CITATION:	*Inquest into the death of Ben James WITHAM* [2012] NTMC 036
TITLE OF COURT:	Coroners Court
JURISDICTION:	Darwin
FILE NO(s):	D0075/2011
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HEARING DATE(s):	28 – 30 August 2012
FINDING OF:	Mr Greg Cavanagh SM
CATCHWORDS:	**Death by natural causes, adequacy of medical care, arsenic poisoning, closure of property.**

REPRESENTATION:

Counsel Assisting:	Ms Elisabeth Armitage
Department of Health:	Ms Leonie Paulson
Women's and Children's Hospital Adelaide:	Mr Todd Golding
Dr Michael Fonda:	Ms Maria Savvas

Judgment category classification:	B
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IN THE CORONERS COURT
AT DARWIN IN THE NORTHERN
TERRITORY OF AUSTRALIA

No. D0075/2011

In the matter of an Inquest into the death of

BEN JAMES WITHAM
ON 24 MAY 2011
AT WOMEN'S AND CHILDREN'S HOSPITAL, ADELAIDE

FINDINGS

(Delivered)

Mr Greg Cavanagh SM:

Introduction

1. Master Ben James Witham (Ben) was admitted into Royal Darwin Hospital on 24 April 2011. He was acutely ill and remained hospitalised until his death. Ben turned 17 in the Women's and Children's Hospital, Adelaide. Just 17 days later, on 24 May 2011, Ben died from multi organ failure with terminal fungal sepsis with a background of acute lymphoblastic leukaemia.

2. Although acute lymphoblastic leukaemia (ALL) was diagnosed at the commencement of Ben's hospitalisation, Ben's presentation was considered so unusual that the diagnosis was questioned. Alternative explanations for Ben's complicated symptoms were considered, including possible exposure to an external, environmental, insult such as poisoning. Accidental environmental exposure to a toxin was thought possible, in part, because Ben lived on a rural property, Mount Bundy Station.

3. On 25 May 2011, the day following Ben's death, test results from a urine sample taken from Ben on 17 May 2011 were returned. The results indicated arsenic levels in Ben's urine that were 14 times higher than the normal upper limit for arsenic. Some doctors thought that some of Ben's unusual

symptoms were consistent with arsenic poisoning. Accordingly, the possibility that Ben was poisoned, perhaps from environmental exposure, was considered real. Further, a potential risk to the public from environmental exposure was identified.

4. On 1 June 2011 residents of Mount Bundy Station provided urine samples for testing. The test results were returned to the local Adelaide River General Practitioner (GP) on 11 June 2011. The GP, who had no previous experience in interpreting arsenic testing results, thought the results showed elevated levels of arsenic. He reported his opinion of the results to the Centre for Disease Control.

5. In light of that opinion and Ben's urine test result, a teleconference was convened by the Chief Executive Officer of the Department of Health. The teleconference was attended by the Adelaide River GP and representatives of the Northern Territory Police Force, Environmental Health, and the Centre for Disease Control. The interpreted test results suggested that residents of Mount Bundy Station were being exposed to arsenic. In the interest of public safety it was agreed that Mount Bundy Station should close pending further investigation of any potential arsenic source. Pursuant to section 32 of the *Coroner's Act*, Mount Bundy Station was declared a restricted area.

6. Over the next days, extensive water and soil sampling was conducted. None of the soil or potable water samples returned elevated results for arsenic.

7. Professor Alison Jones, a poisons expert, was identified and agreed to assist the investigation. All testing results were referred to Professor Jones. On reviewing the residents' urine results, Professor Jones determined that they were not elevated for arsenic. It appeared that the complicated reporting of the results had been misinterpreted by the Adelaide River GP. Following the all-clear on the testing results (urine, soil, and potable water) on 15 June

2011 Professor Jones advised that it was safe to re-open Mount Bundy Station. The restricted area declaration was lifted.

8. Following the autopsy, pathology samples from Ben were tested for arsenic, including hair, blood and tissue. None returned elevated arsenic readings.

