-abdominal haemorrhage related to multi organ failure and coagulopathy shortly before death and this was supported by Dr Cooke.[10]

[9] t 7.3.14 p15
[10] t 7.3.14 p36. Email communication between the Deputy State Coroner and Dr Clive Cooke, Forensic Pathologist, Monday 17 March 2014

TRANSFER TO SIR CHARLES GAIRDNER HOSPITAL

The deceased was admitted to SCGH at 6:30pm 13 May 2011 and transferred immediately to ICU. He required high doses of inotropes to maintain a blood pressure and he had fixed and dilated pupils. He was peripherally shut down and cold. 500mls of old blood had come from his abdominal drain and his haemoglobin levels had now fallen to 109g/L. His acidosis was worsening (pH6.84) and his creatinine had risen to 476. His potassium level remained high and the ICU consultant assessed him as having type 1 and 2 respiratory failure, acute renal failure, massive pressor requirements and likely upper GI ulceration. He was described as critically ill and unlikely to survive the night.[11]

The deceased was treated aggressively in an attempt to correct his renal failure. His coagulopathy was treated by the use of fresh frozen plasma (FFP) but blood continued to drain from the nasogastric tube and the abdominal drain. The clinicians found it difficult to achieve haemostasis.

The deceased's wife had travelled from Geraldton to Perth to be with her husband. He did not recover and passed away at 9:15am on the morning of 14 May 2011.

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[11] Ex 1, Tab 14

POST MORTEM EXAMINATION[12]

The post mortem examination was carried out by the Chief Forensic Pathologist, Dr Clive Cooke at Pathwest.

Dr Cooke located 1600mls of blood clot and blood stained fluid in the deceased's abdominal cavity but no definite bleeding site. He found the heart muscle was pale, and there was coronary and aortic arteriosclerosis with swelling and discoloration of some body organs consistent with the clinical history of multi organ failure.

Microscopy showed changes consistent with multiple organ failure, including fibrous scarring of the heart muscle (ischaemic heart disease) and changes of more recent ischaemia. There was partly calcified arteriosclerosis with narrowing of the left arterior descending coronary artery of up to 60%.

Microbiology indicated no specific infection and neuropathology no significant brain abnormalities.[13]

Toxicology revealed the presence of prescribed medications including those consistent with sedation.

[12] Ex 1, Tab 7
[13] Ex 1, Tab 8

Dr Cooke gave the cause of death as Multiple Organ Failure and Intra-Abdominal Haemorrhage following Cholecystectomy in a man with Ischemic Heart Disease.

In communication with the Deputy State Coroner, Dr Cooke indicated it was his view the intra-abdominal haemorrhage resulted from coagulopathy following multiple organ failure. He believed that was also consistent with the clinical picture, in that the early test results did not indicate a falling haemoglobin until late in the deceased's deterioration.

EXPERT REVIEW

Professor Jeffrey Hamdorf from the School of Surgery, University of Western Australia, was asked to review the management of the deceased. Professor Hamdorf was not concerned with the surgery in the case of the deceased, other than a concern the deceased was always a high risk patient, despite his relatively low anesthetic score, and as such recovery was always likely to be an issue. It may have indicated the deceased should have been placed in HDU from the recovery room initially, or even warranted transferred to a tertiary hospital with an ICU facility for the surgery itself.

Professor Hamdorf believed the deceased's post-operative management would always have been a challenge. He was

in the morbidly obese category with a history of type 2 diabetes, hypertension, cerebrovascular disease hypercholesterolaemia, gastro-oesophaged reflux disease and obstructive sleep apnea which together created a level of complexity whose post-operative management should have been managed in a tertiary centre.[14]

Dr Jamieson acknowledged these difficulties, however, stated it is impossible to predict with certainty which patients, who appear to be managing moderately well pre-operatively, will be those to develop serious complications as the result of the physiological stress of surgery itself. While theoretically one could say a patient fell into the high risk category, realistically, if they appear to be managing well pre-operatively, it would be impractical to send all patients with the deceased's comorbidities to tertiary hospitals in Perth. The health system would simply not be able to cope.[15]

Certainly, when clinicians and surgeons were certain of a patient's post-operative difficulties they endeavoured to send them to Perth for surgery. The situation for the deceased was that post his cholecystitis in March he appeared relatively stable by the time of his elective surgery in May. Had there been a requirement to send him to Perth it is likely he would have waited a much longer time for his surgery. He could well have had further periods of

[14] Ex 1, Tab 5
[15] t 7.3.14 p38

inflammation, serious unwellness and even death while awaiting appropriate and necessary surgery.

