Coroners Act 1996
[Section 26(1)]

Coroner's Court of Western Australia

AMENDED RECORD OF INVESTIGATION INTO DEATH

Ref No: 10/ 19

I, Barry Paul King, Deputy State Coroner, having investigated the death of ***Arthur James Bonney*** *with an inquest held at the* ***Perth Coroner's Court*** *on* ***25 February 2019 and 26 February 2019****, find that the identity of the deceased person was* ***Arthur James Bonney*** *and that death occurred on* ***5 August 2015*** *at* ***Fiona Stanley Hospital*** *from* ***ischaemic heart disease in the context of acute-on-chronic kidney failure in association with contrast-induced nephropathy*** *in the following circumstances:*

Counsel Appearing:

Sgt L Housiaux assisted the Coroner

Ms L Lachal (Aboriginal Legal Service of WA) appeared for Mrs L Bonney

Ms N Eagling (State Solicitor's Office) appeared for the Department of Justice and the South Metropolitan Health Service

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INTRODUCTION

1. At the time of his death, Arthur James Bonney (the deceased) was a minimum security prisoner at Casuarina Prison. His medical history included diabetes, severe cardiac disease and chronic renal failure. He was on stage 3 of the Department of Corrective Service's terminally ill register.

2. On 30 July 2015 the deceased underwent a CT scan of his abdomen at Fiona Stanley Hospital (FSH). He returned to prison on the same day. Over the next two days he was unwell. On 3 August 2015 he was transferred back to Fiona Stanley Hospital where he was admitted into the intensive care unit (ICU) with acute-on-chronic renal failure suspected to have been precipitated by iodinated contrast medium (contrast) administered for the CT scan and by his continuing use of metformin before and after the scan.

3. Despite treatment in the ICU, the deceased's condition deteriorated until he died on 5 August 2015.

4. As the deceased was under the control, care or custody of the Chief Executive Officer of the Department of Corrective Services, he was a 'person held in care' under s3 of the *Coroners Act 1996* (the Act), and his death was a 'reportable death' under the same section.

5. Under s19 of the Act, I had the jurisdiction to investigate the deceased's death because it appeared to me that the death was or may have been a reportable death.

6. Section 22(1)(a) of the Act provides that a coroner who has jurisdiction to investigate a death must hold an inquest if the death appears to be a Western Australian death and the deceased was immediately before death a person held in care. An inquest into the death of the deceased was, therefore, mandatory.

7. Under s25(3) of the Act, where the death is of a person in care, a coroner must comment on the quality of the supervision, treatment and care of the person while in that care.

8. On 25 February 2019 and 26 February 2019, I held an inquest into the deceased's death at the Perth Coroners Court.

9. The main issues at the inquest were: the connection, if any, between the procedures followed at FSH for the CT scan on the deceased and his subsequent death; and the changes that have been made to that procedure since that time.

10. The evidence adduced at the inquest comprised documentary evidence and oral testimony. The documentary evidence comprised a three-volume brief[1] and a copy of an email chain explaining the current processes at FSH for outpatients undergoing CT scans.[2]

11. Oral evidence was provided by (in order of appearance):

    a. Dr Richard Wee, a GP working at the Department of Justice;[3]

    b. Dr Matthew Brookes, a consultant radiologist who was the head of the radiology department at FSH at the time of the deceased's death;[4]

    c. Dr Michael Davis, a consultant cardiologist who managed the deceased's cardiac condition from January 2005 to February 2013;[5]

    d. Richard Mudford, a senior performance officer at the Department of Justice who reviewed the deceased's management while in custody;[6] and

[1] Exhibit 1, Volumes 1, 2 and 3.
[2] Exhibit 2
[3] ts 7 – 23 per Wee, R
[4] ts 23 – 46 per Brookes, M J
[5] ts 49 – 70 per Davis, M D
[6] ts 70 – 92 per Mudford, R P

e. Ms Lillian Bonney, the deceased's wife of 29 years.[7]

THE DECEASED

12. The deceased was born in Kalgoorlie on 22 September 1955, making him 59 years old at the time of his death. He was a Yamagi. His father, Arthur Adams, was from Yalgoo and his mother, Margaret Robertson, was from the Leonora/Kalgoorlie area.[8]

13. When the deceased was five years old, his step-father William Bonney died in a car accident, and the deceased and his five siblings were placed in a mission in Norseman where he experienced mental and physical abuse. He was one of the Stolen Generation.[9]