9. Professor Jones assisted the inquest by reviewing all Ben's medical records and test results. Professor Jones provided an expert opinion that Ben was not exposed to arsenic. Further, Professor Jones considered it likely that the elevated arsenic reading from the 17 May 2011 urine sample was due to "lab error" or "contamination". I accept those opinions. I find that Ben did not suffer or die from arsenic poisoning and that the single positive arsenic test result was caused by contamination or error.

10. Throughout the course of his hospitalisation Ben was seriously ill and suffered from a "complicated constellation of symptoms". In the inquest concerns about Ben's medical care were raised by his parents, James ("Scott") and Sue Witham. I considered the quality and timeliness of the medical care provided to Ben by medical staff at the Adelaide River Clinic, Royal Darwin Hospital and The Women's and Children's Hospital, Adelaide. Save for one significant incident, I find Ben's care to be reasonable and appropriate.

11. On 13 May 2011 Ben's condition deteriorated. He suffered from the onset of severe abdominal pain. Ben's mother and grandmother were desperately concerned about him and repeatedly reported their concerns about his changed condition to medical staff. Ben was reviewed by the consultant oncologist. At the time of review, Ben's symptoms ought to have alerted the consultant to the possibility that Ben was suffering from a perforated stomach or acute abdomen, which demanded further urgent investigation by x-ray, scan, or surgical review. However, no further investigations were ordered. Additional pain relief was provided, but it was inadequate for the level of pain suffered by Ben, and failed to address his critical condition.

The following day, approximately 17 hours after the onset of the acute pain, Ben underwent a laparotomy to repair a perforated stomach.

12. I find that the medical care provided to Ben following the onset of his severe abdominal pain until the surgery was not of a standard that was adequate or appropriate to his critical condition. In particular, further available investigations as to the cause of the pain should have been conducted in a much more timely fashion, that is, overnight. X-rays, scans, or surgical review were available and they should not have been left to the following day. Ben never recovered from this event and I find it to be a contributing cause to his death.

13. Ms Elisabeth Armitage appeared as Counsel Assisting, Ms Leonie Paulson appeared for the Department of Health (NT), Mr Todd Golding appeared for the Women's and Children's Hospital (and its medical staff), and Ms Maria Savvas appeared for Dr Michael Fonda. The death was thoroughly investigated by Detective Acting Sergeant Joedy Kitchen and Detective Sergeant Isobel Cummins. I received into evidence their detailed investigation brief. I also received the medical records from the Adelaide River Clinic, Royal Darwin Hospital and the Women's and Children's Hospital, Adelaide. I heard evidence from Dr John Gilbert, Mr James Scott Witham, Mrs Susan Witham, Dr Michael Fonda, Ms Renae Heath, Dr Rodney Omond, Dr Day Way Goh, Dr Akash Kalro, Dr Stephen Keeley, Dr Celia Cooper, Dr Michael Osborn, Dr Tareq Kamleh, Dr Jane Smith (nee Healy), Dr Tames Revesz, Commander Jamie Chalker, Acting Detective Sergeant Joedy Kitchen, Dr Barbara Paterson, and Professor Alison Jones.

14. Pursuant to section 34 of the Coroners Act, I am required to make the following findings:

> "(1) A coroner investigating –
>
> (a) a death shall, if possible, find –

(i) the identity of the deceased person;

(ii) the time and place of death;

(iii) the cause of death;

(iv) the particulars needed to register the death under the Births, Deaths and Marriages Registration Act;

15. Section 34(2) of the Act operates to extend my function as follows:

> "A coroner may comment on a matter, including public health or safety or the administration of justice, connected with the death or disaster being investigated."

16. Additionally, I may make recommendations pursuant to section 35(1), (2) & (3):

> "(1) A coroner may report to the Attorney-General on a death or disaster investigated by the coroner.
>
> (2) A coroner may make recommendations to the Attorney-General on a matter, including public health or safety or the administration of justice connected with a death or disaster investigated by the coroner.
>
> (3) A coroner shall report to the Commissioner of Police and Director of Public Prosecutions appointed under the Director of Public Prosecutions Act if the coroner believes that a crime may have been committed in connection with a death or disaster investigated by the coroner."

Background

17. Ben was born in Katanning, Western Australia, where his family lived and worked on a station in Broomehill. In 2007 the family bought Mount Bundy Station and moved to the Northern Territory. Ben was 13 years old. Ben was initially home schooled and then commenced Year 9 at Batchelor Area School followed by Years 10, 11 and 12 at Taminmin College.