It was preferable, where possible, to perform relatively simple operations for patients in remote areas as close to their homes as was realistically possible. The deceased lived in Kalbarri and as such GRH was the most appropriate hospital to deal with simple surgery. It was impossible to predict with any certainty the deceased would have been one of the patients who would deteriorate so rapidly post-operatively.[16]

Professor Hamdorf believed the deceased should have been managed in the HDU immediately on transfer from recovery due to his high risk status. The fact he never recovered post-operatively can be seen from his observations and should have been managed earlier. Professor Hamdorf was concerned Clexane had been administered despite a doctor's instruction it should be withheld, but he considered its contribution to the overall decline would have been minimal.

Professor Hamdorf's view the deceased's care should have been escalated much earlier was understandable from the perspective of the availability of tertiary type care in the metro area. The fact of the deceased's deteriorating status had been noted in the evening of 12 May 2011 and steps were taken to manage him. He had been transferred to the

[16] t 7.3.14 p46

HDU earlier in the evening and ED doctors had been called to assist him while the ED still had adequate medical coverage.

While GRH had improved medical coverage in 2011, over that available in 2009, there was still a problem with medical staff availability during the night shift. The deceased's observations had been taken and his deterioration was noted. In hindsight the use of the furosemide, twice, may not have been appropriate, but there was the possibility it could have assisted with his developing renal failure. While Professor Hamdorf seems to be of the view there was a scenario of sepsis, Dr Jamieson believes it was a situation where the deceased's comorbidities prevented his recovery from the physiological shock of surgery.[17]

While one could predict the deceased would be susceptible to renal failure, the extent could not be determined prior to surgery. The fact renal failure was a problem was evident quite early, from the first blood gases results,[18] and the deceased was managed, but the facilities and resources at GRH were such in 2011 that the extent of his deterioration could not be supported. Arrangements were made for his transfer as soon as was realistically possible on 13 May 2011.

[17] t 7.3.14, p33, 40-41
[18] t 7.3.14, p22,32/33,41/42

CHANGES AT GERALDTON REGIONAL HOSPITAL

The inquest heard evidence from Dr Jamieson, Regional Medical Director for the Mid West Region WACHS, and RN Norris, Acting Regional Director of Nursing and Midwifery for the WACHS, both of whom outlined changes instigated at GRH since the death of the deceased.

RN Norris particularly covered the effects of the introduction into WACHS of the "rainbow" or observation record chart (ORC). There are now a number of different types of ORC in use for different situations. In 2010 the original chart was piloted in WACHS to determine its effectiveness. It was not introduced as a permanent tool to WACHS until September 2011, some months after the death of the deceased.

Ms Ellson, Counsel Assisting plotted the deceased's observations on a chart from her review of the deceased's medical file.[19] The advantage of the ORC, as opposed to the previous record keeping, is it forces contemporaneous recording of a patient's observations in one place. It is very easy to assess trends in a patient's observations visually when the chart is accurately completed. In addition, due to the ORC colours requiring specific management at specific points there is some certainty for nurses when deciding on the level of input required for a patient's ongoing care. It also provides nurses with some mandatory management

[19] Ex 4

interventions, which cannot be dismissed by other practitioners as an overreaction to a particular observation.

The deceased's deterioration post operatively is easy to follow on the completed chart.[20] However, the documentation overall in the deceased's medical file is fairly comprehensive, and there is no doubt his observations were taken regularly, and his management generally followed the options available to the nurses at that time. A factor affecting the deceased's deterioration was the lack of comprehensive medical coverage available in GRH, out of hours in 2011 at the time of the deceased's death.

Between midnight and 7:30am it is clear the deceased deteriorated rapidly. With more medical coverage there may have been earlier blood gas analysis review and earlier instigation of tertiary resuscitation pending transfer to a facility which could manage acute renal failure[21] if that could be arranged. The deceased was admitted to SCGH at 6:15pm on 13 May 2011 following the institution of transfer procedures early that morning. Earlier instigation of those procedures is unlikely to have achieved an improvement of more than 4-5 hours and the deceased had been aggressively treated from 8:00am on 13 May 2011 by the intervention of Dr Said and Dr Beckett.

While I appreciate this supports Professor Hamdorf's

[20] Ex 4
[21] t 7.3.14, p44,49

suggestion the deceased should not have been operated on in GRH, it does not assist with the difficult issue of when GRH should decide which patients to treat for apparently simple surgical intervention at GRH. It also runs the risk of so seriously delaying surgery there is scope for critical recurrent inflammation.[22]

Medical coverage in May 2011 may not have been optimal, and it is Dr Jamieson's concern it is still not optimal in that there can be a lack of continuity, however it is now vastly improved. At the time of the deceased's death there was only one consultant physician available in Geraldton. In addition GRH does not have an ICU. While there are clinicians with intensivist specialties available there is no facility in which they can utilise their specific expertise.[23] This was seen very clearly when Dr Said attempted to provide the deceased with ICU care pending his transfer to Perth by RFDS. Not only did Dr Said, but also two anaesthetists, intervene in the ED resuscitation bay to provide the deceased with the type of support he could have expected in an ICU.