14. The deceased completed his schooling at Norseman School and was discharged from the mission when he was 16. He was then placed into the care of missionaries at a boys' hostel in Esperance and then went to caretakers in Perth, where he commenced an apprenticeship as a panel-beater. He returned to the boys' hostel before travelling between Esperance and the Goldfields to look for his parents and family.[10]

15. The deceased finally met up with his mother in about 1974 and was getting to know her and his biological father when she died from a stroke or heart problems, possibly related to alcohol. His father died in about 1978 from tuberculosis.[11]

16. Despite his traumatic upbringing, the deceased was kind-hearted and hard-working, and he had a great sense of humour.[12]

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[7] ts 92 – 97 per Bonney, L
[8] Exhibit 1, Volume 1, Tab 7A
[9] Exhibit 1, Volume 1, Tab 7A
[10] Exhibit 1, Volume 1, Tab 7A
[11] Exhibit 1, Volume 1, Tab 7A
[12] Exhibit 1, Volume 1, Tab 7B

17. On 20 June 2013, the deceased was sentenced to seven and a half years' imprisonment from 21 June 2013 for child sex offences. Prior to that, his involvement with the criminal justice system was minimal and had not resulted in a sentence of imprisonment.[13] He was initially placed in Hakea Prison for about two weeks and was then transferred to Casuarina Prison, where he remained until his death.[14]

THE DECEASED'S MEDICAL TREATMENT IN PRISON

18. By June 2013 the deceased had significant medical problems. He had long-standing insulin-dependent diabetes mellitus, lumbar disc surgery, a myocardial infarction in 1997 for which he received bypass surgery at the time and an implantable cardioverter defibrillator (ICD) in 2005. He had strokes in 2003 and 2012, peripheral vascular disease which led to the below-the-knee amputation of his right leg in 2011 and amputation of his left big toe in 2012, and retinal haemorrhage of his left eye in 2012 which resulted in the loss of sight in that eye. On 1 January 2013 he had an episode of ventricular fibrillation, which the ICD treated.[15]

19. On 27 April 2013, consultant cardiologist Dr Davis wrote a letter to the deceased's lawyers, in which he noted that the stress of imprisonment could precipitate further ventricular fibrillation and that the deceased required regular cardiologist review. Dr Davis said that the deceased's life expectancy was poor, even with optimal medical therapy, and that his chance of surviving for five more years was less than 50%.[16]

20. Two days after the deceased was received at Hakea Prison, he experienced sudden cardiac arrest. He was taken to

[13] Exhibit 1, Volume 1, Tab 13
[14] Exhibit 1, Volume 3, report before Tab 1
[15] Exhibit 1, Volume 1, Tabs 18 and 29
[16] Exhibit 1, Volume 1, Tab 18

Royal Perth Hospital (RPH), where he stayed for eight days. He was returned to Hakea Prison and was then transferred to Casuarina Prison as mentioned above. He resided primarily in the infirmary under a standard privileged regime and was employed as an infirmary worker. Prison staff found him to be polite, respectful, compliant and sociable.[17]

21. Over the two years after he had been moved to Casuarina Prison, the deceased was taken to RPH or FSH about 40 times, and for five of those times he was admitted as an emergency patient for heart-related episodes.[18] He also underwent regular reviews by cardiologists and checks on the function of his ICD.[19] His last appointment with a cardiologist was on 16 February 2015 when he saw Dr Vincent Paul at FSH. He reported that he felt that he was managing poorly and was short of breath. Dr Paul changed his prescription for diuretics and arranged to review him again in three months' time.[20]

22. From 2 June 2015 to 5 June 2015, the deceased was admitted to FSH for management of exacerbation of heart failure. Among other things, he was prescribed the diuretic bumetanide. He was also taking the nephrotoxic drugs spironolactone and perindopril for heart failure, and he was taking metformin for diabetes.

23. On 15 June 2015 the deceased underwent an ultrasound scan of his abdomen. The resultant report indicated that he had a 14mm cystic mass within the 4a segment of the liver. The report recommended that he undergo a multiphase CT assessment.[21]

24. On 22 June 2015, Dr Wee completed a request form for a multiphase CT scan. The form indicated that, as well as

[17] Exhibit 1, Volume 3, Tab 10 and report before Tab 1
[18] Exhibit 1, Volume 3, Tab 10 and report before Tab 1
[19] Exhibit 1, Volume 3, Tabs 34A and 34B
[20] Exhibit 1, Volume 3, Tab 34A
[21] Exhibit 1, Volume 1, Tabs 28

heart-related problems, the deceased had diabetes and stage three chronic kidney disease. The request was faxed to FSH Radiology on 3 July 2015.