18. Ben led a healthy lifestyle enjoying motor cross, horse riding and playing sports such as cricket and volleyball. He was popular with his school friends

and he enjoyed the outdoors and farming lifestyle. Apart from the usual childhood illnesses, he was never sick.

19. Ben is survived and deeply missed by his parents Scott and Sue Witham, his sisters Casey and Rebecca, and extended family.

Relevant circumstances surrounding the death

Presentations at the Adelaide River Clinic and transfer to Royal Darwin Hospital

20. Ben first reported feeling unwell in early April 2011. In a text message to his girlfriend he said he was feeling tired and had aching joints. He had two days off school and on 14 April he saw Dr Fonda, the local GP at the Adelaide River Clinic. Ben reported feeling generally unwell, he was run down, had aching joints, was feverish and had mild headaches. Blood tests were taken and a follow up appointment was made. Dr Fonda thought Ben was probably suffering from a viral infection.

21. On 20 April 2011 Ben re-attended at the Adelaide River Clinic for review. He saw a nurse and she recorded that he reported feeling "well and has no further weakness". No fever was noted. As the first blood test had returned with some abnormalities further tests were conducted to monitor his progress. The second blood test again returned with slightly abnormal results. The abnormalities on both tests were non-specific and consistent with the diagnosis of viral infection. However, a further follow up appointment was arranged.

22. On 23 April 2011, before the next scheduled appointment, Ben's health deteriorated. He had a fever, was tired, vomiting, and experiencing bouts of diarrhoea. Ben attended the Adelaide River clinic with his Mum at about 4.00 pm. He was seen by another district nurse. As procedure required the nurse contacted the District Medical Officer (DMO). She provided detailed information about Ben's condition to the DMO including; temperature, blood pressure, heart rate, respiration rate, oxygen saturations, test results

and general observations. The nurse sought directions on Ben's care and treatment. Acting on those directions, further pathology was taken for testing, and anti-nausea medication (Maxalon), Panadol and oral rehydration salts were provided. Neither the DMO nor the nurse considered hospitalisation was required at that stage and Ben was sent home with his mother. I note in passing that the DMO considered Ben's blood results, and felt they were improving. He also thought them to be consistent with a viral illness.

23. Ben's condition deteriorated overnight with high fevers, delirium and vomiting. The next day was a Sunday. Following a phone call from Mrs Witham, the district nurse attended and opened the clinic specifically to see Ben. The nurse sought further advice from the DMO and continued to monitor, stabilise, and rehydrate Ben in the clinic for about two hours. At about 10.00 am, in consultation with the DMO, it was agreed that Ben was not improving and that he should attend Royal Darwin Hospital. Ben's parents agreed to take him in the family car.

24. Ben arrived at the emergency department at 11.47 am. His health had worsened en route. Ben again deteriorated sharply about 30 minutes after arrival. Ben was given a new diagnosis of acute lymphoblastic leukaemia, septic shock, and tumor lysis syndrome. It was determined he should be transferred to the Women's and Children's Hospital, Adelaide, as soon as he could be stabilised.

25. The Royal Darwin Hospital records contained the following comment:

> "Unfortunately the decision was made to transfer him by car to Darwin and no antibiotics were given".

Accordingly, I considered whether:

(i) the initial working diagnosis of viral infection was reasonable in the circumstances,

(ii) there was any unjustified delay in the decision to send Ben to hospital, and

(iii) the decision to allow Ben to travel to Darwin with his parents was appropriate in the circumstances.

26. As to the initial diagnosis of viral infection, I heard evidence from Dr Fonda, Dr Omond, and Dr Kalro. All agreed that the blood test results were non-specific and consistent with a diagnosis of viral infection. Dr Kalro, a specialist haematologist, explained that the leukemic process can mimic a viral infection and it was not uncommon for patients to experience some weeks of generally feeling unwell before being referred to specialist services. Accordingly, I accept that the initial diagnosis of viral infection was reasonable based on Ben's presenting symptoms and blood results.