GRH now has three consultant general physicians available for more comprehensive cover. It is now much easier to provide a person in the deceased's position with appropriate clinical review and intervention, however, there is still not an ICU and the HDU is usually operating at capacity.

[22] t 7.3.14 p47
[23] t 7.3.14 p39

From the perspective of GRH I note that of the five inquests[24] held in the last 12 months relating to the deterioration and death of patients post operatively, all of the five had significant comorbidities; three were morbidly obese, one centrally obese and the fifth very frail.

Their risks for invasive procedures of any description were high, but the risk in delaying interventions pending waiting lists in the metropolitan area is also significant. The metropolitan region has difficulty coping with the volume of patients requiring timely elective surgery, without an influx from remote regions for relatively routine procedures.

Due to its lack of an ICU and active intensivists, GRH, even now, has to do what it can by the way of emergency responses to deteriorating patients and the level of resourcing it can realistically provide for the needs of its population.

Dr Jamieson also described GRH's progress in constituting Medical Emergency Teams (MET). In 2014 the GRH MET, designed as a medical emergency response, is a predetermined group of practitioners on any shift who are capable of responding to a medical emergency. They carry

[24] 1. **SQUIRES**, John Gregory heard on 29/04-02/05/2013 (F/No. 7044/09)
2. **JONES**, Wilma Ray heard on 19-21,23/08/2013 (F/No. 7015/09)
3. **FORD**, Valma May Ruth heard on 26-30/08/2013 (F/No. 7056/09)
4. **GILBERT**, Julissa Teresa heard on 4-5/03/2014 (F/No. 7030/08)
5. **DAVIES**, Brian John heard on 7/03/2014 (F/No. 528/11)

pagers which are linked, which call them to locations within GRH experiencing a medical emergency and requiring a rapid response. There is usually a nurse with resuscitation skills, two doctors with resuscitation skills and an anaesthetist. At the time of the deceased's death there was a less formal response to medical emergencies, comprising nurses and the closest appropriate medical practitioner.[25]

CONCLUSION AS TO THE DEATH OF THE DECEASED

I am satisfied the deceased was a 57 year old male with serious comorbidities, including obesity, which affected his ability to recover from surgery. While he appears to have been able to compensate for a lessening in his functioning in every day life, the extent of his physiological difficulties was not apparent prior to his cholecystectomy. He appeared relatively well and was stable pre-operatively, with good oxygen saturations. The extent of his arteriosclerosis was not known but would have restricted his respiratory effort.

I am satisfied there was no significant difficulty with the surgery other than the expected adhesions, the result of prior periods of inflammation, and the deceased's obesity which made visualisation difficult. The procedure was changed from laparoscopic to an open procedure, with a hope this would assist the surgeon. Mr Hudson removed part of the gallbladder due to an inability to properly

[25] t 7.3.14, p42-3

visualise during the procedure but otherwise the procedure was completed without further complication at about 3:50pm.

Following his surgery the deceased's oxygen saturations never returned to a satisfactory level, and on the two occasions he did obtain a reasonable oxygen saturation, it was on high levels of supplied oxygen.

The deceased was agitated in recovery but settled with a non-reversible breather mask. He was transferred to the surgery day ward at 5:50pm where he was observed. It was as a result of his agitation and deterioration medical review was sought and there was a request he be placed in HDU. The deceased was moved to HDU at approximately 8:30pm on the day of surgery. His observations caused the nurses to request the assistance of ED doctors two more times that evening for review and management. It was suspected he was experiencing some renal failure.

The deceased continued to deteriorate and it became obvious he was developing renal failure. By the time Dr Said was able to review the deceased at 7:30am on 13 May 2011 he was in extremis and Dr Said instigated resuscitation methods commensurate with ICU management, concluding with the deceased's transfer to SCGH by RFDS. The deceased reached SCGH and was admitted to ICU at 6:30pm on 13 May 2011.

Despite aggressive intervention the deceased's deteriorating status could not be reversed and he died in the morning of 14 May 2011.

It was apparent by that stage the deceased's acute renal failure had triggered multi organ failure and serious coagulopathy.

I find death arose by way of Natural Causes.

E F VICKER
Deputy State Coroner
27 May 2014