25. On 6 July 2015 the deceased received a notice from FSH Medical Imaging Department of an appointment on 30 July 2015 for a CT abdomen. The notice included instructions for him to take his medication as normal.[22]

26. Consultant radiologist Dr Brookes explained that, upon receiving Dr Wee's request, the clerical staff at FSH radiology department would have checked with a radiologist or medical imaging technologist to ensure that the appropriate study had been requested and that any necessary instructions were provided to the deceased.

27. Dr Brookes said that the correct study for the deceased was requested and that, when the deceased attended at the appointment, a second stage of inquiry would occur with him to ask about his allergies, diabetes, and medications such as metformin that might impair his renal function. The person making the inquiry would have access to the last available renal function tests; namely, the ones done in early June 2015.[23]

THE CT SCAN

28. On 30 July 2015, the deceased attended FSH and underwent the upper abdomen CT with contrast. In place at FSH at the time was a three-page procedural guideline headed 'Medical Imaging Department – 2015 Guidelines for Iodinated Contrast and Renal Impairment' (the 2015 guidelines). The aim of the 2015 guidelines was said to be to institute measures to reduce the incidence of contrast induced nephropathy (CIN) during CT and arteriography.

[22] Exhibit 1, Volume 1, Tab 15.7
[23] ts 27-28 per Brookes, M J

The method used to reduce the incidence was said to be the identification of at risk patients.[24]

29. The 2015 guidelines provided that it was essential in contrast/interventional procedures to consider the following risk factors:[25]

    a. Age >60 years
    b. Renal Impairment/Dialysis
    c. Diabetic
    d. Taking Metformin
    e. Nephrotoxic Medication
    f. Recent Iodinated Contrast
    g. Asthmatic
    h. Myeloma
    i. Cardiovascular disease/dehydration
    j. Organ transplantation or chemotherapy

30. If any of the risk factors was identified, the patient's estimated glomerular filtration rate (eGFR) was to be considered. The eGFR is a measure of kidney function in millilitres per minute.

31. It is apparent that the deceased had five risk factors (b, c, d, e and i).

32. As noted, the last time the deceased's eGFR had been checked had been in early June 2015. On 2 June 2015 it was 29, but with treatment it improved to 36 on 5 June 2015.[26]

33. The 2015 guidelines stated that, if the eGFR was between 60 and 30, the patient was considered to be at low to moderate risk of CIN. If a patient had an eGFR of less than 30, the risk of CIN was said to be moderate to high. In either case, alternative imaging techniques were to be

[24] Exhibit 1, Volume 1, Tab 15.4
[25] Exhibit 1, Volume 1, Tab 15.4
[26] Exhibit 1, Volume 1, Tab 16B

considered, *nephrotoxic medication was to be stopped*, the lowest dose of contrast was to be used, and *follow up serum creatinine was to be checked 48 hours after the contrast had been injected* (italics added). In addition, if the risk was low to moderate, oral pre-contrast and post-contrast hydration was to occur or be considered; and if the risk was moderate to high, intravenous hydration was to be arranged by a clinical team prior to the scan. None of those steps were taken with the deceased.

34. As noted, the 2015 guidelines listed 'Taking Metformin' as a risk factor. The 2015 guidelines did not indicate that metformin should be stopped if the eGFR was low, but the practice at FSH at the time was to stop it for two days pending review.[27] In the deceased's case, no steps were taken to stop the metformin.[28]

35. Following the CT scan, the deceased was transferred back to Casuarina Prison. Neither his serum creatinine nor his eGFR level was checked within 48 hours of the CT scan.