27. As to the timing of the decision and method of transfer from Adelaide River clinic to Royal Darwin Hospital, I heard evidence from District Nurse Renae Heath, DMO Dr Omond, Dr Fonda, and Dr Kalro. I am satisfied that prescribed procedures were followed at the Adelaide River clinic and that clinically appropriate decisions were made, based on the information available to the treating practitioners. There was nothing about Ben's presentation which indicated transfer by ambulance was necessary. Ben had been observed for some time and appeared relatively stable when the decision was made to transfer him by private car.

Transfer from Royal Darwin Hospital to Women's and Children's Hospital, Adelaide and quality of care provided at the Women's and Children's Hospital

The Paediatric Intensive Care Unit

28. On Tuesday 26 April 2011 Ben was transferred by Care Flight to the Women's and Children's Hospital, Adelaide. He was admitted to the Paediatric Intensive Care Unit (PICU). He was considered to be critically unwell and was suffering from a complicated constellation of symptoms and

disorders including: multi-system failure with cardiogenic shock (renal, respiratory, and marrow failure), tumor lysis syndrome, was inexplicably deeply comatose, and suffering from a rash.

29. Diagnosis of Ben's primary underlying condition remained acute lymphoblastic leukaemia. Markers suggestive of a leukaemia diagnosis were detected in a bone marrow biopsy on 27 April 2011. Ben was commenced on steroid therapy (Methylprednisolone) as an anti-leukemic intervention.

30. On admission Ben weighed about 70 kilograms. By 12 May 2011 Ben's weight had dropped to about 58 kilograms. The dosage of Ben's steroid therapy was in part determined by his weight. However, the dosage did not change in line with his weight loss.

31. One issue of concern to Ben's parents was the possibility that due to his significant weight loss, Ben was overmedicated. Both Dr Keeley and Dr Osborn gave evidence about this. They told me that the steroid therapy provided was well within the recommended range and that steroid dosages were not normally altered in response to patient weight loss or gain. Their combined evidence refuted any concern that Ben had been "overdosed".

32. Unusually, in response to the steroid therapy alone (and without chemotherapy) Ben's leukaemia went into remission. On 9 May 2011 the medical notes reveal that Ben was no longer neutropenic.

33. As Ben improved, the PICU life-saving interventions were gradually reduced. Ben was able to breathe for himself and was extubated on 10 May 2011.

The Brookman Ward and a stomach perforation

34. On 12 May 2011 Ben was transferred from PICU to Oncology, the Brookman Ward. Ben commenced solid food. Ben's mother and grandmother (a nurse) were concerned at the hardness of the food being offered to Ben.

35. During the afternoon of 13 May 2011 Ben started experiencing stomach pains and diarrhoea of a dark colour. Throughout the afternoon and evening Ben's mother and grandmother were very concerned about what appeared to them to be a significant and obvious change in his condition. They repeatedly reported their concerns to medical staff and requested further investigation of the cause of Ben's abdominal pain.

36. By 9.00 pm Ben was experiencing severe abdominal pain. At 9.30 pm he was seen by Dr Hauser. The medical notes record the following abdominal observations:

> "generalised tenderness, guarding, rebound tenderness, tenderness with auscultation, Imp acute pain, case discussed with Dr Revesz".

37. At about 11.00 pm Dr Revesz attended on-call and determined that Morphine should be given. No notes were made in Ben's records at this time. Mrs Witham told me that Dr Revesz made no independent physical examination of Ben and left without discussing any plan with her. Mrs Witham thought that Dr Revesz had walked away "to organise something", but she was later told by nurses that nothing would happen until the morning.

38. At 11.30 pm Ben was seen by an anaesthetic Registrar. The Registrar recorded the following observations:

> "sudden increase in abdo pain this evening. Pain 7/10 (after 10 mg IV morphine), all over abdo, nowhere else in body, sharp, knife like radiation, worse with movement, no pain like this while in ICU or any time previously, o/e tender throughout, abdo firm, equal pain on palpating and releasing,? Unclear if peritonitis. Reviewed by oncology consultant at approx 23.00, thought to be likely due to diarrhoea? Ulceration but unlikely any surgical pathology at this stage".

39. On 14 May 2011 at 2.30 am Dr Healy recorded the following observations in Ben's notes:

> "Onset of abdo pain at 21.00, constant, cramping, generalised abdo pain, unable to localise, has become progressively worse since then in increasing pain, pain score 9/10….groaning in pain, distressed, abdo distended, tense, peritonitis generalised, percussion on tenderness, unable to hear bowel sounds".

40. In this inquest I heard moving evidence from Ben's mother about that terrible night. She told me Ben was screaming in pain, that:

> "they just gave him more morphine",

and that she felt like they were in:

> "a third world country".

41. On 14 May 2011 at about 11.00 am Ben was reviewed by a surgical Registrar who noted:

> "(stomach) perf could be anywhere + ? how long has been present for".

42. A CT scan was ordered to isolate the location of the perforation. However, the CT scan could not be completed because Ben "crashed" and a code blue was called at 11.45 am. Ben was resuscitated.

43. At about 2.30 pm, an emergency laparotomy was performed to close a gastric perforation.

44. Ben was transferred back to PICU but never recovered.

45. Taking the recorded 9.30 pm (13 May 2001) observations as a starting point, Ben experienced approximately 14 hours of severe and increasing abdominal pain before a scan of his abdomen was attempted. There was approximately 17 hours between onset of severe abdominal pain and surgery to correct a perforated stomach.

46. Dr Goh, who performed the abdominal surgery, told me that the signs of perforation would include, blood passing in the stool, severe abdominal pain, a tender, and rigid abdomen on examination. In my view, those signs

were all present at 9.30 pm and became increasingly extreme over the course of the night. Dr Goh said an abdominal x-ray was the best way to determine if there was a perforation and that x-rays were available over night at the hospital. He told me that a delay of 15-17 hours between perforation and repair would allow peritonitis and bacterial infection to develop. Dr Goh considered that the stomach perforation was a significant contributing cause of death.

47. Dr Keeley told me that the change in Ben's condition on the evening of 13 May 2011 contained classic, clinical, signs that something acute was happening. He said such an acute event called for x-rays, scanning and/or surgical management. Dr Keeley agreed that Ben's symptoms on the night of 13 May 2011 should have been investigated further as a matter of urgency. He agreed that the surgical delay increased Ben's risks of bacterial and fungal infection.

48. Dr Osborn was questioned about the 15-17 hour delay between Bens' apparent stomach perforation and surgery. When asked

> "is that sort of delay an appropriate standard of care from the Women's and Children's Hospital?"

he frankly responded:

> "I would have thought not".

49. Dr Revesz gave evidence. He told me that he relied on the description of the junior doctor as the basis for his decision to, in effect, "wait and see" how Ben progressed. I note that this evidence is consistent with Mrs Witham's observations. Dr Revesz said that:

> "the way Ben's condition was described to me did not lead me to think that it was a significant event at the time".

50. He agreed he would have been better placed to make decisions about Ben's diagnosis had he performed a physical examination himself. Dr Revesz

agreed that the standard of Ben's care from late 13 May 2011 until an x-ray was ordered the following day was inadequate.

51. Dr Gilbert, who performed the autopsy, gave evidence that the gastric perforation was an:

> "overwhelming insult from which Ben did not recover"

and that it was:

> "a significant insult that has contributed to his death".

52. It is abundantly clear that the delay in further investigating Ben's abdominal pain between 9.30 pm on 13 May 2011 and 11.30 am on 14 May 2011 was unacceptable. Further, that the care provided to Ben during the course of that night was inadequate for the acute and severe nature of his condition. I find that Ben was wrongly diagnosed and did not receive the further investigations or surgical review that his symptoms demanded.

53. The missed diagnosis and subsequent failure in care does not appear to have arisen from any systemic failing, rather it arose from a wrong clinical decision on one occasion by Dr Revesz. No recommendations are required.

54. I find that the gastric perforation and the delayed response to it were contributing causal factors to Ben's death.

What caused the perforation?

55. Immediately following Ben's surgery, one of the surgeons spoke to Mrs Witham. Mrs Witham could not recall the name of the surgeon but described him as being of Indian appearance. Mrs Witham sent a text to her husband reporting what she had been told: that Ben had made it through surgery, there was a two inch tear close to his oesophagus, and it was a mechanical tear that may have been caused by the removal of the gastro tube.