EVENTS LEADING UP TO DEATH

36. On the morning of 3 August 2015, the deceased presented to the diabetic parade at Casuarina Prison. He saw Dr Wee and complained of generalised body aches for two days and abdominal pain, nausea, loose stools and reduced appetite. He was not in acute distress and his vital signs were normal. He showed no shortness of breath, chest pain or signs of acute heart failure. Dr Wee formed a working diagnosis of acute viral gastroenteritis and prescribed paracetamol and metoclopramide.[29]

37. At about 4.00 pm that day, the deceased informed a prison nurse that he was not feeling well. He was seen by

[27] ts 42 per Brookes, M J
[28] Exhibit 1, Volume 1, Tab 15
[29] Exhibit 1, Volume 1, Tab 34; Exhibit 1, Volume 3, report before Tab 1

Dr Princewill Chuka, who advised that he be transferred to the emergency department at FSH for further assessment.[30]

38. At FSH the deceased presented as hyperkalaemic and acidotic. He was transferred from the emergency department to the ICU in the early hours of 4 August 2015 and was diagnosed with acute-on-chronic renal failure requiring dialysis, possibly precipitated by recent contrast CT and compounded by continuing to take nephrotoxic medications. Cardiogenic shock/low cardiac state was also diagnosed and an underlying septic source was queried.[31]

39. The deceased was placed on continuous venovenous haemodiafiltration, but in the setting of known severe cardiomyopathy and severe metabolic disturbance. Investigations for sepsis were negative. His potassium level returned to the normal range, and by 1.30 pm on 5 August 2015 his creatinine level had also reduced to about his usual level.

40. Despite the signs of improvement to the deceased's kidney function, his cardiac condition fluctuated. He developed increasing inotropic requirements and intermittent ventricular tachycardia. At about 7.30 pm on 5 August 2015, he went into spontaneous cardiac arrest and died.

CAUSE OF DEATH

41. On 12 August 2015, forensic pathologist Dr C T Cooke performed a post mortem examination of the deceased and found myocardial fibrosis with enlargement of the heart and a pacemaker in place. There was a past coronary artery bypass graft with sub-occlusive and occlusive calcification, as well as calcified coronary arteriosclerosis and sclerosis of the mitral valve and aortic valve. The kidneys showed

[30] Exhibit 1, Volume 1, Tab 34
[31] Exhibit 1, Volume 1, Tab 16

atrophic changes and an area of past infarction in the left kidney. There had also been past infarcts in the spleen and the left side of the base of the brain.[32]

42. Microscopic examination of the heart on 21 December 2015 showed fibrous scarring and changes of acute myocardial infarction. The kidneys showed changes consequent to diabetes.[33]

43. It appeared to Dr Cooke that the deceased had died as a result of the failure of his heart, arising on the basis of significant pre-existing ischaemic heart disease. Dr Cooke formed the opinion that the cause of the deceased's death was ischaemic heart disease.[34]

44. In response to a letter from Sergeant Housiaux, in April 2018 Dr Cook reviewed the microscopy of the deceased's kidneys for possible changes of CIN. He found some changes commonly seen in very ill patients, which changes may or may not have related to CIN.[35]

45. Dr Cooke reiterated how the deceased had presented at the emergency department at FSH on 3 August 2015 with high creatinine and potassium and features of cardiogenic shock. By mid-morning on 4 August 2015, after dialysis and other treatment, the potassium had returned to a normal level and the creatinine was progressively reducing until it reached his usual level by 1.30 pm on 5 August 2015. Dr Cooke noted that, while the deceased's renal function had improved, his cardiac condition fluctuated with labile blood pressure and episodes of arrhythmia until he died that evening.[36]

[32] Exhibit 1, Volume 1, Tab 5B
[33] Exhibit 1, Volume 1, Tab 5A
[34] Exhibit 1, Volume 1, Tab 5A
[35] Exhibit 1, Volume 1, Tab 5C
[36] Exhibit 1, Volume 1, Tab 5C

46. Dr Cooke explained that his post mortem examination of the deceased revealed severe heart disease, a recent heart attack and significant diabetic nephropathy.[37]

47. Dr Cooke said that it seemed to him that the main factor in the deceased's death was the significant underlying heart disease; however, it did appear that the acute renal failure may have been a factor in the death by worsening the cardiac status.[38]

48. Dr Cooke said that he had no previous experience with CIN and was unable to reach a firm conclusion regarding the cause of the deceased's acute renal failure, but in view of the close temporal relationship between the renal failure and the CT scan, it seemed that CIN was a real possibility. He indicated that opinions from medical experts, including the radiologist who had taken the CT scan, would be most valuable.[39]

49. On 12 June 2018, the radiologist provided a letter to the Court in which she explained that she was a registrar at the time of the deceased's CT scan, that she had not seen Dr Wee's booking request for the CT scan, and that her involvement with the scan was limited to preparing the report on it under the supervision of a radiology consultant at FSH. She said that, to her knowledge, she had not seen the deceased before the procedure or any other time, so she could not explain why the 2015 guidelines with respect to metformin use were not followed.[40]

50. Dr Brookes provided a report as acting head of the radiology department at FSH at the time that a request for a report was made by the Court. His report addressed specific questions and included a number of relevant documents as attachments.[41] As noted above, he also gave oral evidence.