56. Over the following days, no other medical staff mentioned a “mechanical tear” to Mrs Witham. PICU staff told her the perforation was caused by ulceration likely due to a weakening of the stomach lining as a side effect of the steroid therapy.

57. As a result of these conflicting opinions, Ben’s family were concerned to know whether there was any evidence that Ben’s feeding tubes had caused the perforation. They also wondered whether starting Ben on solids caused the perforation.

58. Dr Goh was the surgeon who spoke to Mrs Witham immediately following the surgery. In his evidence before me he was adamant that he did not use (indeed had never used) the term “mechanical tear”. He also told me that in his experience feeding tubes do not cause perforations. He thought it likely that the perforation was caused by a gastric ulcer.

59. I experienced some difficulty with this aspect of Dr Goh’s evidence. Firstly, Dr Goh did not have records of his conversation with Mrs Witham and was relying entirely on his memory, while Mrs Witham had recourse to her text message. Secondly, Dr Keeley and Dr Osborn told me that a feeding tube (even a correctly placed one) might contribute to or cause stomach perforations, particularly in a case like Ben’s where the stomach lining might have been weakened from steroid therapy or simply from the general stress caused by ill health and prolonged hospitalisation. Dr Gilbert also agreed that a nasogastric tube could cause or contribute to perforation.

60. Ultimately, neither Ben’s treating doctors nor Dr Gilbert could provide anything other than speculation as to what caused Ben’s stomach to perforate but they did discount any connection with commencement on solid foods. Accordingly, whilst I accept Mrs Witham’s account of the conversation she had with Dr Goh, on the evidence available I am not able to find the cause of the perforation. The weight of evidence suggests that it

occurred as a further complication on a multifactorial and complicated background of ill health.

A fungal infection

61. On 27 April 2011 Aspergillus (a common fungus) was detected in a sample of Ben's nasopharyngeal aspirate. As a precautionary measure against Ben's suppressed immune system, Ben was commenced on an anti-fungal medication (Ambisone). However, there was no continuing evidence of a fungal infection (an MRI conducted on 29 April 2011 was clear), and as Ben's white blood cell count had improved, the anti-fungal medication was ceased on 11 May 2011. It was recommenced on 16 May 2011 following the stomach surgery.

62. On 19 and 22 May 2011 biopsies revealed deep fungal infections around Ben's heart, lungs and brain. According to Dr Gilbert, it was ultimately the fungal infection that overwhelmed Ben resulting in multi organ failure and death.

63. I considered whether there was sufficient evidence to determine when this deep fungal infection took hold, particularly in light of the cessation of anti-fungal medication and the stomach surgery.

64. On autopsy, Dr Gilbert found residual food particles in Ben's abdominal cavity. He considered it possible that fungal spores from stomach contents could have leaked into the abdomen at the time of perforation. He thought that if no anti-fungal medications were given at that time (and the records indicate they were not given), then an opportunity was created for a fungal infection to take hold. However, he could not say with any certainty when the fungal infection took hold. Dr Goh and Dr Keeley agreed that stomach contents could contain fungal spores, Dr Osborn disagreed. However, no-one was able to provide a firm opinion as to the commencement of the infection.

65. I heard evidence from Dr Cooper, a specialist in infectious diseases. She described how Ben's weakened immune system (from both leukaemia and its treatment) made him susceptible to fungal infection. She did not think it was possible to determine when the fungal infection took hold, but did not think the cessation of anti-fungal medication for five days was of significance. She explained that anti-fungal medications build up over time and remain in the system for some time after they are ceased. She felt that cessation of the Ambisone for a few days was unlikely to have caused any significant change in Ben's overall Ambisone levels.

66. In my view, the evidence as to when the fungal infection commenced is inconclusive. Accordingly, I am unable to find when the fungal infection took hold.

The diagnosis is questioned

67. Although Ben's treating doctors were working with an underlying diagnosis of acute lymphoblastic leukaemia, that diagnosis did not appear to sufficiently explain all the multi-system problems he was experiencing. Extensive testing was conducted to determine whether there was another or other additional causes for his illness. As testing had excluded serious and rare infections, Dr Cooper considered whether Ben had been exposed to a chemical or toxic insult such as a poison.