[37] Exhibit 1, Volume 1, Tab 5C
[38] Exhibit 1, Volume 1, Tab 5C
[39] Exhibit 1, Volume 1, Tab 5C
[40] Exhibit 1, Volume 1, Tab 30
[41] Exhibit 1, Volume 1, Tab 15

His evidence, which I shall discuss below, did not relate directly to the cause of the deceased's death.

51. Professor Joyce provided the Court with a comprehensive and detailed report in which he addressed several complex medical and toxicological issues. The following points which he identified are particularly pertinent to the cause of death:[42]

    a. Prior to the deceased's admission to FSH on 3 August 2015, he experienced renal failure as evidenced by potassium retention, acid retention and urea retention and a myocardial infarction as evidenced by a high troponin I level. The high potassium level was also partly because of the treatment with bumetanide, perindopril and spironolactone.

    b. The deceased had cardiogenic shock (failure of tissue perfusion because of critically impaired cardiac function) that was presumably attributed to a combination of severe chronic heart failure, recent myocardial infarction and slow heart rate from the high potassium level.

    c. In the early hours after admission, both acute renal failure and cardiogenic shock were present, as were chronic heart failure and chronic renal impairment.

    d. The cardiogenic shock and the acute renal failure were mutually causative in that the cardiogenic shock was due to the recent myocardial infarction and slow heart rate from the high potassium caused by acute renal failure. The drugs used to treat the heart failure, perindopril and spironolactone, likely contributed to the worsening of the chronic renal failure through volume depletion, and they became more effective at preventing potassium excretion as

[42] Exhibit 1, Volume 1, Tab 31

the chronic renal failure worsened. At the same time, the cardiogenic shock contributed to the acute renal failure through failure of kidney perfusion.

e. Death occurred when the combined benefits of intravascular volume optimisation, blood pressure elevating drugs and other measures became insufficient to counter the heart failure and to restore vital organ perfusion.

f. The contributors to the deceased's deterioration in renal function were the changes in June 2015 to his treatment for heart failure, the myocardial infarction before his admission on 3 August 2015 and possibly the administration of contrast on 30 July 2015.

g. Metformin probably played some role in the deceased's illness on 3 August 2015 by causing lactic acidosis.

52. Professor Greg Perry, Head of Department of Nephropathy at RPH, provided a report in which he said that the time course of the deceased's acute deterioration in renal function was entirely consistent with CIN. CIN continues for seven to ten days before recovery can be seen with kidney tests returning to base-line. The deceased required dialysis, indicating that his was a severe case. [43]

53. Dr Cyrus Edibam, Head of ICU at FSH, provided a report in which he stated that the continuation of metformin as well as bumetanide, perindopril and spironolactone would have no doubt contributed to the extent of the deceased's renal dysfunction and hyperkalaemia but may not have altered his outcome which was largely related to his cardiac comorbidities.[44]

[43] Exhibit 1, Volume 1, Tab 32
[44] Exhibit 1, Volume 1, Tab 33

54. Cardiologist Dr Davis felt that the continuation of perindopril and spironolactone before the contrast had been administered may well have contributed to the deceased's acute renal failure. He said in oral evidence that doctors have to be very cautious when using those medications when the kidney function is abnormal because any other insult, such as dehydration, gastroenteritis or radiographic contrast, on top of them can dramatically worsen kidney function. He said that he routinely withholds nephrotoxic drugs before putting in contrast to reduce the chance of kidney function declining and then re-introduces them later.[45]

55. Dr Davis did not believe that the continuation of metformin was a major contributor.[46]

56. On the basis of the foregoing, I am satisfied that the deceased, who had long-standing chronic kidney failure and chronic heart failure, underwent a CT scan with contrast while taking nephrotoxic drugs, after which he developed contrast-induced nephropathy, acute kidney failure and a cardiac infarction which led to cardiogenic shock and caused his death.