68. On 20 May 2011 Dr Cooper requested that the oldest available urine sample be tested for arsenic and lead. The sample was dated 17 May 2011. The test result was returned on 25 May 2011, the day after Ben's death. The result showed that Ben had 14 times more arsenic in his urine than the normal upper limit. Dr Cooper thought that arsenic poisoning explained many of Ben's unusual symptoms.

69. The autopsy was conducted on 27 May 2011. The provisional cause of death dated 30 May 2011 was "multi organ failure with terminal fungal sepsis,

query underlying arsenic toxicity, pending histology and toxicology". Arsenic poisoning remained a possibility.

70. Pre and post mortem pathology samples, including hair and tissue were subjected to further analysis for arsenic and the results were sent for interpretation to international experts in the field. None of the further tests detected any abnormal levels of arsenic. However, it took some time before all tests were finalised.

71. Ben's medical files, all test results, and the brief of evidence prepared for this inquest, were provided to Professor Jones of the University of Wollongong for an expert opinion. Professor Jones provided a report dated 13 April 2012 and she gave evidence. In summary, Professor Jones concluded that "the urine, blood, hair and tissue chemistry and pathology results are not compatible with arsenic poisoning as a cause of death". Further, it was her opinion that Ben's clinical presentation was not consistent with arsenic poisoning.

72. As to the elevated arsenic reading from the urine sample of 17 May 2011, Professor Jones was of the opinion that it was either a false positive or the sample was contaminated, for example, by dust. Possible contamination from plastic containing an arsenic compound (such as a catheter), postulated by Dr Gilbert, was not excluded.

73. I accept Professor Jones expertise and opinions. I find that Ben did not die from arsenic poisoning, nor did arsenic contribute to his death. I further find that the single urine sample that returned an elevated reading was either contaminated or an error.

74. Ben's initial bone marrow biopsy was sent to the Children's Cancer Institute in Sydney to test for monoclonal markers. Three monoclonal markers were detected. Dr Revesz discussed the results with Professor Jacques Van Dongen, of the Netherlands, an expert in acute lymphoblastic leukaemia.

Professor Van Dongen confirmed that finding monoclonal markers was the best diagnostic test for acute lymphoblastic leukaemia, and it was his opinion that this test confirmed the diagnosis.

75. I find that Ben was suffering from acute lymphoblastic leukaemia when the bone marrow biopsy was taken on 27 April 2011. This illness and its treatment resulted in immunosuppression which provided a background for the development of the terminal fungal infection.

The responses to arsenic being considered a possible cause of death

76. Before further testing was completed on Ben's pathology and while arsenic poisoning was considered a possible cause of his death, Dr Fonda was advised of Ben's positive arsenic result. Dr Fonda spoke to the Centre for Disease Control and the risks associated with a possible case of arsenic poisoning were, appropriately in the circumstances, taken seriously. Dr Fonda was advised to test other residents of Mount Bundy Station for arsenic. Environmental Health Services were tasked to conduct soil and water sampling.

77. On 1 June 2011 ten residents of Mount Bundy Station attended the Adelaide River Health Clinic and provided urine samples for testing.

78. On 3 June 2011 representatives from the Department of Health, Environmental Health officers, Northern Territory Police, Worksafe, and the Department of Natural Resources inspected Mount Bundy Station and took water and soil samples.

79. On 11 June 2011 Dr Fonda received the residents' urine test results. It appeared to him that six of the seven returned results (three were not returned that day) showed elevated levels of arsenic, and that at least one was highly elevated. The recommended response to elevated levels, documented on the result sheet, included "retest on 24 hour urine immediately" and "remove from exposure".

80. Dr Fonda discussed his interpretation of the results with the Centre for Disease Control, who in turn notified the Chief Health Officer and Executive Director of Health Protection, Dr Paterson. The police and the Coroner were also notified.

81. Later that same day a teleconference was convened and chaired by Dr Paterson. Dr Fonda and representatives from the Northern Territory Police Service, the Department of Health, the Centre for Disease Control, and Environmental Health attended. During this teleconference Dr Fonda presented his understanding of the urine test results for the residents of Mount Bundy Station, together with his clinical impressions of some residents which were causing him concern. It was his expressed opinion that six residents had results indicating elevated levels of arsenic. He also advised that symptoms, possibly consistent with arsenic poisoning, were observed in some of the residents.

82. There was joint consensus that, on the information provided, removing persons from possible exposure to arsenic was both necessary and urgent. To this end, and in the interest of public safety, it was agreed that Mount Bundy Station should close pending further investigation.

83. At 6.30 pm on 11 June 2011 a further meeting was convened and the Chief Minister and other relevant Ministers and Department Chief Executive Officers were informed of the course of action.

84. The Chief Executive Officer of the Department of Health did not have the power to secure the property for public health reasons. Accordingly, consistent with the joint consensus, considering the substantial dangers posed to the safety of the public by arsenic poisoning, and exercising the powers of section 32 of the *Coroner's Act*, I declared Mount Bundy Station to be a restricted area.

85. The police were tasked with ensuring the evacuation and isolation of the property. Residents and visitors to the Station were provided with bottled water and asked to leave the following morning. The Station was fully vacated by midday on 12 June 2011 and the property was secured.

86. Soil and water testing was conducted over the following days. No elevated arsenic levels were detected in any of the relevant (soil and potable water) environmental tests conducted.

87. The soil and water testing results, together with the residents' urine test results, were referred to Professor Jones for expert opinion. On review she realised that the urine results had been incorrectly interpreted by Dr Fonda. Her review determined that in fact no residents had elevated levels of arsenic. This, together with the clear environmental results, satisfied Professor Jones that Mount Bundy Station could be safely re-opened. This opinion was provided on 15 June 2011 and the restricted area declaration was lifted.

88. Whilst I consider that the approach taken was appropriate to the reported information, it is clear that the information provided about the residents' urine results was wrong. Unjustified confidence was placed on Dr Fonda's opinion (a GP with no experience of arsenic), without recourse to the results themselves. Further, it is apparent that at the time decisions were being made, there was inadequate reliable information available about arsenic, how people might be exposed to it, and the dangers it posed. Dr Paterson frankly conceded she had difficulty accessing expert information and assistance. Encouragingly, within two days Dr Paterson did locate Professor Jones to provide expert advice.

89. In light of challenges identified on review of this event, the Department of Health put in place a number of measures designed to assist in the better management of any future environmental emergency or disaster situation. These include:

(i) The development and maintenance of a Health Protection Division list of interstate experts including experts in clinical and environmental toxicology,

(ii) An updated environmental fact sheet on arsenic in drinking water,

(iii) Guidelines for health care providers on arsenic poisoning,

(iv) Departmental guidelines for advice to be given on bore water testing,

(v) An update on and reinforcement of the Centre for Disease Control's after hours and weekend procedures,

(vi) A standardised process for receipt of laboratory results, and

(vii) The development of standard operating procedures for rare or unusual events.

90. I am satisfied that the measures taken are an appropriate response to the challenges identified by this incident. I also note that on 2 July 2011 the *Public and Environmental Health Act* 2011 came into force. Pursuant to that Act, the Minister can now declare a public health emergency. Such a declaration can result in a number of actions being taken, including evacuation and isolation of an affected area.

91. I am satisfied that no recommendations arise from this inquest.

Formal Findings

92. Pursuant to section 34 of the Coroner's Act, I find, as a result of evidence adduced at the public inquest, as follows:

(i) The identity of the Deceased person was Ben James Witham born 7 May 1994. The Deceased resided at Mount Bundy Station, Adelaide River, in the Northern Territory of Australia.

(ii) The time and place of death was 11.15 pm on 24 May 2011 at the Women's and Children's Hospital, Adelaide.

(iii) The cause of death as determined by the autopsy was multi-organ failure with terminal fungal (Aspergillus) sepsis on a background of acute lymphoblastic leukaemia. A contributing cause of death was gastric perforation on 13 May 2011.

(iv) Particulars required to register the death:

1. The Deceased was Ben James Witham.

2. The Deceased was a student.

3. The cause of death was reported to the coroner.

4. The cause of death was confirmed by post mortem examination carried out by Dr John Gilbert on 27 May 2011.

5. The Deceased's parents are James Scott and Susan Maree Witham.

Dated this 29th day of October 2012.

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GREG CAVANAGH
TERRITORY CORONER