57. I find that the cause of death was ischaemic heart disease in the context of acute-on-chronic kidney failure associated with contrast-induced nephropathy.

QUALITY OF SUPERVISION, TREATMENT AND CARE IN PRISON

58. The evidence relating to the care provided to the deceased while at Hakea Prison and Casuarina Prison establishes that he had regular ongoing care from infirmary nurses and doctors. He also attended several external specialists and clinics as needed for treatment and assessment.

[45] ts 54 per Davis, M D
[46] Exhibit 1, Volume 1, Tab 29

59. I am satisfied that the treatment and care which the deceased received in prison was timely and appropriate.

60. The evidence relating to the supervision at Casuarina Prison so far as it pertains to his death was also appropriate except, perhaps, with respect to the food provided to him. I note in particular Ms Bonney's oral evidence that the deceased had complained about the food, and that is was inappropriate for someone on a diabetic diet.[47] This issue was not canvassed at the inquest, so I make no relevant finding.

61. I also note that Ms Bonney said in a statement that the prison officers guarding the deceased at FSH were nice and respectful in allowing her and the deceased to have some privacy.[48]

DISCUSSION ON TREATMENT AND CARE AT FSH

62. As noted above, when the deceased attended FSH on 30 July 2015 for the CT scan, he was not managed in accordance with the then current 2015 guidelines. 'Essential' procedures identified in the 2015 guidelines, including stopping nephrotoxic drugs, administering oral hydration and follow-up creatinine testing, were not done. Moreover, assuming that the 2015 guidelines were considered, the eGFR that was available was almost certainly wrong because of the deceased's deteriorating kidney function since it had been checked in early June 2015.[49]

63. Dr Davis emphasised that his practice when doing radiographic studies was to withhold nephrotoxic drugs and

[47] ts 95 per Bonney, L
[48] Exhibit 1, Volume 1, Tab 7
[49] Exhibit 1, Volume 1, Tab 31

always to routinely re-check kidney function after a contrast study.[50]

64. However, Professor Joyce noted that keeping the deceased free of heart failure required continuation of bumetanide, spironolactone, perindopril and fluid restriction. He said that this would have posed a dilemma for the radiologist.[51]

65. Professor Joyce stated that the first question would be whether doing a scan without contrast would provide a better balance of risk and benefit. If contrast was the most appropriate option, the procedure might go ahead with a cautious administration of fluids before and after the procedure and earlier testing for re-emergent heart failure and falling eGFR and urine output after the procedure.[52]

66. Professor Joyce said that the management of the deceased at FSH on 30 July 2015 was not in accordance with the 2015 guidelines, but rigid adherence to them, which I infer to mean withholding nephrotoxic drugs and administering fluids, would have put the deceased at risk of clinical heart failure.[53]

67. Dr Brookes said that he believed that doing the CT scan with contrast was the right decision given what was known at the time.[54] However, he said that, since 2016, FSH had had the technology to check a patient's renal function, including the eGFR, within two or three minutes and that such checks are now routinely done before contrast is administered for a CT scan.[55]

[50] ts 54 per Davis, M D
[51] Exhibit 1, Volume 1, Tab 31
[52] Exhibit 1, Volume 1, Tab 31
[53] Exhibit 1, Volume 1, Tab 31
[54] ts 33 per Brookes, M J
[55] ts 39 per Brookes, M J

68. Dr Brookes also said that stopping medication is not often done because of the potential consequences, especially with cardiac medications for patients with cardiac failure.[56]

CURRENT PROCEDURES

69. Dr Brookes noted that in July 2016 the Royal Australian and New Zealand College of Radiologists (RANZCR) produced a guideline based on the latest available research: Iodinated Contrast Media Guideline, 2016 Version (the 2016 guideline) which FSH now follows, and that the current version of the FSH guideline relates only to metformin management. In other words, nephrotoxic medications are not addressed, indicating that RANZCR does not recommend stopping them before CT scans with contrast.

70. It is worth noting that the 2016 guideline states that the risk of intravenous 'contrast-induced acute kidney injury', which I take to be the same or similar to CIN, is uncertain for patients with an eGFR of 30 to 45, but if there is a risk, it is greatest for those with an eGFR of less than 30.[57]

71. Dr Brookes also said, in relation to ceasing metformin where a patient's eGFR is less than 30, that the issue of metformin was not so relevant in such cases because it is unlikely that contrast will be given at all.[58]

72. The 2016 guidelines recommend that, in patients with severe renal function impairment (eGFR less than 30 or actively deteriorating renal function) careful weighing of the risk versus the benefit of the use of contrast needs to be undertaken. However, such impairment should not be regarded as an absolute contraindication to medically indicated use of contrast.[59]

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[56] ts 35 per Brookes, M J
[57] Exhibit 1, Volume 1, Tab 15.6
[58] ts 42 per Brookes, M J
[59] Exhibit 1, Volume 1, Tab 15.6

73. On the basis of the foregoing, it is clear that, when the deceased attended FSH on 30 July 2015 to undergo a CT scan, he was not managed in accordance with the 2015 guidelines. However, his care would have accorded with current practices under the 2016 guidelines had his actual eGFR been determined and considered before proceeding with the administration of the contrast. As noted above, if the eGFR from June 2015 was used, it was misleading.

74. Had the deceased's actual eGFR been identified as less than 30, it seems clear on Dr Brookes' evidence that the deceased would not have been given the contrast.

75. Dr Brookes said that the current practice at FSH is for the eGFR to be checked on the day of a planned CT scan and, if it is less than 30, metformin is ceased for at least 48 hours and not restarted until the eGFR is back to an acceptable level. Pre and post-procedure hydration is only considered in consultation with a cardiologist.

76. Following the inquest, Dr Brookes provided through Ms Eagling an email in which the current process for CT outpatients at FSH is further described.[60] That process is as follows:

    a. all CT requests received in the radiology department are reviewed by the Chief Medical Imaging Technician (Chief MIT) prior to any booking being made;

    b. the Chief MIT ensures that the request is appropriate and brings any concerns, such as patients with poor renal function, diabetes or allergies, to the attention of a radiologist;

    c. the radiologist then advises if the booking is to be made or if further information is needed from the requesting practitioner;

[60] Exhibit 2

d. due to the increased risk of problems following up CT scans administered to prisoners, all requests for prisoners must be referred by the Chief MIT to a radiologist, who can contact the relevant prison or prison doctor; and

e. on the day of the CT scan, a patient questionnaire is completed and if contrast is used, a blood sample is taken for an eGFR test. The results of the test are available within minutes, and the contrast CT does not commence until the results are reviewed by a radiologist.

HOW DEATH OCCURRED

77. In my view, the administration of contrast to the deceased on 30 July 2015 probably led to CIN and contributed to his cardiac failure. The degree of that contribution is not possible to ascertain, but from a practical standpoint, the chronology of the deceased's deterioration from 30 July 2015 to 5 August 2015 suggests that there was some contribution. However, the evidence also indicates that the treatment in the ICU reduced the symptoms of kidney failure so that, by 5 August 2015, it was no longer acute.

78. While Professor Joyce indicated that the acute kidney failure both caused and was caused by the heart failure, the evidence also indicates that, as Dr Cooke said, the main factor was the significant underlying heart disease. To the extent that not withholding the nephrotoxic drugs may have led to CIN and contributed to the heart failure, those drugs were, ironically, necessary to ensure that heart failure did not occur.

79. In these circumstances, and notwithstanding my conclusion that CIN made some contribution to the heart failure, I find that death occurred by way of natural causes.

CONCLUSION

80. Though the deceased was relatively young at the time, when he was first admitted to prison he had several significant medical conditions, including severe cardiomyopathy. Within two days of being admitted, he had a cardiac arrest. His medication was adjusted but, as was anticipated, his condition deteriorated further over time, requiring ongoing treatment and investigations.

81. Following an ultrasound scan in May 2015, in late July 2015 the deceased underwent a CT scan with iodinated contrast which was followed by acute kidney injury and treatment in the ICU. That treatment rectified the symptoms of the kidney injury, but he experienced a spontaneous cardiac arrest and died.

82. The evidence at the inquest established that the use of contrast in the CT scan and the resulting acute kidney injury likely had some contribution to the death, but that the deceased's underlying heart condition was the major cause of death.

83. Welcome changes to procedures at FSH have reduced the likelihood that patients who attend the radiology department as outpatients for a CT scan will develop contrast-induced kidney injury.

B P King
Deputy State Coroner
12 August 2019